A Dissertation on

"A COMPARATIVE STUDY OF MINOR PHYSICAL ANOMALIES AND SOFT NEUROLOGICAL SIGNS IN PATIENTS WITH SCHIZOPHRENIA AND GENERAL POPULATION"

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements
For the award of degree of

M.D. (PSYCHIATRY)
(Branch-XVIII)

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THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,
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CERTIFICATE

This is to certify that this dissertation titled **"A COMPARATIVE STUDY OF MINOR PHYSICAL ANOMALIES AND SOFT NEUROLOGICAL SIGNS IN PATIENTS WITH SCHIZOPHRENIA AND GENERAL POPULATION"** submitted by **Dr. JENNIFER SANGEETHA. S.** to the faculty of PSYCHIATRY,

The Tamil Nadu Dr. M.G.R. Medical University, Chennai, inpartial fulfilment of the requirements in the award of degree of M.D. (PSYCHIATRY) Branch -XVIII for the April 2016 examination is a bonafide research work carried out by her during the period of Feb 2015 to July 2015 at Government Stanley Medical College & Hospital, Chennai, under our direct supervision and guidance of **Prof. Dr. W.J. ALEXANDER GNANDURAI M.D. DPM.,** Professor and Head of the department, Department of Psychiatry at Stanley Medical College, Chennai.

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DECLARATION

I, **Dr. JENNIFER SANGEETHA. S** solemnly declare that the dissertation **"A COMPARATIVE STUDY OF MINOR PHYSICAL ANOMALIES AND SOFT NEUROLOGICAL SIGNS IN PATIENTS WITH SCHIZOPHRENIA AND GENERAL POPULATION"** is a bonafide work done by me during the period of Feb 2015 to July 2015 at Government Stanley Medical College and Hospital, under the expert supervision of **Prof. Dr .W.J.ALEXANDER GNANADURAI M.D., DPM.,** Professor and Head of Department Of Psychiatry, Government Stanley Medical College, Chennai. This thesis is submitted to The Tamil Nadu Dr .M.G.R. Medical University in partial fulfillment of the rules and regulations for the M.D. degree examinations in Psychiatry to be held in April 2016.

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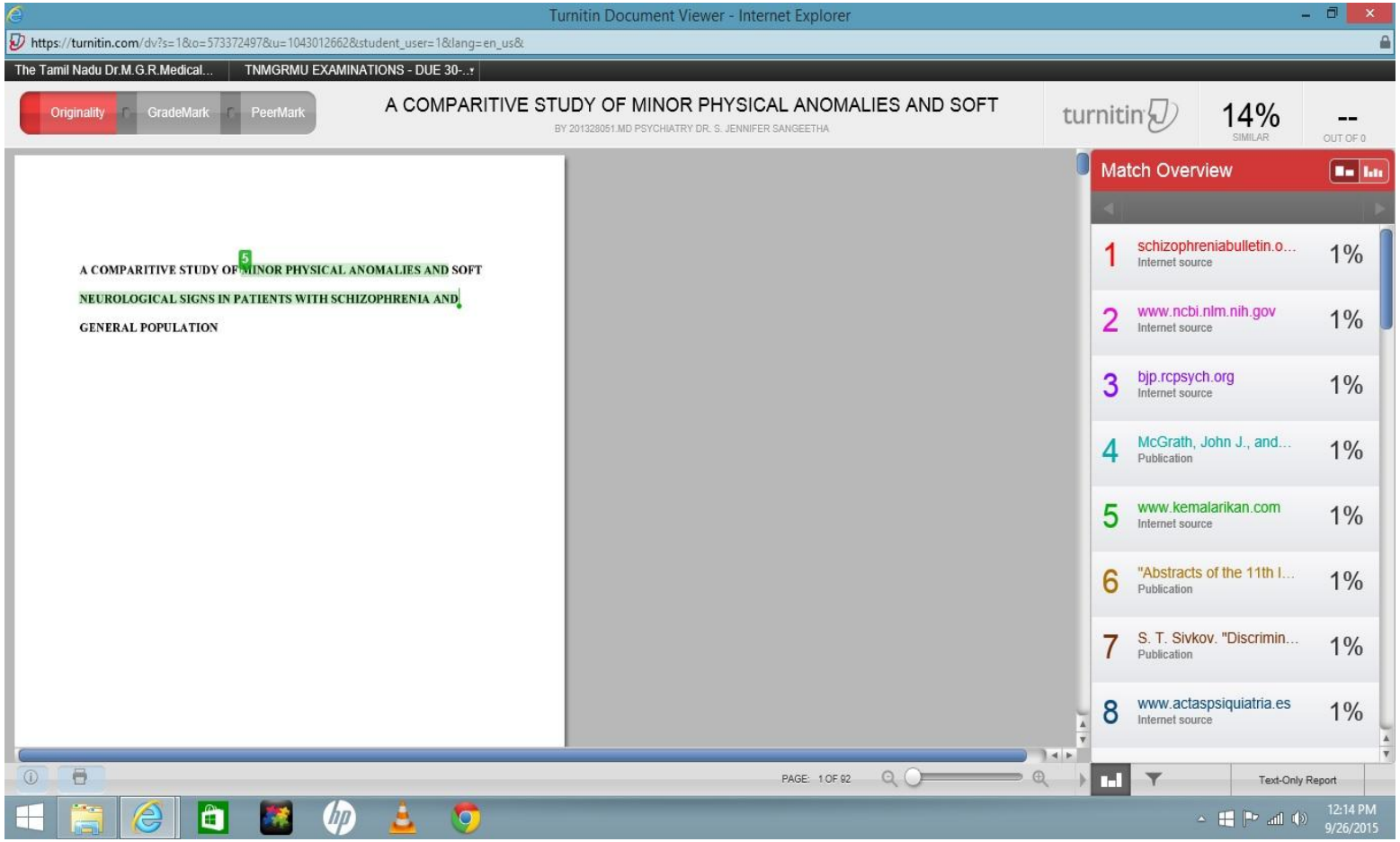
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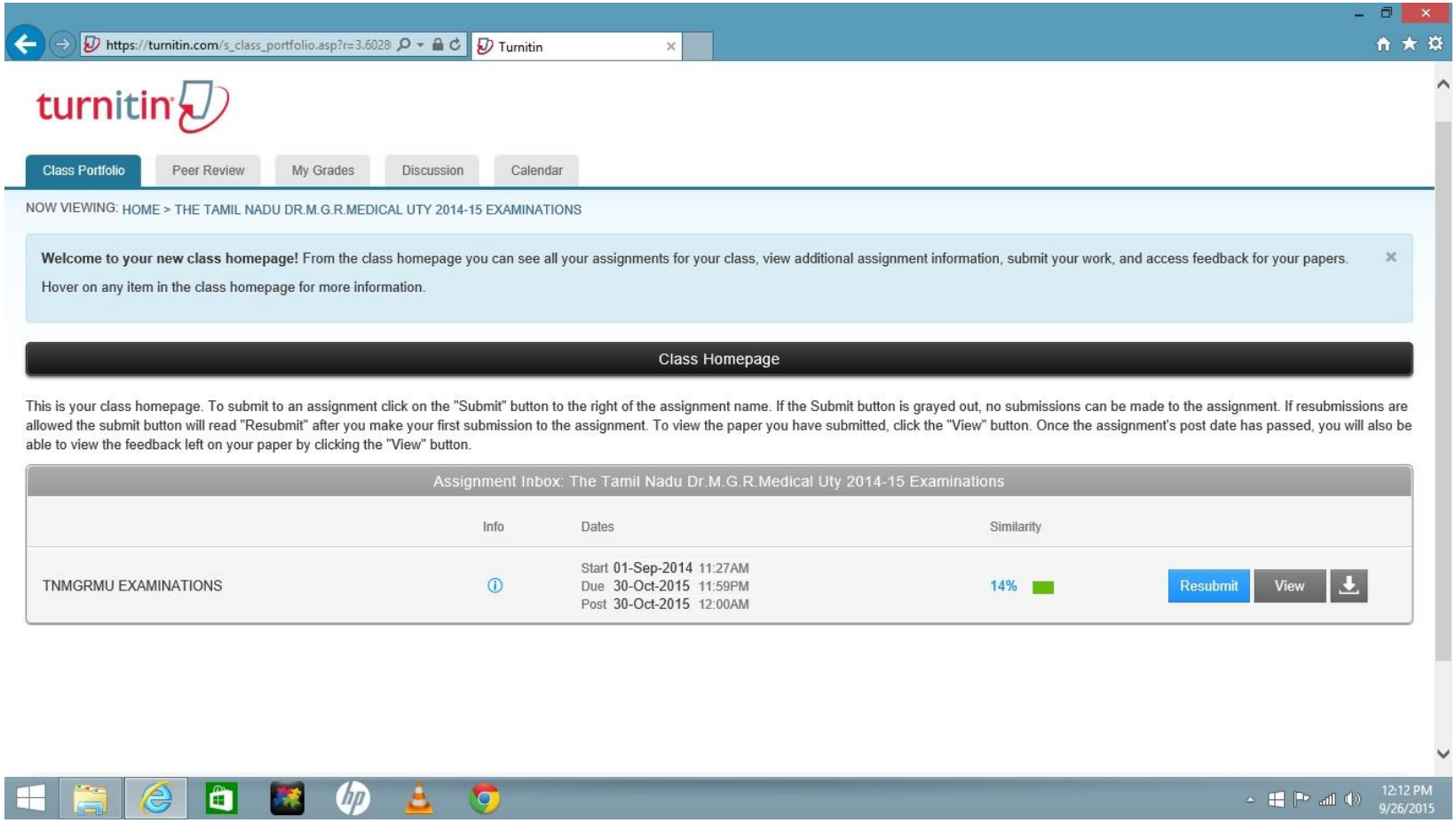
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INTRODUCTION

INTRODUCTION

Schizophrenia is perhaps the most dramatic and tragic manifestation of mental illness known to mankind. An understanding of schizophrenia as a human brain disease did not develop until 19th century. Schizophrenia is conceptualized in today's classification system as a highly complex disorder caused by varied composition of both genetic and environmental factors. The clinical syndrome represents the participation of different multiple pathogenetic pathways.

NEURODEVELOPMENTAL MODEL[3]:

The neurodevelopment hypotheses of Schizophrenia affirm that a proportion of Schizophrenia initiates with impaired fetal or neonatal neurodevelopment rather than in young adulthood when the psychotic symptoms first manifest.

Following are evidences of neurodevelopmental disorder:

- The strongest documentation of disordered neurodevelopment comes through the observation of premorbid behavioral, neurocognitive function and minor physical anomalies (MPA) as well as evidence of risk factors essential for brain adversity.

- Craniofacial dysmorphology gives clue to the neurodevelopmental process in Schizophrenic illness. As the cerebral morphogenesis is significantly equated to craniofacial morphogenesis, Minor physical anomalies(MPAs) rep-

resents biological markers of first and early second trimester dysgenesis and have been found to exist more in schizophrenic patients.

- Neurological soft signs (NSS) has been closely associated with schizophrenic patients. These are neurological abnormalities of minor significance in sensory and motor performance found by clinical examination and they reflect failure in the integration within or between sensory and motor systems. The prevalence of NSS in schiophrenic patients were determined by Heinrichs and Buchanan.[1] In comparison to control groups which showed a prevalence of 5%, the study estimated a prevalence range of 50-65% in patients with schizophrenia.

- Increased prevalence of abnormal septum pellucidum
- Environmental risk factors mostly obstetric complications
- Increased prevalence of abnormal dermatoglyphics.
- Reduced volume of the PFC and temporal lobe,Enlarged lateral and third ventricles
- Reduction in grey matter volumes.

NEURODEVELOPMENTAL THEORIES:

The dominant model of schizophrenia was the Kraeplinian concept of a degenerative disease- Dementia praecox. However the earliest modern conception dates back to Thomas Clouston[2]who discovered developmental dysmorphic deformity like high arched palate in adolescent insanity patients

and neuropathologist Southard who noticed brain changes that were assigned to developmental aberrations .

CHRONOLOGICAL ORDER OF 'HIT' MODELS[4]

Timing of the developmental pathophysiology is based on the clinical observation that characterizes the course of schizophrenia.

(i) Premorbid deficits pointing to early developmental model.

(ii) Characteristic onset in adolescence favours late developmental model.

(iii) Post-illness progression model explains deterioration during early course of schizophrenia.

THE EARLY DEVELOPMENTAL MODEL

- This model denotes many factors operating both intranatally or perinatally during early gestational period .

- The process of programmed cell death, neuronal migration or proliferation of synapses that begin in the second trimester were also found to be involved.

- In "early" neurodevelopmental models, proposed by Murray et al[5] and (Weinberger)[6] ,early life fixed aberration gets engaged in normal brain maturation, supported by cytoarchitectural abnormalities indicative of a possible error in developmental process of neural genesis and its migration in schizophrenia.

- History of obstetric complications ranging from viral infections to starvation to autoimmune processes and other such problems in pregnant mother.

- These suggest that the insult to the brain early in fetal development could contribute to the cause of schizophrenia.

- Disruption in ectodermal development resulting in minor physical anomalies.

- These risk factors may all have the final common pathway of reducing nerve growth factors, and also stimulating certain noxious processes that kill the neurons such as cytokine, viral infections, hypoxia, trauma, starvation, or stress meditated either by apoptosis or by necrosis.

- All these lead on to structural abnormalities or more subtle problems like selection of the wrong neurons to survive in the fetal brain, neuron migration to the wrong places, neuron innervation of the wrong targets,

- reduced number of synaptic vesicles, aberrant synapse formation, and delays or reduction in synapse formation.

CONCEPT OF ENDOPHENOTYPES

Endophenotypes, posits towards biological variant equivalent to genetic risk for schizophrenia. This has been proposed by Shields and Gottesman (1972)[8] and cited as unnoticed "internal" trait. They are heritable,

biological abnormalities, and they lie between genes and clinical symptoms and they provide more power for finding disease genes.

The list of putative intermediate phenotypes includes eye tracking, cognitive, neurophysiological, and neuroimaging measures, minor physical abnormalities and neurological soft signs, symptom rating of positive and negative symptomatology has been evaluated as an intermediate phenotype. The personalities which looked odd among the relative schizophrenic cases has been explored as endophenotype by Kraeplin and Bleuler.

RISK FACTORS FOR SCHIZOPHRENIA:

A certain neurodevelopmental hypothesis advocating a possible environmental or genetic influence on the brain development in early periods of life having a negative impact on mental health in adulthood was evidenced by research studies on the role played by environmental factors on schizophrenia (Murray & Lewis, 1987; Weinberger, 1987)[5,6]

EARLY EXPOSURES TO BIOLOGICAL HAZARDS:

Season of birth:

It is observed that there is increase in the possibility of schizophrenia for subjects born during the months of winter-spring. During these months it is known that chances of prenatal viral exposures (Torrey et al., 1997)[9] is more as well as vitamin deficiencies (McGrath, 1999)[10]. It is also observed that the possibility is increased by high latitudes.

Risk of occurrence of schizophrenia is increased by the nutritional deprivation during prenatal months. (Brown et al., 1996)[11]. The risk of occurrence of schizophrenia is also increased by very low vitamin D levels in winter, during the third trimester of pregnancy (McGrath et al.,2003a)[10].Schizophrenia is also associated with low weight at birth as well as during infancy (Wahlbeck et al., 2001a).

Prenatal infection:

Increase in number of births with schizophrenia during late winter–spring months were associated with maternal exposure to winter- borne viruses especially influenza virus. Prenatal exposure to herpes simplex virus type 2 (HSV2) may also increase the risk of schizophrenia .

Pregnancy and birth complications:

Neurodevelopment will be affected if there are complications during pregnancy and birth (PBCs). Some of the effects are: antepartum bleeding, diabetes mellitus, Rh incompatibility, pre-eclampsia, low birth weight (LBW), congenital malformations, small head circumference, atony of uterus, asphyxia, and emergency cesarean section.

One of the study confirmed that schizophrenic patients who had a genetic predisposition are more likely to develop decreased hippocampal volume and an enlarged ventricular volume in response to early prenatal and perinatal events or complications (Cannon et al., 1989). Some of the findings implied that the hippocampal and ventricular volume has been regulated by the

genetic factors operating in schizophrenia and its combination of influence along with fetal hypoxia caused by the early complications of perinatal life. (Nicodemus et al., 2008).

Advanced paternal age:

Most of the study comes from Sipos et al. (2004) [12]which proved the influence of increasing paternal age on the etiology of schizophrenia, which has been studied in Sweden. The risk ratio for development of schizophrenia advances for every increase of 10 year increase in paternal age which was around 1.47. The correlation between the advancing paternal age having an influence on schizophrenia, by increasing the risk for development of schizophrenia, has been found with cases without a family history of schizophrenia. But such association was not present in patients having positive family history (Malaspina et al., 2002; Sipos et al., 2004).[12]This possibility implies a de novo mutations in paternal sperm with aging contributing to the risk. It is probable that, in the sperm of older fathers, there may exist a compromise in epigenetic processes and such mechanisms serve to increase the risk of schizophrenia in the children born to these fathers. (Perrin et al., 2007) [13].

Exposure to later biological candidate risk factors:

Schizophrenia-like psychosis can be induced by the abuse of substances like amphetamine (Carroll, 1958).[14] Genetic factors found to play an important role that cause the increase in sensitivity: the development of psychosis was not evident in patients, with a negative family history, abusing the drug as opposed to patients with a positive family history, who experienced psychotic symptoms

on abusing the drug. Vulnerability to psychosis after cannabis use has a genetic basis.(Capsi et al 2005)[15].

Early Rearing Environment:

During the first 2 years of life the brain continues to develop even though it is susceptible to adverse environmental factors. This early rearing environment influence has later emotional and psychological outcome (Bowlby et al.,1944, Kendler et al 1992., Murray et al).

Impact of abnormal gestational experience:

The most frequently observed complication is fetal hypoxia (Cannon et al., 2008)[16] . Cannon et al. (2008) elucidated the association between hypoxic indices at birth (low Apgar scores) and circulating quantity of brain-derived neurotrophic factor (BDNF) in the cord blood sample . They inferred that increased BDNF levels were associated with hypoxia of early life events and the levels were reduced in people who obviously developed schizophrenia in adulthood . Many studies also specified that increase in the risk of adult onset of the disorder can be caused by abnormalities of the intrauterine environment in early development, during the first two trimesters during which neuronal production and its migration to their final locations tend to occur.

GENETICS OF SCHIZOPHRENIA:

Schizophrenia is considered a genetic disorder due to the following reasons.

- Increased incidence of schizophrenia seen in family members of people suffering from schizophrenia.
- Increase in the incidence of disease propotionate to the degree of relatedness to the individual
- Increased incidence in children adopted away from psychotic parents
- Adopted away offsprings of normal parents even if reared by schizophrenic parents do not have increased incidence.

TWIN STUDIES

A metaanalysis of the twin studies done by Sullivan et al shows a greater value of heritability in schizophrenia to be around 81 % and also found a common environmental effect on the liability to schizophrenia .The common environment refers to the process that makes members of twin pairs similar regardless of zygosity and this encompasses pre and post natal environmental factors and also genomic processes like DNA methylation patterns.

ADOPTION STUDIES

These studies were done to elucidate the role of environmental and genetic factors in the transmission of schizophrenia. The important studies were by Leonard Heston, David Rosenthal. There were highly significant excess of schizophrenia in adopted away off springs of schizophrenic parents.

Linkage studies in schizophrenia:

Association is found with chromosome 1 q, 5 q , 6p, 8p , 10p , 13 q , 15 q , 22q .

Chromosomal abnormalities:

Further chromosome analysis has revealed candidate genes like

1. Alpha 7 nicotinic receptor
2. DISC-I,2- Disrupted in schizophrenia
3. GRM-3- Glutamate receptor
4. COMT- Catechol O methyl transferase
5. NRG-1 -Neuregulin 1
6. RGS-4 - Regulator of G-protein signalling
7. G-72- D amino acid oxidase
8. NOTCH-4

CONCEPT OF MINOR PHYSICAL ANOMALIES:

Schizophrenia is a neurodevelopmental disorder which manifests abnormalities of ectodermal structures formed simultaneously with cerebral cortex during intrauterine development. An indirect evidence for cerebral maldevelopment is the presence of MPA.

It was found by Thomas Clouston in the later part of the 19th century, that abnormalities of palate were a common entity in 'adolescent insanity'- which is a form of psychosis having a very strong familial etiology. MPAs are slight variation in soft tissue, cartilaginous and bony structures which may be due to interplay between the environmental factors and genetic factors and its influence along with early hypoxia that work prenatally. These include

differences in proportion of head, face, fingers, mouth, hands and toes and shape of head

MPAs serve as persistent markers or fossilized evidence of deviance in development in fetal life, particularly as markers of events in early life that might have had an impact on brain development, and hence are studied with great interest. The timing of the early disruption in embryological development may be identified by clues presented by MPAs. MPAs were related with large varieties of environmental insults during gestation like maternal infection, toxin and fetal anoxic ischemia which act on genetically programmed early development.

SCHIZOPHRENIA AND MORPHOGENESIS OF MPA:

A detailed illustration of the neurodevelopmental etiology for schizophrenia was put forward by Waddington et al [17] The cerebral impairment in schizophrenia is concerned with changes in the prefrontal/cingulo-striato-pallidothalamo-cortical/fronto-temporal tracts that affect the interconnections within the midline. Morphogenesis of Cerebral and craniofacial region are intimately related and they share a common origin from the ectodermal region. They are identified as abnormalities in organogenesis .The palate, originates between 6 and 9 weeks of gestation and develops postnataly between 16 and 17 weeks. It is associated to the vertical elongated growth of the middle part of the face and to broadening of the frontonasal region. These developmental changes in the early developmental period reflect lesions that take place during embryological development of the craniofacial region. The

hippocampal region differentiates in 9 to 10 weeks of gestation. The hippocampal volume reduction is seen in people with schizophrenia. The significance of MPAs in schizophrenia is that it shows a substrate that is acquired or inherited due to injury and may result in initiation of disease process in susceptible individuals[79].

It may be possible that MPAs-which, are fixed markers present through childhood and adolescence well before the onset of the prodrome and psychosis may have utility in terms of risk stratification for future preventive efforts It's possible that detailed MPA assessments will enhance early prediction of risk for schizophrenia and this may sharpen the application of future preventive efforts.

MPA AND PRENATAL INSULTS:

Timing of the prenatal insult influences the nature and severity of the anomaly. As the process of developments are due to interplay between maternal genes, the genotype of the embryo, and environmental influences, the etiological origin of MPAs is likely to be complex. Specific pregnancy and birth complications like low birth weight, prematurity, small for dates status, preeclampsia, pro-longed labour, asphyxia and hypoxia may contribute to development of MPA.

MPA AND GENETICS:

There is a strong association between family history of psychosis and MPAs in schizophrenic patients. One study of MPAs in patients with

schizophrenia showed that MPA scores were associated with a positive family history of schizophrenia in a first-degree relative and along with obstetric complications.

SOFT NEUROLOGICAL SIGNS:

Soft neurological signs (SNS) are slight neurological abnormalities. It includes deficits in sensory integration, motor coordination & sequencing of complex motor acts. These abnormalities have been called "soft" in general as they were thought to lack its specificity, validity or localizing value. Neurological signs such as rigidity, tremors, and imbalance have been found in patients with Schizophrenia since the time of Kraeplin. Other authors believe that a developmental lag is a general sign rather than a fixed abnormality. Studies have shown that a incidence of soft signs is high in children who were born premature or low birth weight birth, meningitis, and malnutrition.A considerable body of research has established that Soft Neurological Signs are more common in patients with Schizophrenia than in healthy subjects. The occurrence of Soft Neurological Signs in Schizophrenia varies from 50% to 65% compared to 5% in healthy controls.

In 1988, Heinrichs and Buchanan reviewed the importance of SNS for our better evaluation and understanding of etiological part of schizophrenia.

Increased NSS scores were associated with deficient executive functions, long duration of untreated psychosis, poor premorbid social adjustment, pregnancy and birth complications. Bombin et al.[11] observed that the presence of neurological soft signs is in predominance in patients with

schizophrenia independent of demographic and medication variables. It were strongly associated with negative symptoms and cognitive impairment. It has been mentioned that there is evidence for these signs be under genetic control and that they may represent a trait feature of schizophrenia.

PREVALENCE OF NSS:

As per earlier studies, it is estimated the prevalence of NSS in schizophrenic patients is between 50% and 65%, against 5% in control groups.

There is strong evidence to imply that NSS including cerebellar signs may form an inherent part of schizophrenia. This supports the neurodevelopmental hypothesis for the etiopathogenesis of schizophrenia as well as the model of "cognitive dysmetria" to explain some of the features seen in this enigmatic disorder.

Previous studies showed evidence that schizophrenia is a neurodevelopmental disorder as shown by significantly higher presence of neurological soft signs and minor physical anomalies .There has been relation-ship shown between neurological abnormalities, family history of major mental illness, symptom dimensions, obstetric complication, course of illness and being a high risk individual.

SCOPE OF THE STUDY:

There is a need to study MPA and NSS and their relationships in patients with schizophrenia. Such a study may help in further understanding of schizophrenia, preventing expression of illness in those at high risk, and plan

for future target of treatment for various dimensions as MPA probably represent the complex link between maternal genes, the genotype of the embryo, and environmental factors during fetal development it strengthens the neurodevelopmental etiology of schizophrenia.

REVIEW
OF LITERATURE

REVIEW OF LITERATURE

Based on the review of literature evidences suggest that schizophrenic patients are associated with abnormal prenatal development, obstetric complications, dietary deficiency, influenza virus exposure in prenatal period, anoxia and bleeding. As the central nervous system also develops from ectoderm, the presence of excess physical anomalies was related to abnormal development of CNS. It represents a derangement in embryological development paving the way for the illness.

Most of the previous studies associated with schizophrenia showed increased neurological signs and there were evidences suggesting relationship between symptom dimension and neurological abnormalities, family history of mental illness, course of the disease. Regarding such neurologic abnormalities, the most interesting area for research has been the soft neurological signs. Though soft signs were described in other disorders too, the association with schizophrenia is of particular interest and significant.

Both minor physical anomalies and soft neurological signs were analyzed and compared with normal controls in many works.(Ismail et al, 1998a , b; McNeil et al, 2000) [18] Earlier Indian studies have found significant increased MPAs in schizophrenic patients than normal population.

More than 11 different studies (Lohr and Flynn[35] ; Green et al. 1994 [23]; O'Callaghan et al.[22],,) have proved increased incidence of minor anomalies in adult schizophrenic groups and fewer studies had not shown this association

A shared neuropathological basis was evidenced by a study conducted by slater et al [24] between schizophrenic patients and those suffering from temporal lobe epilepsy. an absence of gliosis and change in neuronal alignment with migration of cortical cells to their final position through radially arranged glial fibres suggested a possible neurodevelopmental etiology as opposed to an earlier believed neurodegenerative theory. Additionally strengthening the above results was the observation of increased obstetric complications such as periventricular hemorrhage, ischemic and hypoxic injuries in schizophrenic patients.

Cannon and colleagues[16] have reported that interplay of fetal hypoxia and increased genetic risk for schizophrenia on brain structure.

He found that labor and delivery complications (LDCs) [26], in particular perinatal hypoxia, are the most replicated obstetric correlates of schizophrenia and it is found to be a major risk factors than pregnancy complications (including viral exposure) and fetal maldevelopment.

This complication act additively or interacts with genetic factors in influencing the risk for disease. Obstetric complications has been proved to be a best-reproduced "environmental" risk factors and should stay at the forefront to elucidate the causal factors and gene-environment interactions.

MINOR PHYSICAL ANOMALIES:

Minor physical anomalies start to develop during the period of the first or early second trimester of gestation. Bodily structures involved in the

expression of minor physical anomalies share their embryonic origin with that of the brain, they represent valuable indices of disturbances in early neurodevelopment.

Another cause for the MPA is neurological damage which has been seems to reflect the interaction with the prenatal or perinatal birth difficulty. (Laufer et al.1957 and Steward, 1970)[36].

Green et al. (1994)[23], found greater, MPA scores in female compared to in male schizophrenics but in their control group males had greater MPAs than the females group. (Alexander, 1994) [27]. Lal and Sharma (1987) [26].

SCALE FOR MPA:

Assessment of minor physical anomalies was elaborated by Goldfarb and Botstein [28](1967) . The listing has 21 anomalies previously, later Waldrop and Halverson [29] (1971) minimally changed the original list, recognising specific minor physical anomalies that has been used to delineate a group of schizophrenic patients .Except Lane et al[30]., the other past researchers have used Waldrop scale for assessing MPA..

DISTRIBUTION OF MPA:

Elizabeth and cantor study[31] have found out that the minor physical anomalies has not been limited to head and facial region alone in the schizophrenia population. Morever mid- line cranial anomalies like enlarged

cavum septum pellucidum and corpus callosum have been found to be increased in patients with schizophrenia.

In a study by Nizame et al [32]the sex difference when considered, the MPA scores showed highly significant difference ($p<.01$) between the male and the female acute schizophrenic patients. It was greater in the chronic (6.6±3.26) than in the acute psychotic patients (5.7±2.2) .Regarding sex difference, males have more minor physical abnormalities (Halverson et al., 1976) [33]Also the SNS were positively correlated with the MP A in acute ($p < .05$) and chronic ($p<.001$) schizophrenic patients in this study.

Akabaliev and Sivkov[34] compared the distribution of MPA in relation to gender and found men had a higher score than women and men had more distribution of MPA in the head region.

Ismail et al 2000 [19] and Compton et al 2007, have found that distribution of minor physical anomalies were not only limited to head and face region .In a study by Waddington et al [17]. (1999), which finds that the palatal anomalies cited as 'midline anomalies' constituted as pathological brain morphogenesis, as there is deep seated developmental relation between the craniofacial region and fetal midline structures .

Numerous studies have reported increased percentage of MPAs in the craniofacial region of schizophrenic individual. (Green et al 1994)[23] (Lane et al 1997, Akabaliev et al 1998) [34]. Anomalies of mouth in particular palatal region were in a study conducted byMcGrath et al., 1994. , green et al 1984

study shows that when men and women were studied together, the difference between them was found to be significant also men showed greater significant change in mouth area and female showing difference in head circumference . More female patients (Thirty-five percent) has a large head circumference, and few (21 percent) had a small head circumference. In a study by Poyraz BÇ, Turan Ş, Arikan MK[37]MPA were highest in patients, low in healthy controls and in between the values in healthy siblings. The common MPA is abnormality of tongue like furrowed tongue (53%) and height arched palate (49%) .

Earlier Indian studies in the field of minor physical anomalies have found increased MPAs in schizophrenic patients than normal population[32].

The onsets of schizophrenia were found to be earlier by 3-5 years in male compared to female group. These has been proved again by a study by Leung Cheu regarding the age of onset, premorbid behavioral patterns, negative symptoms and cognitive deficits.

Also structural brain and neurophysiological abnormalities were also found to be more in males. Females show more affective symptoms, auditory hallucinations and persecutory delusions and rapid and great response to anti-psychotics in the premenopausal period .

Green et al., [23]showed schizophrenia with onset at or before 18 years had more MPAs pointing more towards the neurodevelopmental etiology.

FAMILY HISTORY AND MPA:

In Maudsley family study (38),they separated schizophrenic patients into two groups, one group with no family history (sporadic) and the other with familial schizophrenia positive patients (first-degree family members with schizophrenia). They found greater score of MPAs in sporadic schizophrenia and related this finding that these anomalies represents early developmental markers of insult to brain .

A study was conducted to evaluate MPA on patients with a family history of schizophrenia by shiffman et al [39]and evidences from the study concluded an additive cerebral insult with the positive family history of schizophrenia contributed to the development of the disease in these patients with the MPAs serving as markers for the same

Many studies has shown the significance of total scores of anomalies compared to the individual anomaly pointing towards neurodevelopmental etiology.

AGE OF ONSET AND MINOR PHYSICAL ANOMALY :

Regarding the association between MPAs and age at onset of illness, Green et al [23](1989) Hata et al (2003). reported that a subset of schizophrenics with onset under age 18 years had a significantly increased score on MPA subjects.

(Akabaliev and Sivkov, 2003[34]; Lohr and Flynn, 1993 [35]; O'Callaghan, in their studies have found no correlation to age of onset and MPAs.

Study by Leila Gassab and Mauna Aissi,[40] they studied MPAs in a Tunisian population: in subjects with schizophrenia, their healthy siblings and controls. .MPAs were inversely related with age of onset of the disease, and age of first hospitalisation, and directly related with number of hospitalizations. There is positive correlation with negative symptoms and severity of illness

Hata et al[41] studied that early onset schizophrenia <18yrs have more abnormalities in the cephalic region like hypertelorism, high arched palate, low set ears, furrowed tongue. MPAs were studied in other disorders also where there is disturbance of morphogenesis.

O'Callaghan et al[22]found that age of onset was not relevant to the anomaly. Early onset disease and MPA association point towards neurodevelopment etiology.

An evaluation by Andreasen et al. (1986) using imaging (MRI) had found that male schizophrenics had significantly smaller cranial size compared to normal controls. Waldrop & Halverson (1971)[29] found that large head circumference and hypertelorism were seen in patients with more than in controls, but Green *et al* (1989) [23]found head circumference changes in female patients, and Lane *et al* (1997) found a widened skull base but a narrow binocular diameter in patients. But most of the studies have reported enlarged head circumference. More than ten studies reported insignificant difference (Ward et al 1996).

MPA have not been associated with positive or negative symptomatology of schizophrenia and has been shown in studies. Several studies have proved no positive association between MPA and positive and negative psychopathology on PANSS score as shown few studies done by Compton and Mc Grath[43,44]

SOFT NEUROLOGICAL SIGNS:(SNS)

INTRODUCTION:

Soft neurological signs (SNS) are non-localizing abnormalities that has been due to the damage in the connection between the cortical and subcortical structures.(Dazzan et al 2004)[45].

It represents a failure of association between sensory and motor systems (Griffith et al 1998)[38]. They have been stable over time as they appear like trait features. (Marcus et al). SNS appears early in illness. They do not appear to be secondary to medications (Arango et al 2000) and they can be measured. Most of the studies proved the association between the soft neurological signs and schizophrenia compared to other psychiatric disorder. (Coxludwig et al 1994).

ASSESSMENT OF SNS:

Cambridge neurological inventory[46] is an instrument which is comprehensive, reliable and easy to administer. It includes items from the functional areas of interest like sensory integration, motor coordination and sequencing of complex motor acts.

STUDIES OF SNS IN SCHIZOPHRENIA:

In a study by Anwar et al found a strong correlation between Soft neurological signs and schizophrenia independent of their respective ethnic and socioeconomic groups.

Many studies have done to show the association between SNS and gender (Bjorck statin). Duggal et al said that there is an influence over motor sequencing tasks by sex variable (Duggal HS and Keshavan).

Severe neurological impairment was found more in males than females. But this has been contradicted and showed no significance between sex and neurological signs as shown by Bombin et al 2005.

There has been an association between both negative symptoms and the treatment response which has been shown in a study.

The prevalence rates found in many of the studies were about 50 to 65% in schizophrenic patients and about 5% in normal controls[47] .

Most of the studies showed a positive association between SNS and schizophrenia and only few studies like that have been done in Nigeria found no association.

When compared to the distribution of SNS in schizophrenia and to other disorders like OCD, substance abuse, it was found to be less in these groups. But when compared to affective disorder the prevalence is equal.

Studies conducted by Keshavan et al[3]., proves disturbance in sensory integration but not in motor dysfunction when schizophrenics were compared to normal population.

SOFT NEUROLOGICAL SIGNS (SNS) AND PSYCHOPATHOLOGY:

There is no positive association between positive symptoms and SNS as shown by studies like Arangokirkpatrick and Buchana[48]. SNS have found not significantly associated with age of patient, age of onset, hospitalisation and to severity of illness like positive or negative, disorganisation symptoms as shown by Arikan et al in a study.

Patients with positive SNS has shown to have poor social adjustments and poor premorbid adjustments (Rochford et al) (Quitkin et al). Acute schizophrenics have less SNS compared to chronic as shown by Nizamie et al study specifying a neurological cause for the disease.

Total SNS score was associated with negative scale and dis-organization symptoms scale as shown in a study by Arikan et al.

SOFT NEUROLOGICAL SIGNS (SNS) AND ANTIPSYCHOTICS:

Antipsychotics initiation leads on to development of extrapyramidal signs mistaken as neurological signs, hence the study to prove the etiology of SNS as it is due to brain impairment which is the central factor for schizophrenia.

Gupta et al[69] compared antipsychotic naive and treated patients and found increased rate of SNS in treated group. Scheffer et al[71] applies the scale and assessed neurological signs prior to treatment initiation to a group of drug naive patients and patients who were on drugs and found no modification in SNS scores.

Keshavan et al (2003) [70]have found the first episode drug naive patients showing higher score in sensory integration area.

Schroder found antipsychotic naive has more neurological signs than on treated patients. In a work by Arango, kirkpatrick[48] 83 clinically stable patients with schizophrenia were studied for delusions, hallucination, disorganisation and deficit syndrome dimensions were studied in relation to SNS signs and the disorganisation symptoms were related to total score to sensory integration and to complex motor acts and integration of sensory symptoms were related to sensory symptoms.

97 first episode schizophrenia patients were followed up for neurological symptoms which got improved when psychopathology came down in a study by Whitty. P.Clark. [72]

In another study by Venkadasubramaniam et al 2003 [49] found higher SNS in drug naive patients and there were no relation to the duration of illness.

A study was conducted between schizophrenic patients and their non-affected siblings by Schreiber et al. 1995 [73];Ismail et al. 1998 ,with 11.5 as the cut off score for SNS and it was found that there was an increased frequen-

cy of NSS in schizophrenic patients compared to their non-affected sibling counterparts. The motor coordination, motor and sensory integration subscores were higher in schizophrenic patients and their siblings. There has been a study to find correlation between illness severity and to the negative and disorganisation symptomatology.

More impairment in neurological function in schizophrenic persons ,and found that to be there with a family history of psychosis, pregnancy and complications during birth, male patient, and a continuous course of illness. Smith et al[74] found no change in course of illness in chronically ill patients with schizophrenic diagnosis over a period of 5-years.

Emsley et al [75] have found that except for the motor sequencing sub-scale, NES scores did not change over a 1 year period which has been associated with symptom reduction also.

97 first episode schizophrenic patients were followed up for neurologic symptoms which got improved when the psychopathology came down in a study by Whitty, P, Clarke.

To assess neurological signs in preschizophrenic children, Walker and others did a study and found that, early motor signs as precursors to illness. Rosso et al found siblings of pre-schizophrenic children shown more motor co-ordination than normal controls. The incidence of NSS in normal control has been found to be from 5% (Hertzig& Birch, 1968; Rochford et al, 1970) 50% (Kennard, 1960; Cox & Ludwig, 1979), hence the significance in comparing the neurological signs in first episode and treated patients.

Greater NSS scores in motor sequencing scale forecasts worst treatment response which has been shown by a study by Smith and Kadewari .[50]

Chen et al [51]examined neurological signs as familial vulnerability factors. Fifteen schizophrenic patients, their siblings 21, and 26 healthy volunteers were included. They were matched for age, sex, and education and their neurological signs were assessed.

It when has been found that both the patients and their siblings when compared to controls has more impairment in neurological signs suggesting different etiological origins for different subgroups of neurological signs.

Another study on genetic liability for schizophrenia were done by Niethammer et al[63] and he explained hereditary factors showing greater significance for neurological soft signs and the hemispheric laterality.

The twin study in schizophrenia which compared between unaffected and affected twin predicting more signs on left half of body thus proving the neurological signs and the laterality as genetically inherited. There has been no consistent association between the neurological signs and symptomatology.

The association between neurological soft signs and clinical dimensions of different psychopathological symptoms were not consistent and has been shown only in few studies.

The neurological cause for the disease has been strengthened by study by Nizame et al[21] who showed increased prevalence of MPA and NSS in chron-

ic schizophrenics compared to acute schizophrenics. Also similar works has been replicated by other studies (Torrey, 1980; Weller et al 5) [52].

The association between the course of the illness and neurological soft signs has been studied by Wood et al. (1986)[54] and Weller et al. (1979) [53]but found no association between them.

Nizamie et al [21]studied the MPA and SNS in schizophrenic patients and 107 patients were included in the study and SNS found to be positive in 53.33% in chronic and in acute it was around 17.32 %. The MPA scores were found to be more in chronic groups which are around 6.6% and 5.7 % in the acute groups.

When comparing the genders it was found that the minor physical anomalies were more in male when compared to female.[76,77]

Regarding soft neurological signs the gender distribution showed more signs in males than females.

The positive association between MPA and SNS has been studied in only few studies and it was found to be positive in Nizamie et al study and this were more in chronic schizophrenic.

Waldrop et al. (1971) and Paulsen et al. (1979)[55] also described similar findings regarding this association. Luria considered that there is damage to subcortical frontal structures for the emergence of release reflexes. He also found the coincidence of symptoms to the appearance of release reflexes.

The off-springs of schizophrenic patients were studied to find the association between MPA and SNS by Marcus et al [78]in his study , and found that chronic patients has more SNS and MPA, and also they used to be found together.

Similar association study regarding the neurological signs was done by Michael Compton regarding the neurological signs and found higher score on sensory integration and with total MPA scores.

The over expression of these signs in schizophrenia has been established and has been compared with the other disorders also. Tiryaki et al. (2003)[56] showed increased sores in sequencing of motor acts along with negative symptoms.

But in our study though there is increased response in sequencing of complex motor acts it shows no association with positive or negative symptomatology.

Flashman[57] demonstrated a poorer performance on neuropsychological tasks that assessed timed motor speed and motor coordination and found it to be present even after the start of the drug. Mohr et al. gathered and argued that "cognitive disorganization" correlates with NES scores.

Behavioral disorganization symptoms and its association with complex motor acts and sensory integration by Kirkpatrick, B, Buchanan[48]. Frontal signs with visuospatial memory and visuospatial processing were also done by Kirkpatrick.

Mohr et al and the relation between the executive function and sequencing of complex motor acts were studied by Smith et al.

Scheffer et al[58] applied the NES prior to treatment initiation to 26 patients who were not on drugs and 3 drug-free patients with schizophreniform psychosis before and after 6-week treatment with drugs and found no change in the neurological soft sign scores.

A study was done by Arango and Kirkpatrick[48] to find the association between SNS and positive symptoms like delusion and hallucination and with disorganisation symptoms, negative symptoms and it was found that the disorganisation symptoms has been correlated to total score in sensory integration scale and the deficit syndrome to sequencing of complex motor acts.

More impairment in neurological function in schizophrenic persons and found that to be more with a positive family history of psychosis, pregnancy and problem during birth, male patient, and also a continuous course of illness. Smith et al showed no change in course of illness in chronically ill patients with schizophrenic diagnosis many times over a period of 5-years.

Bachman and colleagues[59] in a prospective study of patients with first episode of schizophrenia showed that despite reduction in the severity of NSS and having not associated with psychopathology they still remain elevated in patients compared to healthy subjects.

Quitkin and colleagues 1976 found increased soft signs in unmedicated schizophrenics. Merriam et al 1990[60] found a significant relationship between the drug dose and neurological soft signs.

King et al also recorded association between drug use and soft signs. Study by Claude .M.J. Braun[61] showed no association between neurological signs and positive and negative symptomatology of schizophrenia.

Another study on genetic liability for schizophrenia were done by Niethammer al [63] explained a genetic basis for neurological soft signs and the hemispheric laterality.30 monozygotic twins, 13 pairs discordant for schizophrenia and 17 healthy twin pairs were the study population. The twins for schizophrenia showed increased number of neurological signs.

Relation between positive family history and the impairement in the neurological signs were studied and were found to be negative and it has been studied by Egan et al[64]. He studied between patients with schizophrenia and their siblings and compared to normal controls.And all these studies finally prove the genetic inheritance of these signs. Among these, motor signs were not related to obstetric complications.

The association between the positive symptoms and neurological signs were studied by Addington *et al.* (2007)[65] and it was found to be not associated with the signs. The poor performance on neurological function were found to be related to negative and disorganisation symptoms and not to delusions and hallucinations and were studied by Brazo *et al.* (2005), Tosato and Dazzan (2005)[66].

MINOR PHYSICAL ANOMALIES (MPA) AND SOFT NEUROLOGICAL SIGNS (SNS) ASSOCIATION:

John et al (2008)[67] compared MPA scores and NSS as a composite endophenotype for schizophrenic subjects with a matched healthy controls and noted that increased occurrence of MPA and SNS in schizophrenia when compared to healthy controls . There were studies to find the relationship between SNS and MPA.

Nizamie et al study has shown a positive relationship between SNS and MPA in acute and chronic schizophrenics.[21]

Quinn and Rapopport studied no association between them. But in a study by Paulsen and O'Donnel[55] found association between the MPA and SNS.

Waldrop and Halverson (1971) and Marcus et al. (1985) recorded a modest positive relationship between the two groups.

These studies between the association with the schizophrenic diagnosis and the soft signs enlarge our understanding towards the neuropathology and these signs may be used as a trait marker which improve the knowledge on genetic cause,

So, still more to be dwelled and a widened knowledge to be sought in the area of soft signs in schizophrenia .

When MPAs are regarded as imprints of early dysontogenetic processes (Ismail et al. 1998) which has been unchanged by the disease process and its course, their over expression in schizophrenia patients indicate a injury in perinatal period that may increase the risk of diseases in their late phases of life (O'Callaghan et al. 1991)[68].

AIMS AND OBJECTIVES

AIM AND OBJECTIVES

AIM:

To assess the presence of minor physical anomalies (MPA) and soft neurological signs (SNS) in patients with schizophrenia and to compare them with general population.

OBJECTIVES:

- To assess the frequency and topographical pattern of minor physical anomalies (MPA)in schizophrenia
- To explore the possible relation between severity of schizophrenia and presence of soft neurological signs
- To identify the association of minor physical anomalies with the severity of schizophrenia
- To compare the presence of minor physical anomalies in patients with schizophrenia and with general population
- To observe any difference in soft neurological signs in schizophrenics against the general population

HYPOTHESIS:

- The prevalence of minor physical anomalies is more in schizophrenia patients.
- Schizophrenics exhibit a higher prevalence of soft neurological signs
- The severity of illness is associated with minor physical anomalies and soft neurological signs.

MATERIALS & METHODS

MATERIALS AND METHODS

INCLUSION CRITERIA:

1. Cases and controls were selected in the age group between 15 and 45 years (both males and females).

2. Persons who meet ICD 10 criteria for schizophrenia were chosen as cases.

3. Persons accompanying patients attending medical OPD were chosen as controls.

4. Patients who were selected as cases were both first episode drug naive and patients who were on drugs.

5. Cases and controls who are willing to participate in the study and who have consented to the study.

EXCLUSION CRITERIA:

1. Cases and controls with current or past medical or neurological illness

2. Patients having mental retardation, dementia and substance dependence.

3. Participants who are ill, exhibits aggressiveness and violent behaviour.

4. Cases and controls who are not willing to participate in the study

INSTRUMENTS:

1. Socio-demographic Proforma.

2. Waldrop scale for Minor physical anomalies

3. Cambridge neurological inventory for soft neurological signs

4. Positive and negative syndrome scale PANSS

5. M.I.N.I. (Mini International Neuropsychiatric Interview)

1. SOCIODEMOGRAPHIC PROFORMA:

Proforma includes personal demographic details, personal history, past history, family history, physical and mental status examination and biochemical investigations.

2. WALDROP SCALE:

This scale is a widely used standard scoring system for the evaluation of minor physical anomalies. It evaluates 6 anatomical regions which includes eyes, ears, mouth, head, hands and feet. It includes assessment of 18 anomalous features and takes 5- 10 min for examination. Each MPA is given weightage ranging from 0-2. The total Waldrop score would give an indication as to the number and severity of MPA present in a subject.

The cutoff score of 3 was chosen because this was the upper limit of normal population in many studies. (Green et al. 1998)[42].

3. CAMBRIDGE NEUROLOGICAL INVENTORY[62]:

It is a bedside neurological evaluation checklist which includes subtle neurological signs in six categories:

1. Motor (including casual gait, tandem gait, Romberg's test)
2. Complex motor coordination (including fist- edge- palm, alternating fist palm, diadochokinesis, finger opposition, rhythm tapping)
3. Extra-ocular movements (visual tracking, gaze persistence)
4. Other motor signs (mirror movements, motor persistence, heel shin test, synkinesis, tremors, and choreoathetosis)
5. Primitive reflexes (glabellartap, palmar grasp, palmomental, snoutreflexes).
6. Sensory Integration (Stereognosis, Graphaesthesia, face–hand extinction, R-L disorientation)

2. POSITIVE AND NEGATIVE SYNDROME SCALE:

It is a 30-item, 7-point (1–7) rating scale. It is a comprehensive and thoroughly standardized scale to assess psychopathology in schizophrenia. It derives from behavioral information plus a 35 to 45 minute clinical interview. It is administered as clinical interview over 1⁄2-an hour, behavioral information is obtained. It has been standardized to assess the psychopathology in schizophrenia. The items in the table are precisely defined also the numerical ratings of each of them. It includes positive subscale, negative subscale and general psychopathology subscales. The subscale scores are found to be independent of each other.

The scale is sensitive and specific when treatment is initiated and the scale is administered at a later date. Its validity has been established by classification of patients according to the predominant symptoms present. Though the subscale score are associated with cognitive, treatment, clinical variables, pre morbid adjustment but not with outcome. The PANSS scores of patients is seen to be consistent over the illness course, one of its major strength.

3. **M.I.N.I (Mini International Neuropsychiatric Interview):**

M.I.N.I (Mini - International Neuropsychiatric Interview) is developed by psychiatrists , based on the psychiatric conditions classified in DSM–IV, and in ICD – 10.

It is a short and structured screening diagnostic interview. The time to administrate this diagnostic tool is approximately fifteen minutes. It can be used as a potential first step tool for the screening of psychiatric disorders. It was designed for the clinical trial needs and an epidemiological study needs and it is an accurate short structured psychiatric interview tool and has better reliability and validity. It is a potential tool for the screening of psychiatric disorders.

OPERATIONAL DESIGN:

This study was conducted at department of Psychiatry OPD , Stanley medical college and hospital after getting approval from institutional ethical committee.

CASE GROUP:

Patients who attended psychiatry OPD diagnosed as schizophrenia as per ICD 10 diagnostic criteria were recruited for study on basis of inclusion criteria.

CONTROL GROUP:

Attenders or relatives accompanying the patients attending medical OPD were chosen as controls. The patients and controls were screened and grouped according to the inclusion and exclusion criteria.

Subjects has been explained about the nature of the study and obtained informed consent. Then semi structured proforma was applied to collect socio demographic information. Positive and negative syndrome scales were administered for all patients to assess the psychopathology.

For controls who satisfied the criteria of both inclusion as well as the exclusion criteria, were screened initially by Mini International Neuropsychiatric Interview,

Then MPA were assessed with Waldrop scale and soft neurological signs assessed with CNI for the patients and controls separately. Likewise 60 subjects were tested.

STATISTICAL ANALYSES:

Statistical analysis will be done using computerized software (SPSS-20). Descriptive statistics like frequencies, percentages, means and standard deviations will be computed. Parametric and non-parametric analysis will be used appropriately depending on the data collected.

FINDINGS AND INTERPRETATION

FINDINGS AND INTERPRETATION

Findings

The survey data analyzed quantitatively yielded the following results. The findings are reported in the following section under socio-demographic characteristics, scores obtained from various scales and different statistical tests to explore the relationships between variables studied.

Socio-demographic characteristics

Age

Figure 1A illustrates the distribution of age between cases and control. A majority of the study population fall in the category of 20-30 years: Case 51.7% and control 46.7%

Figure 1A: Age distribution among cases and control

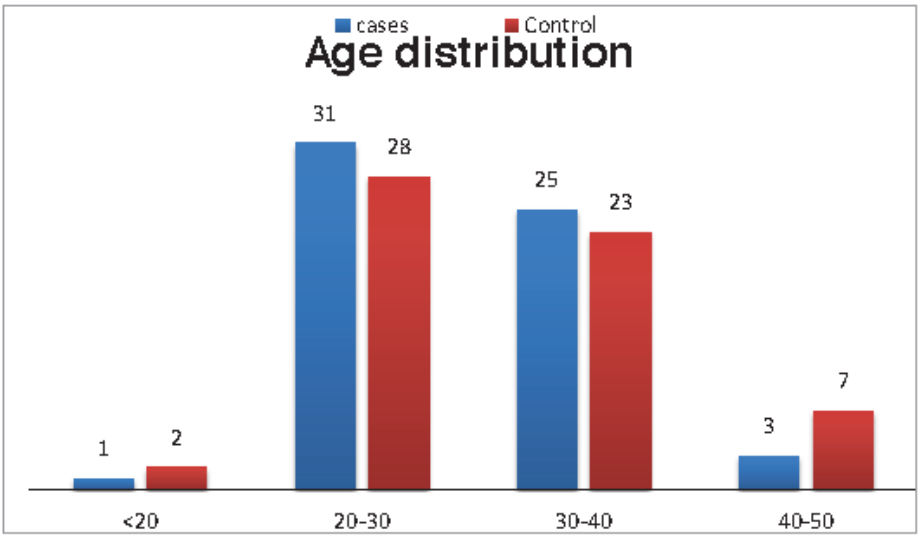
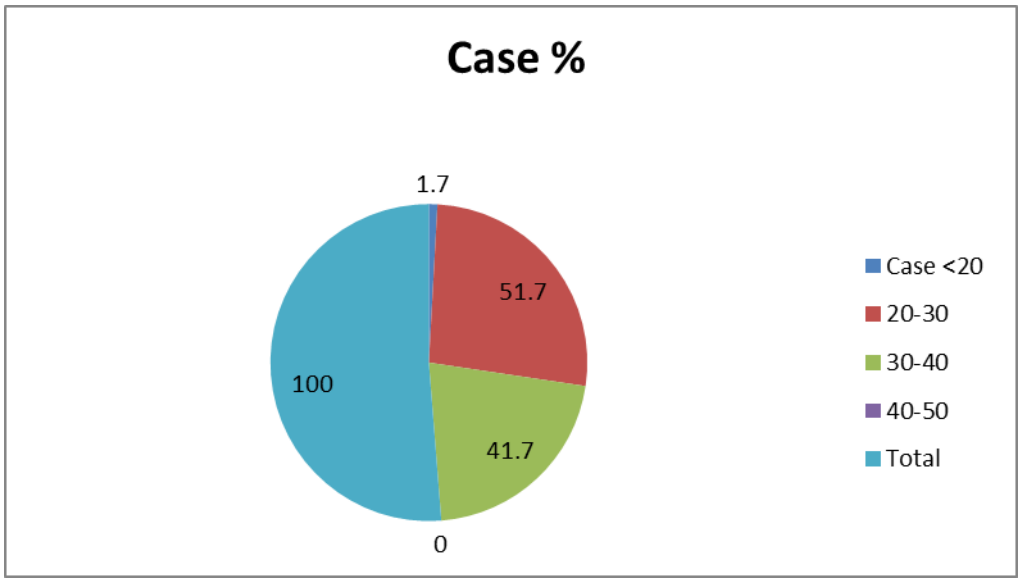


Figure 1B: Age distribution among cases



Majority of Schizophrenic patients in our sample fall in the category of 20-30 years (Case: 51.7% and Control: 46.7%)

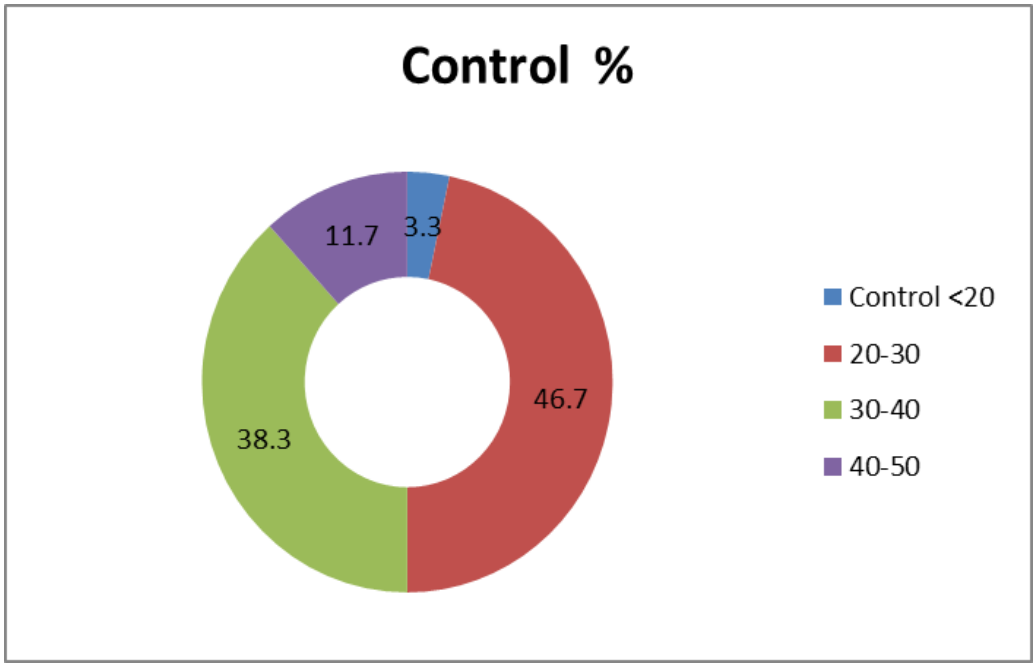
Among the 60 cases, 1 (1.7%) Belong to <20 years,

31(51.7%) Belong to 20-30 age group,

25(41.7%) Belong to 30-40 age group and

3(5%) Belong to 40-50 age group.

Figure 1C: Age distribution among Controls



Among the 60 controls

2(3.3%) belong to <20 years,

28(46.7%) belong to 20-30 age group,

23(38.3%) belong to 30-40 age group and

7(11.7%) belong to 40-50 age group.

Gender distribution

The following image depicts the gender distribution of the cases and control with a preponderance to males in cases and the inverse in the control group.

Figure 2A: Gender distribution cases and control

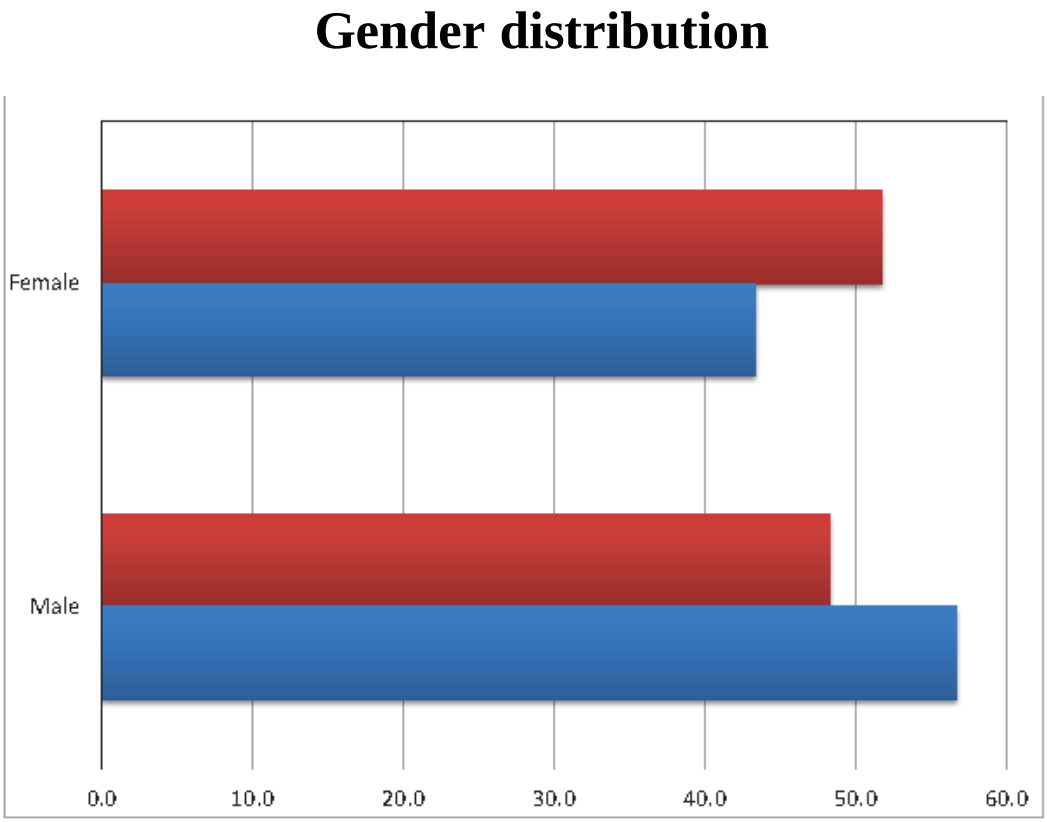
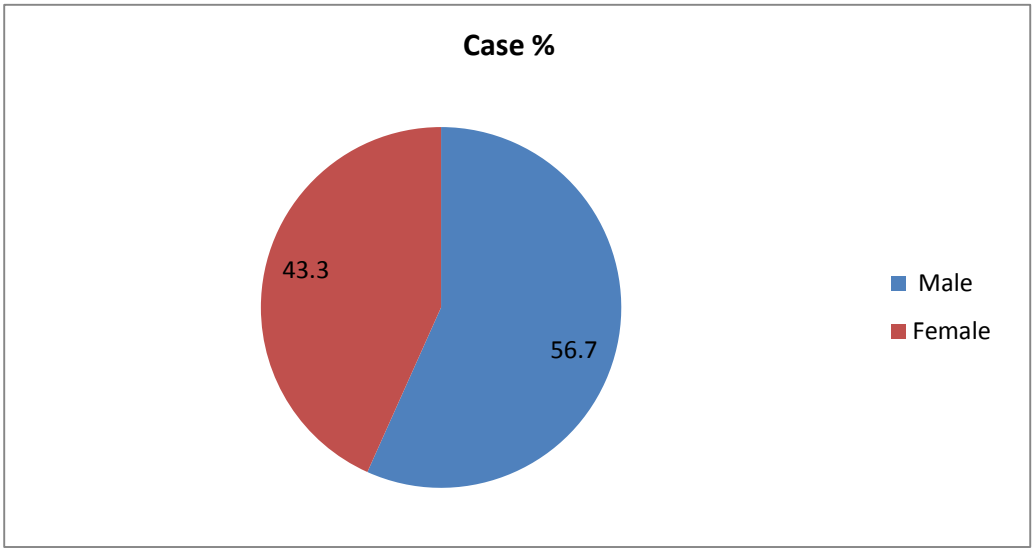
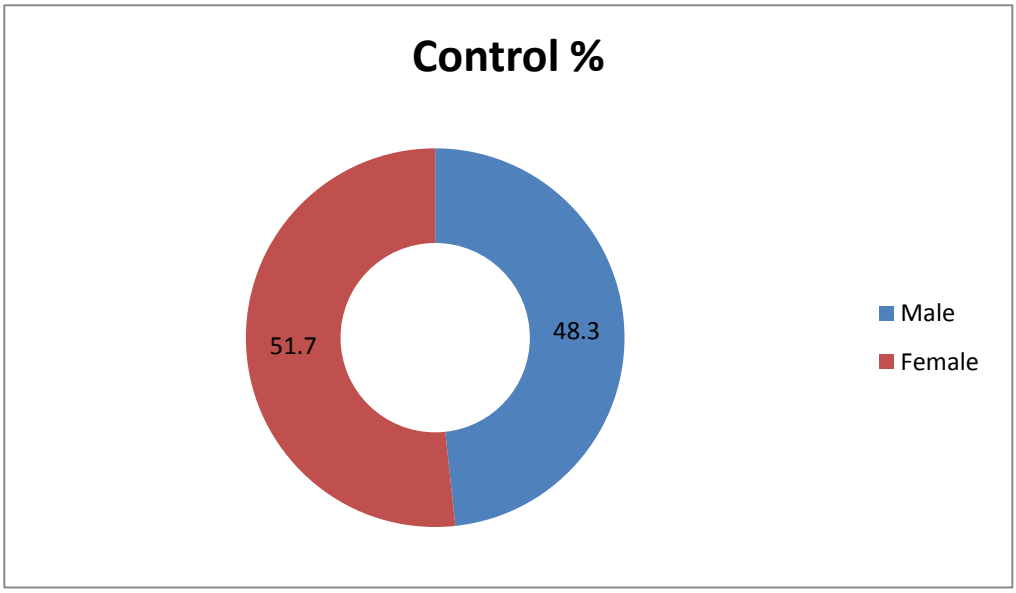


Figure 2B: Gender distribution among cases.



Among the 60 cases, male constituted 56.7% (34) and female constituted 43.3% (26)

Figure 2C: Gender distribution among control.



Among the 60 controls, male constituted 48.3% (29) and female constituted 51.7% (31).

Education

Around 42% of the cases and 43% of the controls have an education less than 5th standard. Only a small number, 6.7% in cases and 3.3% in controls have an education of 11th -12th. Table 1 shows the level of education of the participants in cases and control.

Table 1: Level of education of the participants in cases and control.

EDUCATION			Frequency	Percent
Case		illiterate	2	3.3
		<5thstd	25	41.7
		6-8thstd	21	35.0
		9-10thstd	8	13.3
		11-12thstd	4	6.7
		Total	60	100.0
Control		<5thstd	26	43.3
		6-8thstd	24	40.0
		9-10thstd	8	13.3
		11-12thstd	2	3.3
		Total	60	100.0

Figure 3A: Level of Education among case and control

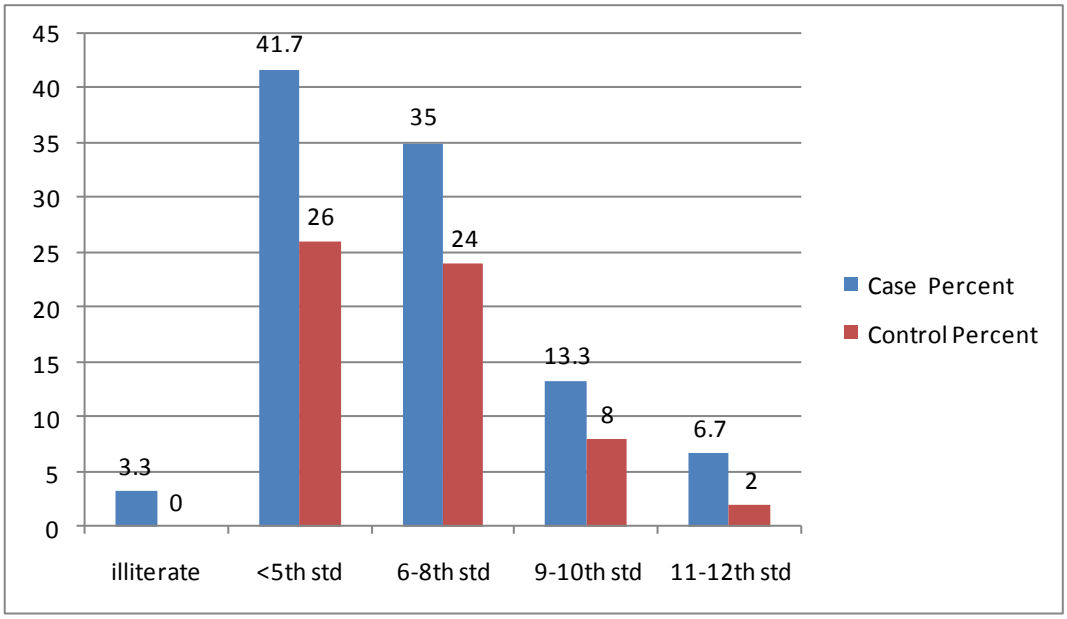
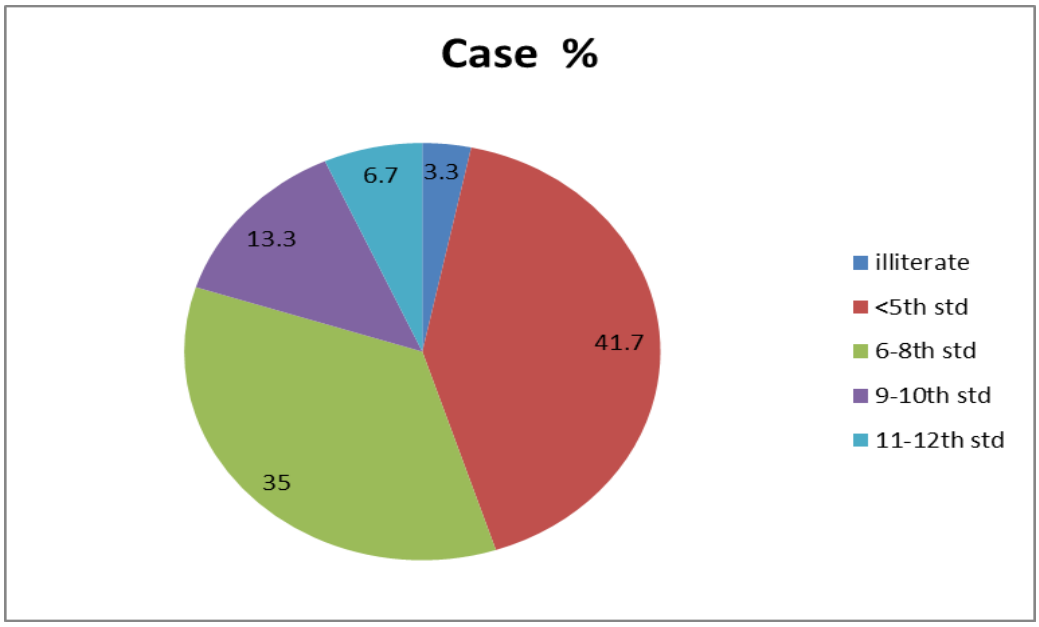


Figure 3B: Level of Education among cases.



Among the 60 cases:

41.7% (25) has education <5th Std.

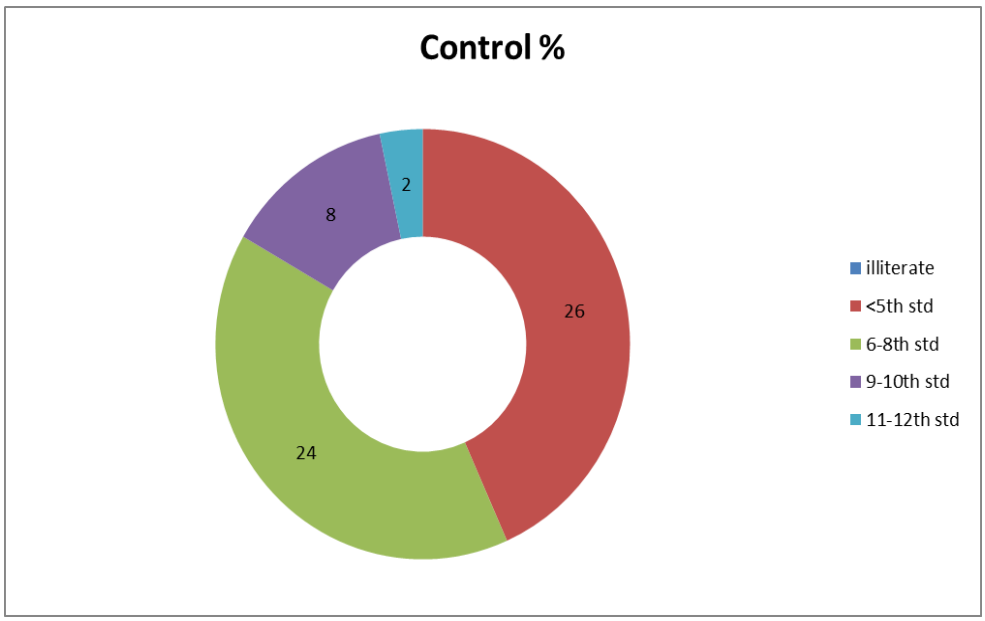
35% (21) of the patients had education of 6th to 8th Std.

13.3% (8) had education up to 9th to 10th Std.

6.7% (4) had education between 11th to 12th Std.

3.3% (2) were illiterate.

Figure 3C: Level of Education among controls.



Among the 60 controls:

43.3% (26) had only education up to 5th Std.

40% (24) had education of 6th to 8th Std.

13.3% (8) had education up to 9th to 10th Std.

3.3% (2) had education between 11th to 12th Std.

Socio-economic status:

Most of the participants were from the lower socioeconomic status (n=72). A 61.7% of the cases belong to the lower socioeconomic status against 58.3% of the control.

Figure 4A: Shows the socioeconomic status of the participants in the two study groups.

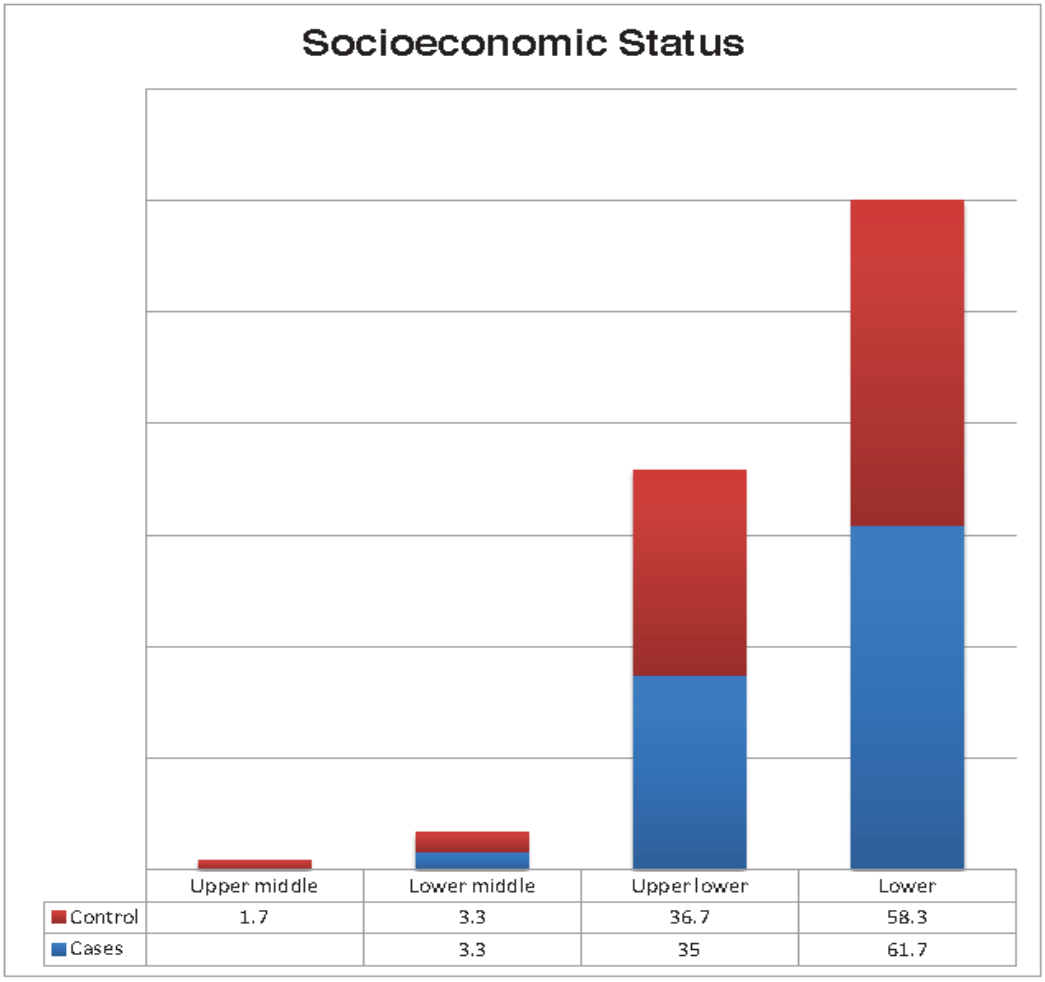
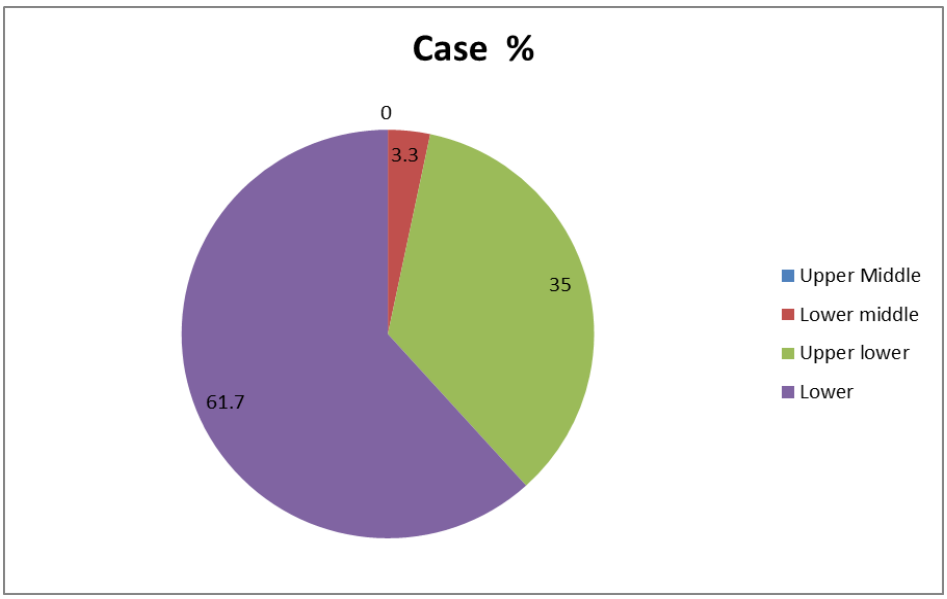


Figure 4B: Socioeconomic status of the participants among cases.



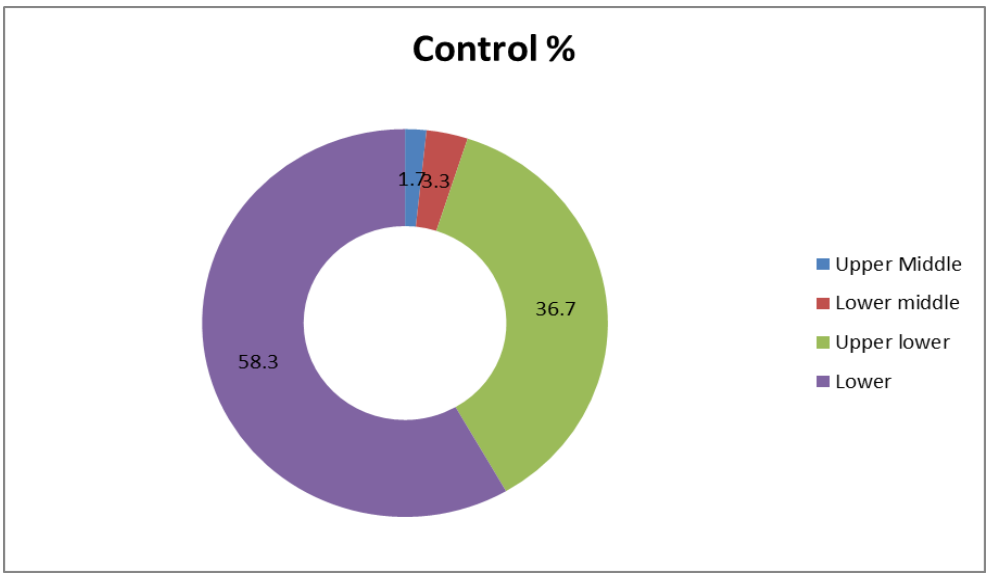
Among the cases:

61.7% (37) of them belong to lower Socio Economic Status.

35% (21) of them belong to upper lower SES.

3.3% (2) of them belong to lower middle SES.

Figure 4C: Socioeconomic status of the participants among controls.



Among the control:

58.3% (35) of them belong to lower SES.

36.7% (22) of them belong to upper lower SES.

3.3% (2) of them belong to lower middle SES.

1.7% (1) of them belong to Upper middle SES.

Occupation:

The following table displays the occupation of the participants with no particular pattern noted. There is a near equal distribution among cases and controls across all occupation.

Fig 5A: Occupation of the participants in the two study groups.

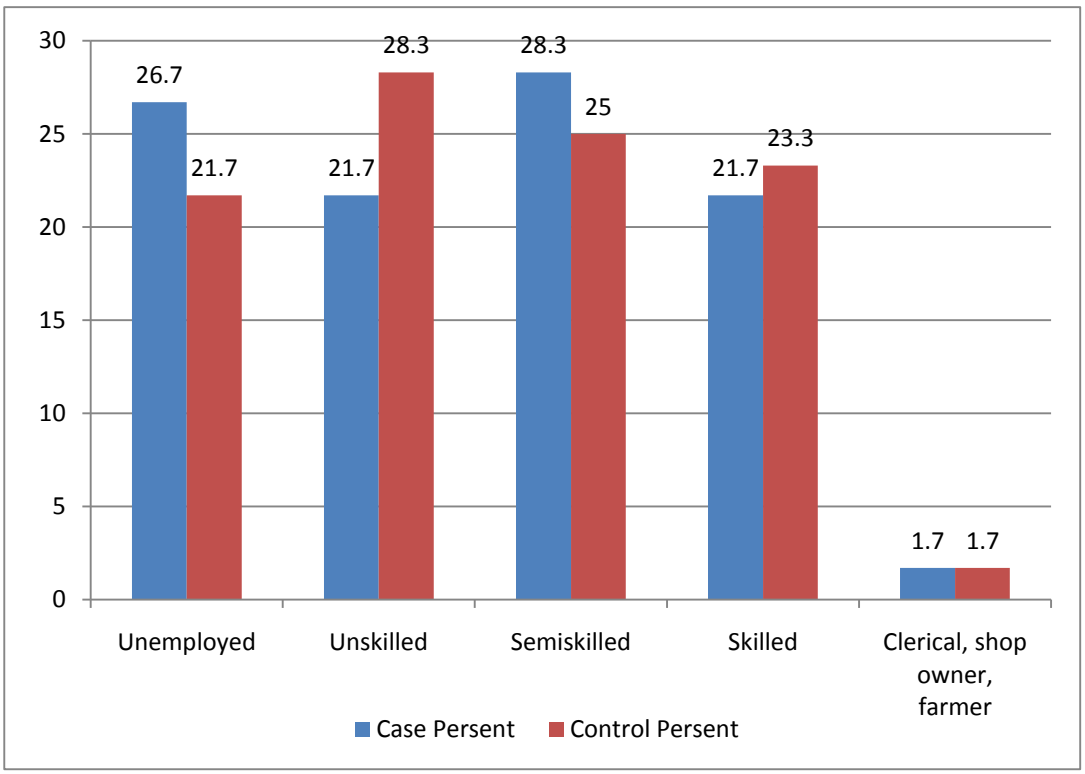


Table 2: Occupation of the Cases

		OCCUPATION	Frequency	Percent
Case		Unemployed	16	26.7
		Unskilled	13	21.7
		Semiskilled	17	28.3
		Skilled	13	21.7
		Clerical, shop owner, farmer	1	1.7
		Total	60	100.0

Fig 5B: Occupation of the participants among cases.

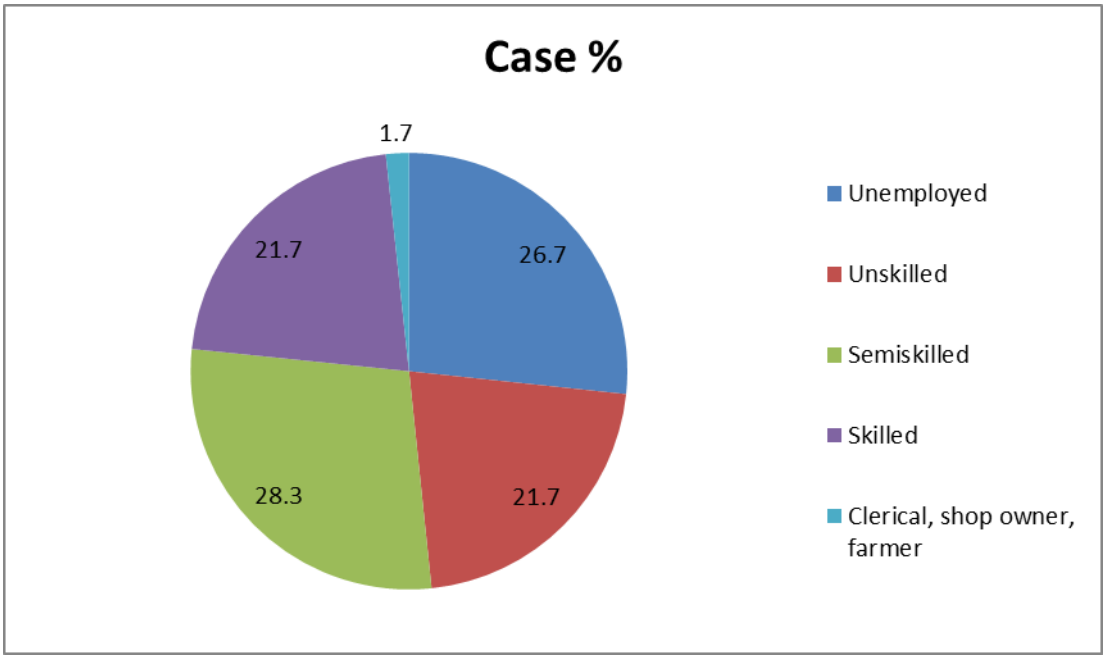
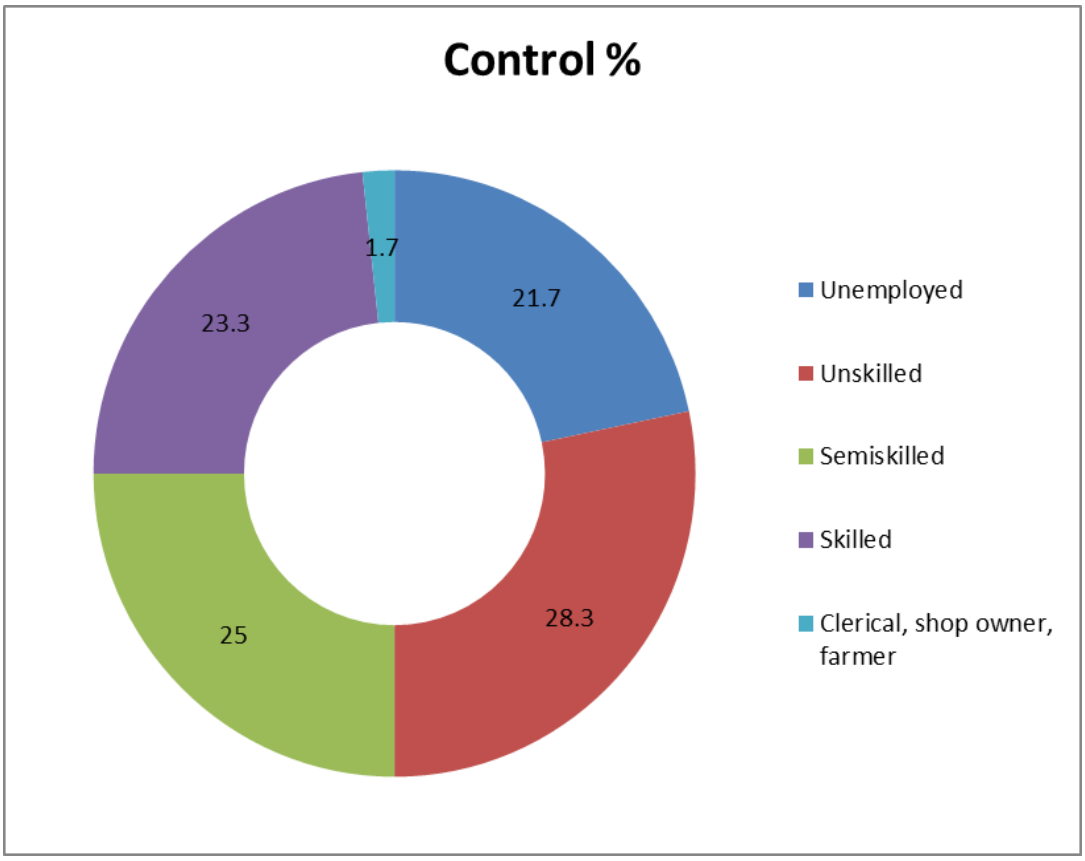


Table 3: Occupation of the participants among controls.

Control		Unemployed	13	21.7
		Unskilled	17	28.3
		Semiskilled	15	25.0
		Skilled	14	23.3
		Clerical, shop owner, farmer	1	1.7
		Total	60	100.0

Fig 5C: Occupation of the participants among controls.



Marital status

Most of our participants were married. Of the 60 participants of each group, 61.7% were married in the cases group compared to 53.3% of the control group. This is followed by unmarried people while a small number is constituted by widows and those married and separated.

Figure 6A: Marital status of the participants in the two study groups.

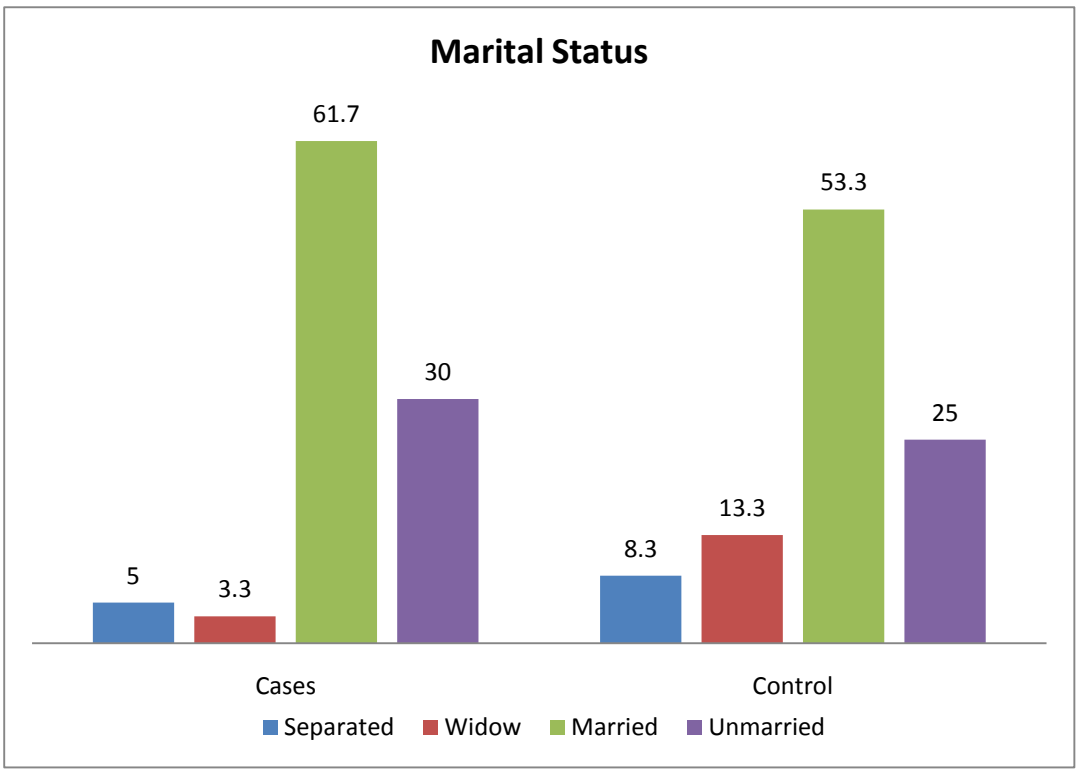
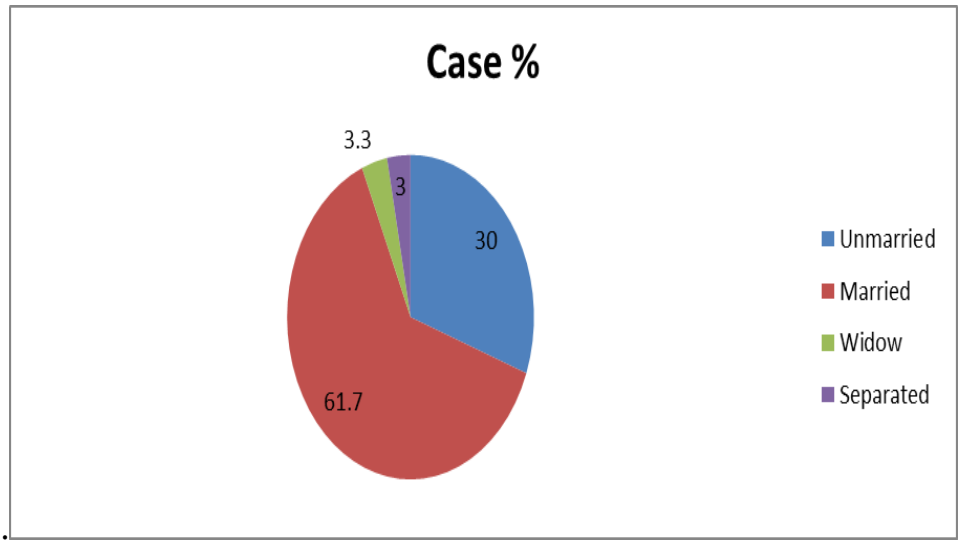
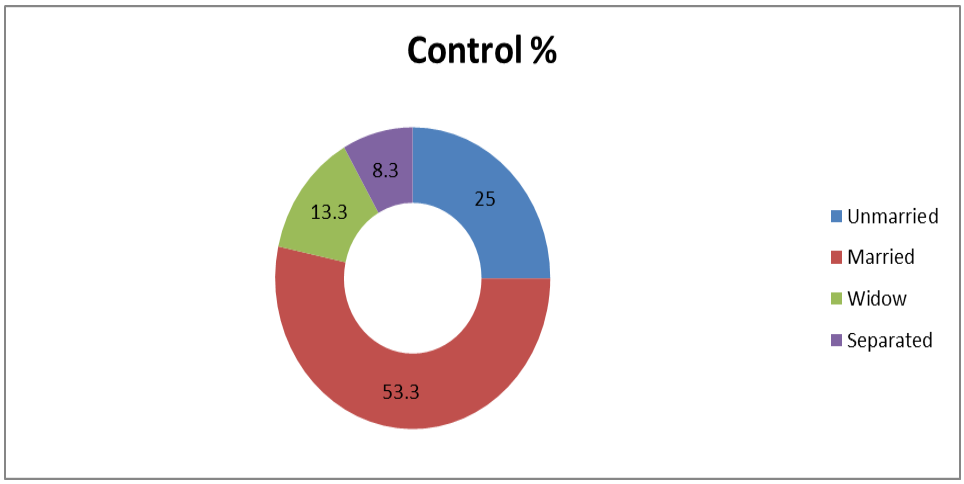


Figure 6B: Marital status of the participants among cases



Among the 60 cases:
Unmarried - 30% (18)
Married - 61.7% (37)
Widow - 3.3% (2)
Separated - 5% (3)

Figure 6C: Marital status of the participants among controls.



Among the 60 controls:
Unmarried -25% (15)
Married - 53.3% (32)
Widow - 13.3% (8)
Separated - 8.3% (5)

Residence

A majority of our participants came from urban and semi-urban areas. Table 4 & 5 shows the distribution of the residence between cases and control. There is no significant difference between the two groups.

Figure 7A: Residence of the participants in the two study groups.

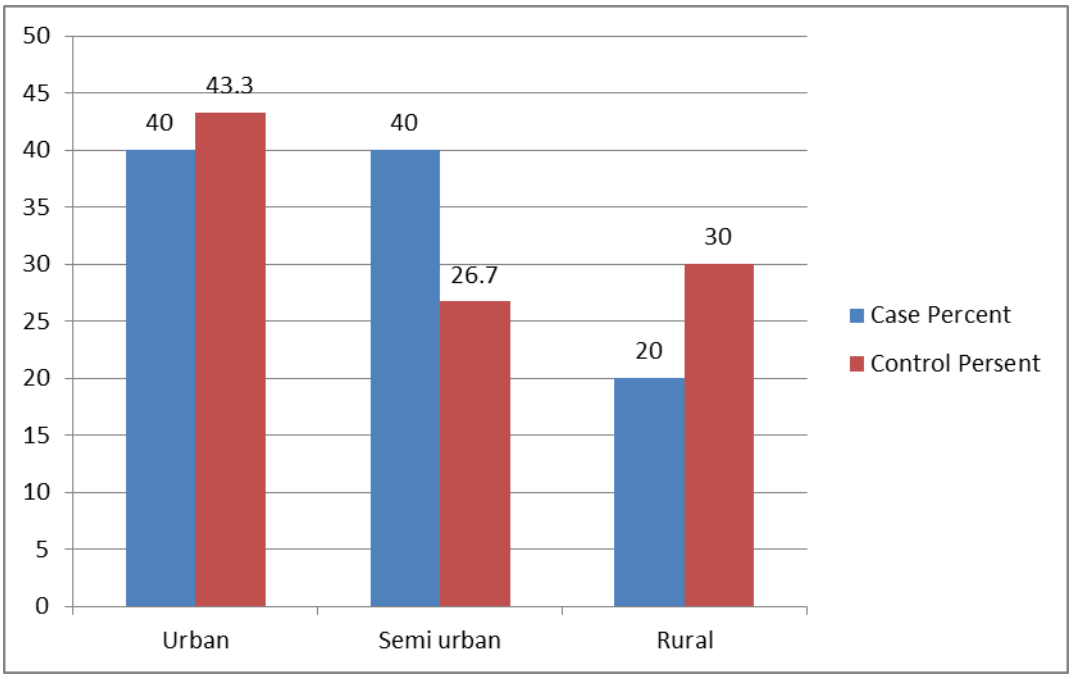
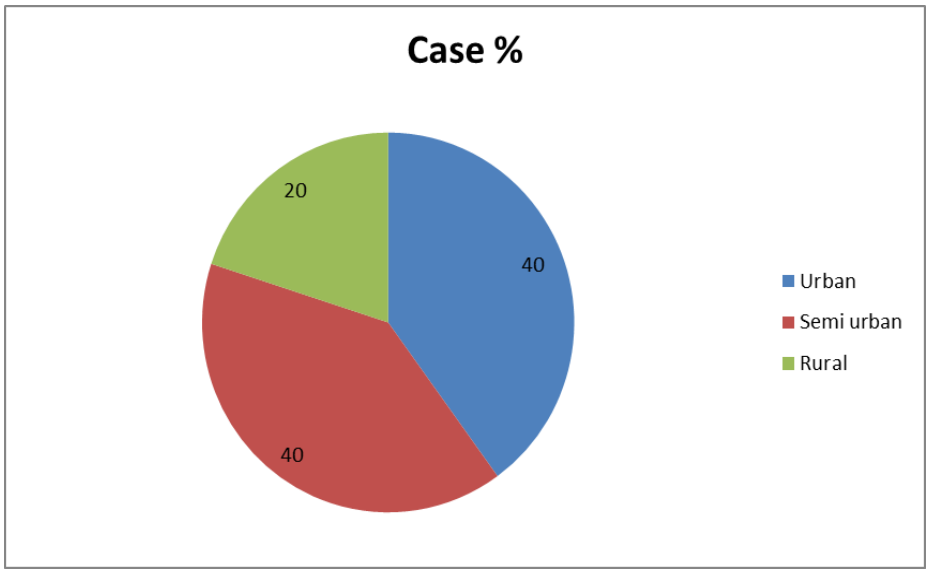


Table 4: Residence of the participants among cases

RESIDENCE			Frequency	Percent
Case		Urban	24	40.0
		Semi urban	24	40.0
		Rural	12	20.0
		Total	60	100.0

Figure 7B: Residence of the participants among cases.



Among the 60 cases:

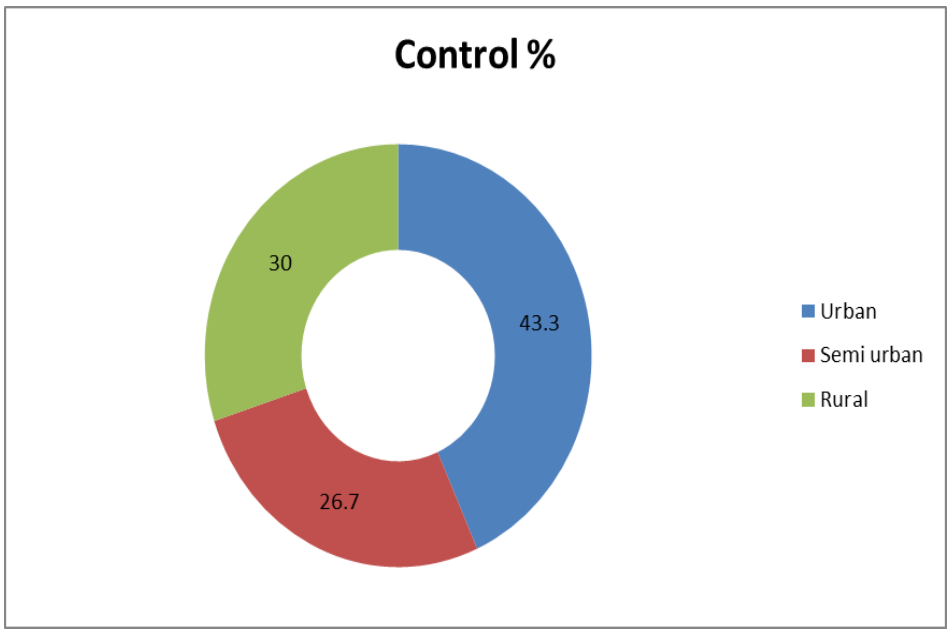
40% (24) were from Urban and Semi- Urban area.

20% (12) were from rural area

Table 5 : Residence of the participants among controls

Control		**Urban**	**26**	**43.3**
		Semi urban	16	26.7
		Rural	18	30.0
		Total	60	100.0

Figure 7C: Residence of the participants among controls.



Among the 60 controls:

43.3% (26) were from Urban

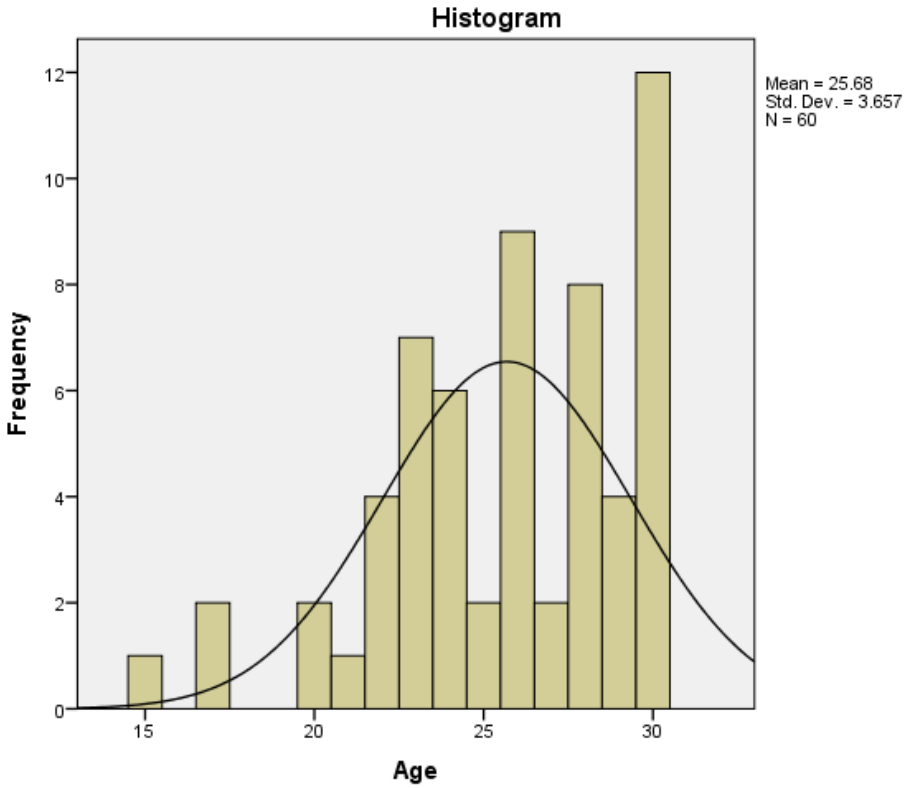
26.7% (16) were from Semi- Urban area.

30% (18) were from rural area

Age of onset of Schizophrenia

For the cases N=60, the mean age of onset of schizophrenia is 25.68 years. The following histogram shows the distribution of age of onset in the cases.

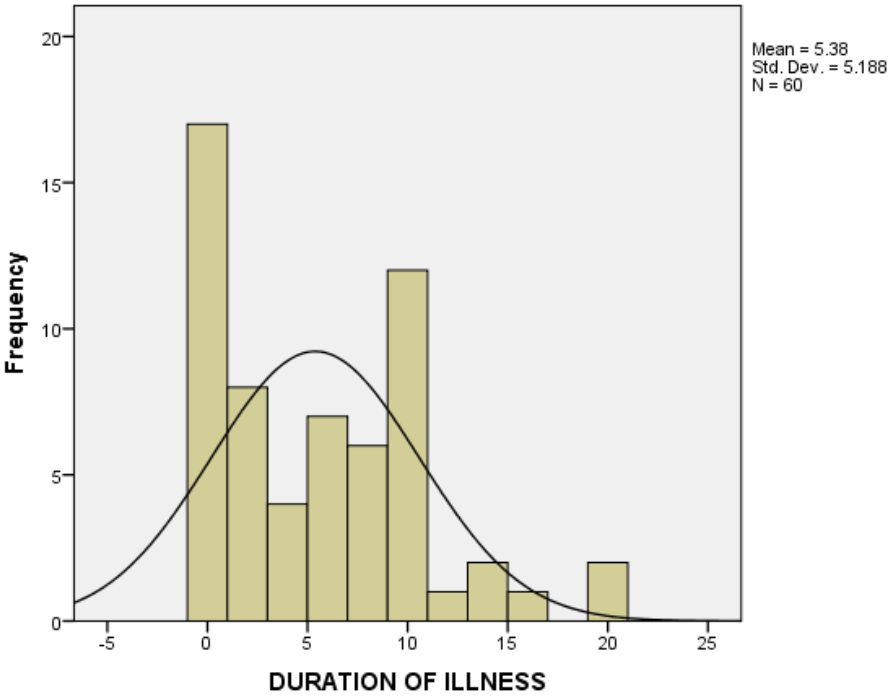
Figure 8: Mean age of onset of schizophrenia



Duration of illness

Seventeen participants had the first episode of schizophrenia whereas 43 of them had their first episode already. The mean duration of the illness is 5.38 years with a standard deviation of 5.19. The mean duration of illness is depicted in the following histogram.

Figure 9: Duration of illness



Among the 60 cases:

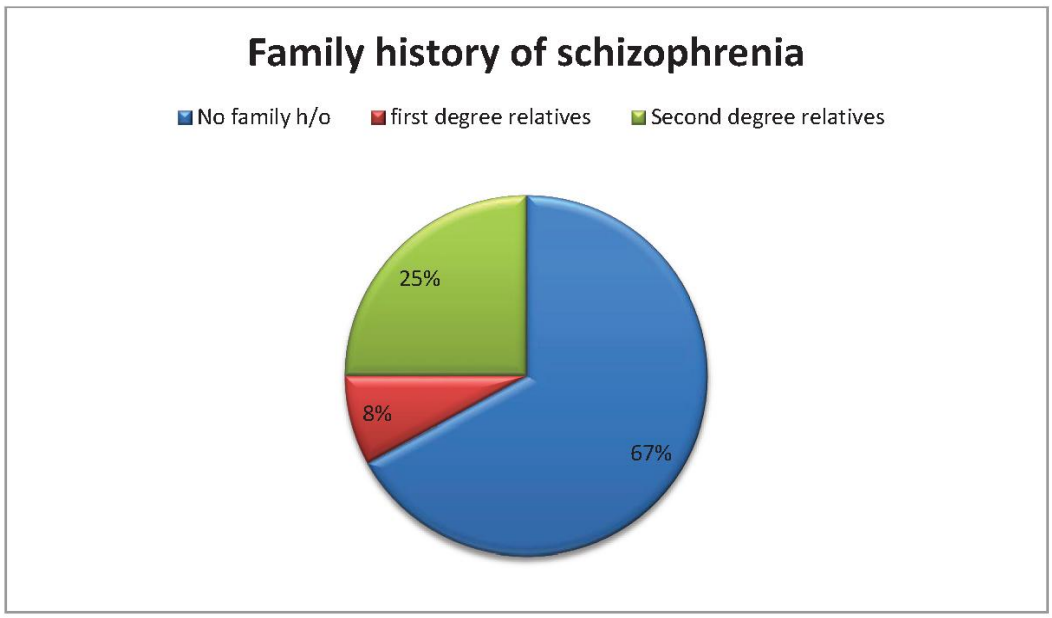
17 cases (28.3%) were first episode, drug naïve.

43 cases (71.7%) had variable duration of illness (11.7% has 9 years duration of illness, 10% has 8 years duration, 8.3% has 10 years duration and 6 years duration of illness, 6.7% has 1 year and 2 years duration of illness and 5% has 4 years duration of illness).

Family history of schizophrenia

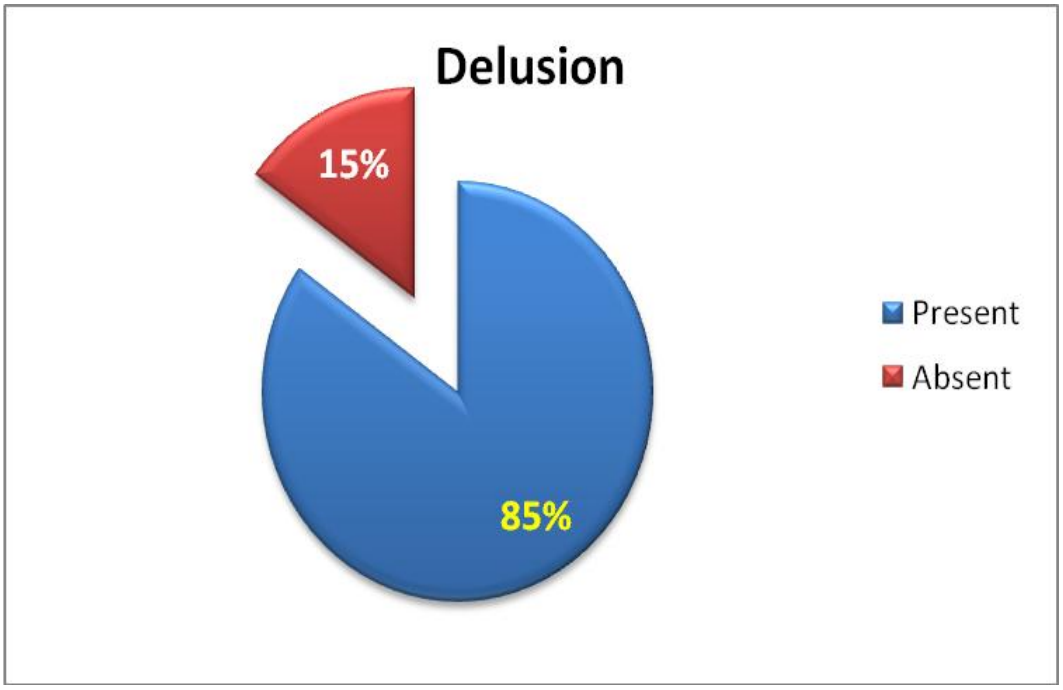
A majority of the cases did not have a family history of schizophrenia whereas 25% of them had second degree relatives and 8% of them had first degree relatives**.**

Figure 10: Family history of schizophrenia



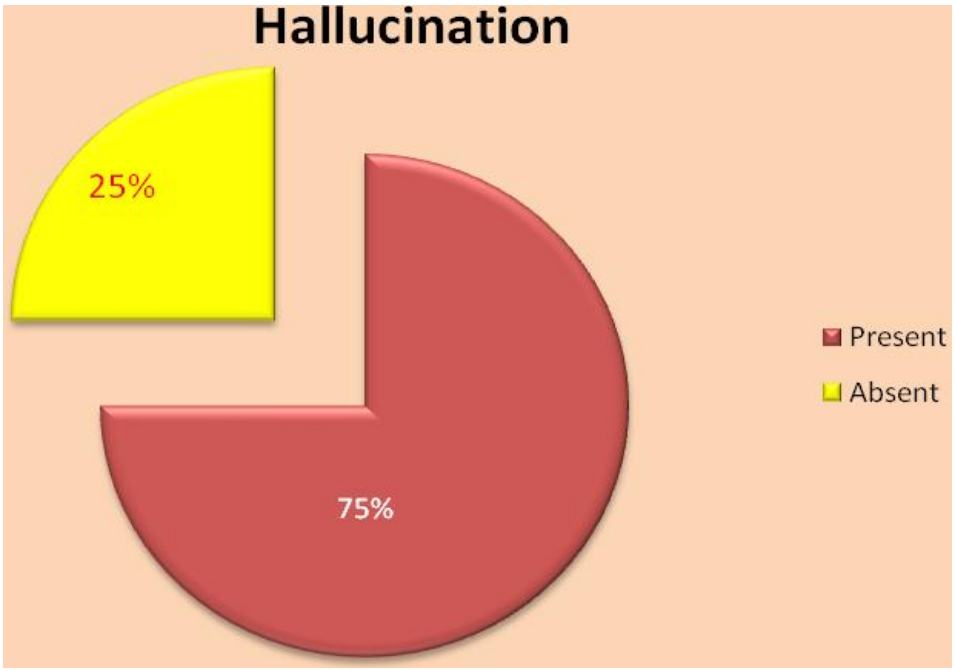
Delusion and Hallucination : Figure 11(A&B) show the prevalence of delusion and hallucination in the study sample.

Figure 11A: Prevalence of delusion



Among 60 cases, 85% had Delusion (falling within minimal, mild, moderate, moderately severe, severe and extreme categories) and 15% had no (absent) Delusion.

Figure 11B: Prevalence of Hallucination



Hallucination: Among 60 cases, 75% had Hallucination (falling within minimal, mild, moderate, moderately severe, severe and extreme categories) and 25% had no (absent) Hallucination.

PANSS Mean scores

Table 6 gives the mean scores of different components of PANSS of the cases. Positive symptoms, Mean=13.97, Negative symptoms, Mean=13.25 and General Psychopathology, Mean=21.83.

Table 6: Mean scores of schizophrenics on PANSS

PANSS Mean scores of schizophrenics				
Statistics		**POSITIVE**	**NEGATIVE**	**GEN. PSY**
	N	60	60	60
	Mean	13.97	13.25	21.83
	Std. Deviation	3.914	4.169	5.133

Minor Physical Anomalies

The following table shows the frequency of different minor physical anomalies in schizophrenics and controls. The frequency distribution shows the predominance of craniofacial anomalies compared to hands and feet.

Table 7: Frequency of Minor Physical Anomalies

Anomaly			Schiz	Control
Head	Fine Electric hair	Very Fine Hair	1 (1%)	0(0%)
		Soon Awry	3 (3%)	0(0%)
	Two or More Hair Whorls		2(2%)	1(4.7%)
	Head Circumference	>1.5 SD	3 (3%)	2(8.3%)
		>1.0<1.5 SD	5 (6%)	2(8.3%)
Eyes	Epicanthus	Deeply Covered	1 (1%)	0(0%)
		Partly Covered	3 (3%)	0(0%)
	Hypertelorism	>1.5 SD	4 (4%)	2(8.3%)
		>1.0<1.5 SD	7 (8%)	2(8.3%)
Ears	Low Seated Ears	Lower by>0.5 cm	2(2%)	2(8.3%)
		Lower by<0.5 cm	6(8%)	2(8.3%)
	Adherent ear lobule	Lower edge up and back towards the crown of head	4 (4%)	3(12.5%)
		Straight back to-ward rear of the neck	8(9%)	1(4.7%)
	Malformed ear		0(0%)	0(0%)
	Asymmetrical ear		2(2%)	0(0%)
	Soft and pliable ear		0(0%)	0(0%)
Mouth	High steepled palate	Definitely steepled	10(11.4%)	2(8.3%)
		Flat and narrow at the top	7 (8%)	1(4.7%)
	Tongue	Furrowed tongue	3 (3%)	2(8.3%)
		Tongue with Rough and smooth spots	0(0%)	0(0%)

Anomaly			Schiz	Control
Hands	Curved fifth finger	Markedly curved towards other finger	1 (1%)	0(0%)
		Slightly curved towards other finger	3 (3%)	0(0%)
	Single transverse palmar crease		0(0%)	0(0%)
Feet	Third toe longer than second	Definitely longer than the second toe	2(2%)	
		Appear equal in length to second toe	6(8%)	
	Partial syndactyly of two middle toes		0(0%)	
	Big gap between first and second toe		4 (4%)	2(8.3%)

Comparison of the participants with lesser minor physical anomalies and more than three anomalies between cases and controls.

Table 8: Minor Physical Anomalies scores <3 ,>3

	<3	>=3
Cases	41(68.3%)	19 (31.7%)
Control	11 (18.3%)	4 (6.7%)

Minor physical anomalies are more in Schizophrenics with 68.3% of them having less than three anomalies compared to control that have only 18.3%. The control group did not have more than three anomalies as opposed to cases that had 31.7% [Table 8]. The above table shows the frequency of minor physical anomalies divided between two groups with less than and greater than three anomalies.

Soft Neurological Signs in cases:

Table 9: Presence of soft neurological signs in cases

SOFT NEUROLOGICAL SIGNS	N=60
Present	45
Absent	15

Non parametric Statistical analysis of Minor physical anomalies scores between two groups using Mann Whitney test

Table 10: Non parametric Statistical analysis of minor physical anomalies scores between two groups using Mann Whitney test

Anomaly	z	p
Total Minor physical anomalies	-0.203	.839
Head	-3.332	**.001**
Eyes	-2.749	**.006**
Ears	-2.333	**.020**
Mouth	-3.671	**.000**
Feet	-2.310	**.021**

Table 10 shows that the two groups statistically differ inMinorphysical anomaliesscores which indicate that Minor physical anomalies are more in schizophrenics. The value of $p<0.05$ is considered to be significant. In the above non-parametric analysis, the derivation shows that Minor physical anomalies scores in head, eyes, ears, mouth and feet are significantly different in cases and control.

Correlation between Minor physical anomalies (MPA>3) and PANSS

Bivariate analysis of Minor physical anomalies and PANSS reveals no significant correlation between them. The value of $p<0.05$ was considered to be statistically significant.

Table 11: Correlation tests between Minor physical anomalies and PANSS

Variables	r	P
Minor physical anomalies and Positive	0.077	0.754
Minor physical anomalies and Negative	-0.024	0.924
Minor physical anomalies and Gen psy	0.865	0.042

Soft neurological signs

The following table shows the frequency and percentage of soft neurological signs of cases and control. Motor coordination signs predominated in both schizophrenics (53.3%) and control (16.7%).

Table 12: Soft neurological signs frequencies and percentages

Sign	Frequency / percentage	Case n=60	Control n=60	Chi-square p-value
Motor	**n**	**17**	**1**	0.092
	%	**28.3**	**1.7**	
Motor coordination	**n**	**32**	**10**	**0.000**
	%	**53.3**	**16.7**	
EOM	**n**	**17**	**4**	**0.010**
	%	**28.3**	**6.7**	
Others	**n**	**26**	**9**	**0.002**
	%	**43.3**	**15**	
Primitive reflexes	**n**	**13**	**0**	**0.002**
	%	**21.7**	**0**	
Sensory integration	**n**	**12**	**0**	**0.000**
	%	**20**	**0**	

Correlation tests show that cases and controls statistically differ in the following soft neurological signs: Motor coordination, Extra ocular movements, other motor signs, primitive reflexes, and sensory integration with p value <0.05.

Table 13: Distribution of Soft Neurological Signs among males and females

Sign	Frequency / percentage	Males=39	Females=28
Motor	**n**	**11**	**7**
	%	**17.5**	**12.3**
Motor coordination	**n**	**24**	**18**
	%	**38.1**	**31.6**
EOM	**n**	**11**	**10**
	%	**17.5**	**17.5**
Others	**n**	**22**	**13**
	%	**34.9**	**22.8**
Primitive reflexes	**n**	**8**	**5**
	%	**12.7**	**8.8**
Sensory integration	**n**	**10**	**2**
	%	**15.9**	**3.5**

Table 14 and Table 15 shows the mean scores of Soft Neurological signs in cases with drug naive first episode schizophrenia and those with a patients under treatment. The mean score is significantly higher in schizophrenics with first episode of Schizophrenia.

Table 14: Mean scores of Soft Neurological Signs in drug naïve patients

First Episode drug naive	N		17
			0
	Mean		4.12
	Median		5.00
	Std. Deviation		2.870
	Range		10

Table 15: Mean scores of Soft Neurological Signs in schizophrenic patients on drugs

Patients under treatment	N		43
			0
	Mean		3.07
	Median		2.00
	Std. Deviation		3.247
	Range		11

Relationship between age of onset of schizophrenia and Soft Neurological Signs

Table 16: Relationship between age of onset of schizophrenia and Soft Neurological Signs

r	**-0.071**
P	0.635

The correlation tests between mean age of onset of schizophrenia and Soft Neurological Signs shows that there is a negative relationship between them. But p value does not show any statistically significant results (significant $p<0.05$) [Table 16].

Relationship between Minor Physical Anomalies and Soft Neurological Signs

The correlation tests between Minor Physical Anomalies and Soft Neurological Signs show that there is a positive relationship between them. The value of p value show statistically significant results (significant $p<0.05$) [Table 17]. There is a significant correlation between Minor Physical Anomalies and Soft Neurological Signs with $p=0.001$.

Table 17: Relationship between Minor Physical Anomalies and Soft Neurological Signs

r	**0.444**
P	0.001

Relationship between PANSS and Soft Neurological Signs

The correlation tests between PANSS and Soft Neurological Signs show that there is a positive relationship between them. The value of p value show statistically significant results (significant $p<0.05$) [Table 18].

Table 18: Correlation between Soft Neurological Signs and PANSS

Variables	p
Soft Neurological Signs and Positive	0.067
Soft Neurological Signs and Negative	0.056
Soft Neurological Signs and Gen psy	0.092
Soft Neurological Signs and Hallucination	**0.036**
Soft Neurological Signs and Delusion	**0.042**

The two groups statistically differ in delusion and hallucination with p value <0.05. There is a positive correlation between SNS and individual symptoms - delusion and Halluciation (p value <0.05)

Relationship between different variables under study (only cases)

The correlation tests between different variables show that there is a no statistically significant relationship (significant $p<0.05$) [Table 19].

Table 19: Correlation tests between different variables

Variables	P	Significant/ Not significant
Minor Physical Anomalies & delusion	0.624	Not significant
Minor Physical Anomalies & hallucination	0.565	Not significant

Correlation tests to see the relationship between age of onset and Soft Neurological Signs.

Table 20: Correlation tests to see the relationship between age of onset and Soft Neurological Signs.	
r	-.105
p	.481

There is a negative correlation between age of onset and Soft Neurological Signs but it is not statistically significant.

Table 21: Correlation tests to see the relationship between age of onset and Minor Physical Anomalies

r	-.065
p	.622

There is a negative correlation between age of onset Minor Physical Anomalies but it is not statistically significant.

DISCUSSION

DISCUSSION

In the present study of 60 cases of schizophrenia and 60 subjects as control, findings were obtained after analysis. The following section discusses the salient features of the results, relevant connections with the existing literature, claims and corresponding justifications to support the claims. Alternate and rival explanations are also discussed with the findings that were developed contrary to the existing evidence.

The case control study helps us to compare and contrast various features between the two groups. For instance, this particular study helps us to understand the minor physical anomalies and soft neurological signs in the schizophrenics as opposed to the control subjects.

It is essential to understand the characteristics of the sample. The age groups of the participants are comparable with only minor variations in the distributions between cases and control. This has relevance in the light of comparative study. This is true in case of distribution of gender too. The education level of the participants is not more than high school, which can be attributed to the profile of cases that visits the government tertiary care center. There are no professionals among the study participants and most of them come from lower socioeconomic status. Urban population is more in our study owing to the location of the study in the urban setting.

Systematic analysis of the previous studies suggests that schizophrenic patients are associated with abnormal prenatal development and the presence of excess physical anomalies were attributed to the abnormal development of CNS. Ismail *et al*, 1998, McNeil*et al*, 2000 studied schizophrenics along with controls.

The association of minor physical anomalies in schizophrenia and control and the prevalence of these anomalies among the patients has been studied by Compton MT Walter and more than 14 studies have compared the scores between the cases and normal population.

Previous Indian studies show that there is an increased incidence of minor physical anomalies in schizophrenics. Scores greater than 3 is significant (Green et al). In our study, 19cases had greater than three anomalies. But 4 control was found to have greater than 3 anomalies. Other studies also substantiate this claim (Lohr and Flynn 1993; Green et al. 1994; O'Callaghan et al. 1995). Table 8 shows that the two groups statistically differ in minor physical anomalies scores, which indicate that minor physical anomalies are more in schizophrenics. The value of $p<0.05$ is considered to be significant. In the non-parametric analysis, the derivation shows that minor physical anomalies scores in head, eyes, ears, mouth and feet are significantly different in cases and control.

Ismail et al 2000 and Compton et al 2007 found that minor physical anomalies were not just limited to the craniofacial region. The current study

substantiates this claim by the prevalence of anomalies of hand and feet too [table 7]. And there is a significant difference between cases and control.

Waddington et al. (1999 showed that craniofacial anomalies dominate the picture of minor physical anomalies which is attributed to the intimate developmental relationship between the craniofacial region and fetal midline and temporal lobe brain structures. Further our study shows that craniofacial anomalies are more in number than hand and feet [table 7].

Minor physical anomalies have not been associated with positive or negative symptomatology of schizophrenia (Compton MT, Bollini AM, Mack LM, Kryda AD, Rutland J, Weiss PS, et al). In our study, bivariate analysis of minor physical anomalies and PANSS reveals no significant correlation between them [Table 11] similar to a study by Dazzan.

Existing knowledge on schizophrenia showed increased neurological signs in the cases (coxludwig et al 1994). Anwar et al 2000 found there is a high prevalence of Soft Neurological Signs in patients with schizophrenia independent of their respective ethnic and socioeconomic groups. The present research also augments the evidence in support of this statement. Table 12 shows that soft neurological signs are more in cases and Motor coordination signs predominated in both schizophrenics (53.3%) and control (16.7%). This has been noted in previous studies by Gourion et al 2004: Mechri et al 2009 which also showed greater Motor coordination signs.

Arango et al 2000 says that soft neurological signs appears early in illness. They do not appear to be secondary to medications and can be reliably

measured. Table 14 and Table 15 shows the mean scores of soft neurological signs in cases with drug nave first episode of schizophrenia and those who were on drugs. The mean score is significantly higher in schizophrenics with drug name first episode of Schizophrenia.

There is no positive association between positive symptoms and soft neurological signs as shown by studies like Arango, Kirkpatrick and Buchana. The correlation tests in our study between PANSS and Soft Neurological Signs show that there is no statistically significant results (significant $p<0.05$) [Table 18]. Total soft neurological signs score was associated with negative scale and disorganisation symptoms scale in a study by Arikan et al. This is contradictory to our study that can be attributed to the difference in the sample and a smaller sample size.

Keshavan et al have found the first episode drug naive patients with schizophrenia showing higher score in sensory integration area. Schroder also found antipsychotic naive has more neurological signs than on treated patients. In another study by Venkadasubramaniam et al 2003 found higher soft neurological signs in never treated patients and their lack of association with illness duration. Table 14 and Table 15 supports this claim showing a higher incidence of soft neurological signs in drug naïve patients. The correlation tests between mean age of onset of schizophrenia and soft neurological signs shows that there is a negative relationship between them. But p value does not show any statistically significant results (significant $p<0.05$) [Table 20].

John et al (2008) noted that subjects with schizophrenia had significantly higher frequency of minor physical anomalies and soft neurological signs compared to healthy controls. This is substantiated in our study [Table 7 and Table 12]. Nizamie et al study showed a positive relationship between soft neurological signs and minor physical anomalies in acute and chronic schizophrenics.

In a study by Paulsen and O'Donnel found a positive relationship between the minor physical anomalies and soft neurological signs. Waldrop and Halverson (1971) and Marcus et al. (1985) recorded a modest positive relationship between the two groups.

The correlation tests in our study between Minor Physical Anomalies and Soft Neurological Signs show that there is a positive relationship between them. The value of p shows statistically significant results (significant $p<0.05$) [Table 17]. There is a significant correlation between minor physical anomalies and soft neurological signs with $p=0.001$.

In this study, different lines of associations between soft neurological signs were explored. There is positive relationship between the soft neurological signs and delusion and hallucination with p value $< .05$

It has been studied and this significant association proves the etiopathogenesis and the symptom dimension of the illness. In our study the patients without delusion or hallucination has been compared with the patients with delusion and hallucination of variable severity with that of the MPA and SNS and there was a significant association of SNS with the symptoms.

(table 18) This throws light for future studies to prove the association in a bigger sample and helps in viewing the symptom dimension from a different angle and helps the MPA ans the SNS by using it as a putative marker.

Smaller sample size has influenced the statistical results in not providing adequate ground to the observations. Another reason may be the recruitment of the samples from the tertiary care center which has resulted in high concentration of the samples in one particular group with no uniform distribution among the different strata of the society. Future studies should be considering capturing a wider population with variation thereby providing a comprehensive picture to the existing scenario.

In spite of its limitations, this study proves to be significant in understanding the prevalence of minor physical anomalies and soft neurological signs in patients with schizophrenia. Different lines of comparisons and exploration of associations shows that the development of minor physical anomalies and soft neurological signs is multifaceted and multifactorial and proves the neuro developmental etiological model .

CONCLUSION

CONCLUSION

Minor physical anomalies and soft neurological signs were widely prevalent in schizophrenic population. These minor physical anomalies represents fossilized imprints of early embroyonic development Regarding the topographical distribution of these minor physical anomalies, there is an increased distribution of minor physical anomalies in head and in mouth region compared to the periphery. The increased mouth anomaly has been showed already by Stefan et al. which strengthens the neurodevelopmental etiology.

The increased occurrence of soft neurological signs in schizophrenic patients also points towards the developmental etiology. More neurological signs in drug naive patients proves the cerebral insult as its etiology and it also shows that these signs were not been related to the side effects of neuroleptic medications. The relationship between soft neurological signs and psychopathology has been influenced by the phase of illness, antipsychotic drugs effects .Minor physical anomalies develop due to altered physical development which reflects early brain insults. Along with the altered early cerebral insults , the environmental insults during the early stage acts as a triggering event for the development of schizophrenia. Correlating the minor physical anomalies with their siblings may expand our knowledge about the developmental cause for the disease.

The study in the area of positive and negative symptoms in relation to the presence of minor physical anomalies and the soft neurological signs should be explored further to elucidate better in early prediction of the course

and the severity of illness .Our study illustrates the importance of family history in schizophrenic patients to prove the genetic cause and this is better studied if the cases were compared to their siblings which has been studied earlier. Also our study highlights the importance of MPA and SNS as endophenotypes in identifying the schizophrenic illness.

The study in finding the association of the MPA and SNS in relation to delusion and hallucination will help in understanding the symptom dimension and etiopathogenesis of the disease process.

In our study though the association between PANSS and SNS were not significant, on evaluating individual symptoms like delusion/ Hallucination with that of SNS were found to be significant (<0.05).

There is a need for further study with large sample size for better understanding of the association of these signs with the symptomatology and to the severity of illness and treatment response. This will help in identifying high risk individual and possible interventions may be initiated early to prevent the disease occurrence.

LIMITATIONS

LIMITATIONS

- The sample size is small consisting of 60 cases and controls.
- The study was conducted in tertiary care center and the findings cannot be generalized.
- All the minor physical abnormalities were not studied.
- The relationship between soft neurological signs and psychopathology may get influenced by the phases of illness, antipsychotic drugs effects.
- The cognitive burden of the patients in recalling few retrospective events.
- Cross-sectional nature of the study makes the findings non-generalisable.
- Only one researcher for this study create a bias.

BIBLIOGRAPHY

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1. Neurodevelopmental indices and the development of psychotic symptoms in subjects at high risk of schizophrenia -Stephen m. Lawrie, Majellabyrne, Patrick miller, Ann hodges, Robert a. Clafferty, David g. Cunningham owens, eve c. Johnstone The British Journal of Psychiatry Jun 2001, 178 (6) 524-530; DOI: 10.1192/bjp.178.6.524

2. CloustonTS :neuroses of development: the Morrison lectures for 1890, Edinburg, Scotland, Oliver & Boyd 1891.

3. MS. Neurodevelopment and schizophrenia: Quo vadis? In: Keshavan MS, Murray RM, editors. Neuro development and Adult Psychopathology. 1st ed. London: Cambridge University Press; 1997. pp. 267–77.

4. Woods BT. Is schizophrenia a progressive neurodevelopmental disorder? Toward a Unitary pathogenetic mechanism.Am J Psychiatry. 1998;155:1661–70.

5. Murray RM, Lewis SW. Is schizophrenia a neurodevelopmental disor-der? BMJ (Clin Res Ed) 1987; 295: 681– 2.

6. Weinberger DR. The pathogenesis of schizophrenia: a neuro developmental theory. In The Neurology of Schizophrenia (eds RA Nasrallah & DR Weinberger): 387– 405. Elsevier, 1986 .7. Murray

8.. Gottesman, 1.1., and Shields, J. Schizophrenia: The Epigenetic Puzzle. Cambridge: Cambridge University Press, 1982.

9. Torrey, E.F.; Miller, J.; Rawlings, R.; Rawlings, R.; and Yolken, R.H. Seasonality of births in schizophrenia and bipolar disorder: A review of the literature. Schizophrenia Research, 28:1-38, 1999

10. J. J., van Os, J., Hoyos, C., et al (1995) Minor physical anomalies in psychoses: associations with clinical and putative aetiological variables. Schizophrenia Research, 18, 9 -20.

11. Brown AS, Cohen P, Greenwald S, Susser E. Nonaffective psychosis after prenatal exposure to rubella. Am J Psychiatry 2000; 157: 438-43.

12. Sipos A., Rasmussen F., Harrison G., Tynelius P., Lewis G., Leon D.A., Gunnell D. Paternal age and schizophrenia: a population based cohort study. British Medical Journal. 2004;329:1070–1073.

13. Perrin M, Harlap S, Kleinhaus K, Lichtenberg P, Manor O, Draiman B, Fennig S, Malaspina D. Older paternal age strongly increases the morbidity for schizophrenia in sisters of affected females. Am J Med Genet B Neuropsychiatr Genet. 2010;153B:1329–1335.

14. Carroll, K. M., Power, M.-E. D., Bryant, K., et al (1993) One year follow up status of treatment seeking cocaine abusers: psychopathology and dependence severity as predictors of outcome. Journal of Nervous and Mental Disease, 181, 71-79.

15. Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyl transferase gene: longitudinal evidence of a gene X environment interaction. Caspi A1, Moffitt TE, Cannon M, McClay J, Murray R,

Harrington H, Taylor A, Arseneault L, Williams B, Braithwaite A, Poulton R, Craig IW.

16. Cannon T. D., van Erp T. G., Rosso I. M., Huttunen M., Lonnqvist J., Pirkola T., et al. (2002b). Fetal hypoxia and structural brain abnormalities in schizophrenic patients, their siblings, and controls. Arch. Gen. Psychiatry 59, 35–41 10.1001/archpsyc.59.1.35

17. Waddington, J. The relationship of minor physical anomalies and other putative indexes of developmental disturbance in schizophrenia to abnormalities of cerebral structure on magnetic resonance imaging. Biological Psychiatry, 38:516-524, 1995.

18. Ismail, B., Cantor-Graae, E., McNeil, T.F., 1998. Minor physical anomalies in schizophrenic patients and their siblings. American Journal of Psychiatry 155, 1695–1702.

19. Ismail, B., Cantor-Graae, E. & McNeil, T. F. (1998b) Neurological abnormalities in schizophrenic patients and their siblings. American Journal of Psychiatry, 155, 84 -89.

20. Lal R, Sharma S. Minor physical anomalies in schizophrenia. Indian J Psychiatry. 1987;29:119–

21. Nizamie SH, Nizamie A, Sangma MW, Sharma PL. Soft neurological signs and minor physical anomalies in schizophrenia. Indian J Psychiatry. 1989;31:230–7.

22. O'Callaghan E, Buckley P, Madigan C, Redmond O, Stack JP, Kinsella A, Larkin C, Ennis JT, Waddington JL: The relation- ship of minor

physical anomalies and other putative indices of developmental disturbance in schizophrenia to abnormalities of cerebral structure on magnetic resonance imaging. Biol Psychiatry 1995; 38:516–524

23. Green, M.F.; Satz, P.; and Christenson, C. Minor physical anomalies in schizophrenia patients, bipolar patients, and their siblings. Schizophrenia Bulletin, 20(3):433-440, 1994

24. Slater E, Beard AW, Glithero E. The schizophrenia-like psychoses of epilepsy. Br J Psychiatry 1963;109:95-150.

25. Dalman, C., Allebeck, P., Cullberg, J., et al (1999) Obstetric complications and the risk of schizophrenia. Archives of General Psychiatry, 56, 234 -240.

26. Lal R, Sharma S: Minor physical anomalies in schizophrenia. Indian J Psychiatry 1987; 29:119–122

27. Alexander RC, Mukherjee S, Richter J, Kaufmann CA: Minor physical anomalies in schizophrenia. J NervMent Dis 1994; 182:639–644

28. Goldfarb, W., and Botstein, A. "Physical Stigmata in Schizophrenic Children." Unpublished man- uscript, Henry Ittleson Center for Child Research, Brooklyn, NY, 1967.

29 Waldrop MF, Bell RQ, McLaughlin B, Halverson CF Jr. Newborn minor physical anomalies predict short attention span, peer aggression, and impulsivity at age 3. Science 1978;199:563-565.

30. Lane A, Kinsella A, Murphy P, Byrne M, Keenan J, Colgan K, Cassidy B, Sheppard N, Horgan R, Waddington JL, Larkin C, O'Callaghan E:

The anthropometric assessment of dysmor- phic features in schizophrenia as an index of its developmen- tal origins. Psychol Med 1997; 27:1155–1164

31. Minor Physical Anomalies in Schizophrenic Patients and Their Siblings Baher Ismail, M.D., Elizabeth Cantor-Graae, Ph.D., and Thomas F. McNeil, Ph.D.

32. Nizamie SH NA, Sangama MW, Sharma PL. Soft Neurological Signs, and minor physical anomalies in schizophrenia. Indian Journal of Psychiatry 1989, 31: 230-7.

33. Waldrop MF, Halverson CF: Minor physical anomalies and hyperactive behavior in young children, in Exceptional Infant. Edited by Hellmuth J. New York, Brunner/Mazel, 1971, pp 343–381

34. Akabaliev VH, Sivkov ST. Sexual dimorphism in minor physical anomalies in schizophrenic patients and normal controls. Compr Psychiatry 2003;44:341-8.

35. Lohr, J.B., and Flynn, K. Minor physical anomalies in schizophrenia and mood disorders. Schizophrenia Bulletin, 19(3):551-556, 1993.

36. Laufer MW, Denhoff E, Solomons G. Hyperkinetic impulse disorder in children's behavior problems. Psychosom Med. 1957;19:38–49.

37. Aksoy-Poyraz C, Poyraz BC, Turan S, Arikan MK (in press, Corrected Proof) Minor physical anomalies and neurological soft signs in patients with schizophrenia and their siblings. Psychiatry Research.

38. J NeurolNeurosurg Psychiatry. 1998 Jan;64(1):56-60. Minor physical anomalies in familial and sporadic schizophrenia: the Maudsley family study. Griffiths TD1, Sigmundsson T, Takei N, Frangou S, Birkett PB, Sharma T, Reveley AM, Murray RM.

39. Schiffman J, Ekstrom M, LaBrie J, Schulsinger F, Sorensen H et al. (2002) Minor physical anomalies and schizophrenia spectrum disorders: a prospective investigation. Am J Psychiatry; February; 159(2): 238–43.

40. Prevalence and score of minor physical anomalies in patients with schizophrenia and their first degree relatives: A Tunisian study-Leila Gassab , MounaAissi, HélaSlama, LotfiGaha, Anwar Mechri,

41. Hata K, Iida J, Iwasaka H, Negoro HI, Ueda F, Kishimoto T. Minor physical anomalies in childhood and adolescent onset schizophrenia. Psychiatry ClinNeurosci. 2003;57:17–21

42. Schizophr Bull. 1989;15(1):91-9. Minor physical anomalies in schizophrenia. Green MF, Satz P, Gaier DJ, Ganzell S, Kharabi F.

43. Neurological soft signs and minor physical anomalies in patients with schizophrenia and related disorders, their first-degree biological relatives, and non-psychiatric controls. Compton MT, Bollini AM, McKenzie Mack L, Kryda AD, Rutland J, Weiss PS, Bercu Z, Esterberg ML, Walker EFSchizophr Res. 2007 Aug; 94(1-3):64-73.

44. Minor physical anomalies in psychoses: associations with clinical and putative aetiological variables.McGrath JJ, van Os J, Hoyos C, Jones PB, Harvey I, Murray RMSchizophr Res. 1995 Dec; 18(1):9-20.

45. Dazzan P, Morgan K, Chapple B, et al. The effects of atypical and typical antipsychotics on brain structure in the AESOP first-onset psychosis study. Schizophrenia Res. 2004;67:95.

46. EricY.H.Chen, Jane Shapleske, Rogelio Luque, Peter J. McKenna et al The cambridge neurological inventory : A clinical instrument for assessment of soft neurological signs in psychiatric patients.

47. Significance and meaning of neurological signs in schizophrenia. Heinrichs DW, Buchanan RW, Am J Psychiatry. 1988 Jan; 145(1):11-8.

48. Am J Psychiatry. 2000 Apr;157(4):560-5. Neurological signs and the heterogeneity of schizophrenia.Arango C1, Kirkpatrick B, Buchanan RW.

49. Neurological soft signs in schizophrenia – The past, the present and the future ShivaramaVarambally, GanesanVenkatasubramanian, and Bangalore N. Gangadhar

50. Biol Psychiatry. 1996 Nov 15;40(10):1056-9.Neurological soft signs and response to risperidone in chronic schizophrenia.Smith RC1, Kadewari RP.

51. Chen Y. (2011). Abnormal visual motion processing in schizophrenia: a review of research progress. Schizophr. Bull. 37, 709–715 10.1093/schbul/sbr020

52. Torrey, E. F. (1980). Neurological abnormalities in schizophrenic patients. Biological Psychiatry, 15, 381-388.

53. Weller, M. and Kugler, B. T. (1979).Tactile discrimination in schizophrenic and affective psychoses. In: Gruzelier, J., Flor-Henry, P. (Eds.), Hemis- phere asymmetries in function in psychopathology. Amsterdam: Elsevier, North Holland.

54. Woods, B. T.; Kinney, D. K. and Yurgelun-Todd, D. (1986). Neurological abnormalities in schizoplire- nia patients and their families-I: Comparison of schizophrenic, bipolar, and substance abuse patients and normal controls. Archives of General Psychiatry, 43, 657-663.

55. Relationship between minor physical anomalies and "soft signs" of brain damage (1979), by K A Paulsen, J P O'Donnell

56. Tiryaki A, Yazici MK, Anil AE, Kabakc E, Karaaaolu E, Gou A. Reexamination of the characteristics of the deficit schizophrenia patients. Eur. Arch. Psychiatr. Clin.Neurosci. 2003;253:221–227.

57. Am J Psychiatry. 1996 Apr;153(4):526-32. Soft signs and neuropsychological performance in schizophrenia. Flashman LA1, Flaum M, Gupta S, Andreasen NC.

58. Scheffer RE. Abnormal neurological signs at the onset of psychosis. Schizophr Res. 2004;70:19–26

59. Bachmann S, Bottmer C, Schröder S.. Neurological soft signs in first-episode schizophrenia: a follow-up study. Am J Psychiatry (2005) 162:2337–43.10.1176/appi.ajp.162.12.2337

60. Merriam, A.E.; Kay, S.R.; Opler, L.A.; Kushner, S.F.; and van Praag, V.M. Neurological soft signs and positive-neg- ative dimension in schizophrenia. Biological Psychiatry, 28:181-192, 1990.

61. Neurological soft signs in schizophrenia: Are they related to negative or positive symptoms, neuropsychological performance, and violence? Claude M.J. Braun, Dominique Lapierrea. Arch Clin Neuropsychology.

62. Chen EYH, Chan CK. The Cambridge Neurological Inventory: clinical, demographic, and ethnic correlates. Psychiatr Ann. 2003;33:202–210.

63. Niethammer, R, Weisbrod, M, Schiesser, S, et al. Genetic influence on laterality in schizophrenia? a twin study of neurological soft signs. Am J Psychiat2000 ;157272–274.

64. Egan MF, Goldberg TE, Gscheidle T, Weirich M, Bigelow LB, Weinberger DR. Relative risk of attention deficits in siblings of patients with schizophrenia. Am J Psychiatry. 2000;157:1309–1316.

65. Addington J, Cadenhead K, Cannon T (2007). North American Prodrome Longitudinal Study: a collaborative multisite approach to prodromal schizophrenia research. Schizophr Bull 33:665-672.

66. Tosato S, Dazzan P (2005). The psychopathology of schizophrenia and the presence of neurological soft signs: a review. CurrOpin Psychiatry 18:285-288.

67.John JP, Arunachalam V, Ratnam B, Isaac MK. Expanding the schizophrenia phenotype: a composite evaluation of neurodevelopmental markers. Compr.Psychiatry. 2008;49(1):78–86.

68. O'Callaghan, E., Gibson, T., Colohan, H. A., et al (1992) Risk of schizophrenia in adults born after obstetric complications and their association with early onset of illness: a controlled study. British Medical Journal, 305, 1256 -1259.

69. Gupta, S, Andreasen, NC, Arndt, S, et al. Neurological soft signs in neuroleptic-naive and neuroleptic-treated schizophrenic patients and in normal comparison subjects. Am J Psychiat1995 ;152191–196.

70. Keshavan, MS, Sanders, RD, Sweeney, JA, et al. Diagnostic specificity and neuroanatomical validity of neurological abnormalities in first-episode psychoses. Am J Psychiat2003 ;1601298–1304.

71. Scheffer, RE. Abnormal neurological signs at the onset of psychosis. Schizophr Res 2004 ;7019–26.

72 . Whitty P, Clarke M, Browne S, McTigue O, Kamali M, Feeney L, et al. Prospective evaluation of neurological soft signs in first-episode schizophrenia in relation to psychopathology: State versus trait phenomena. Psychol Med. 2003;33:1479–84

73. Schreiber,H., Rothmeier,J., Becker,W., Jurgens,R., Born,J., Stolz-Born,G., Westphal,K.P., Kornhuber,H.H., 1995. Comparative assessment of saccadic eye movements, psychomotor and cognitive performance in schizophrenics, their firstdegree relatives and control subjects. Acta Psychiatr Scand. 91, 195.- 201.

74. Smith RC, Calderon M, Ravichandran GK et al. (1984), Nuclear magnetic resonance in schizophrenia: a preliminary study. Psychiatry Res 12(2):137-147.

75. Emsley R, Turner HJ, Oosthuizen PP, et al. Neurological abnormalities in first-episode schizophrenia: Temporal stability and clinical outcome correlates. Schizophrenia Research 2005; 75(1): 35-44

76. Compr Psychiatry. 2003 Jul-Aug;44(4):341-8. Sexual dimorphism in minor physical anomalies in schizophrenic patients and normal controls. Akabaliev VH1, Sivkov ST.

77. Guy, J.D.; Majorski, L.V.; Wallace, C.J.; and Guy, M.P. The incidence of minor physical anomalies in adult male schizophrenics. Schizophrenia Bulletin, 9:571-582, 1983.

78. Marcus, J., Hans, S. L., Lewow, E., et al (1985) Neurological findings in high-risk children: childhood assessment and 5-year follow-up. Schizophrenia Bulletin, 11, 85 -100.

79. Minor Physical Anomalies and Vulnerability in Prodromal Youth -Vijay A. Mittala,b and Elaine F. Walkerc

80. James .B. L Ohr and Klrsten Flynn minor physical anomalies in schizophrenia 1993.

தகவல் படிவம்
சிறிய உடல் முரண்பாடுகள் மற்றும் மென்மையான நரம்பியல் அறிகுறிகள் மனச்சிதைவு நோயாளிகளிடையே ஓர் ஒப்புமை ஆய்வு

ஆராய்ச்சியின் நோக்கமும், பயன்களும்:
உங்கள் பங் கேற்பு திட்டமிடப்பட்டுள்ள இந்த மருத்துவ ஆராய்ச்சி ஆய்வின் நோக்கம்:

சிறிய உடல் முரண்பாடுகள் என்பது வெ ளிதோற்றத்தில் ஏற்படும் விலகல்கள். நரம்பு மண்டலமும், தோல் பகுதியும் தோற்றம் ஓரிடம் என்பதால் சிறிய உடல் முரண்பாடுகள் என்பது முக்கியத்துவம் வாய்ந்ததாகிறது. நோயின் தீவிரத்தன் மை அளவிடவும் முடிகிறது. மனச்சிதைவு நோய் கரு உருவாவதில் உள்ள இடையூரின் பேரில் இரு ந்தே ஆரம்பமாவதால் இதனை அளவிடுதல் அத்தியாவசியமாகிறது.

மென்மையான நரம்பியல் அறிகுறிகள் என்பது குறியிட முடியாத தன்மையுடையது.. மனச்சிதைவு நோயாளிகள்
மத்தியில் நிலவும் இந்த அறிகுறிக ளை அளவிடுவதனால் நோயின் வீரியத்தன்மை கண்டடையவும் உதவுகிறது. இந்த அறிகுறிகள் நிகழ்வு மரபணு கட்டுப்பாட்டின் கீழ் உள்ளது. இவை மனச்சிதைவு நோயின் இந்த பண்பாகும் அம்சம் பிரதிநிதித்துவம் என்று சுட்டிக் காட்டப்பட்டுள்ளது.

ஆய்வு நடைமுறைகள்:
அடிப்படையாக , குறைந்தது ஐந்தாண்டுக்கு மேலாக மனச்சிதைவு நோயென்று கண்டறியப்பட்டவர்கள்,ஆண்,
பெண் இருபாலரும் இந்த ஆய்வில் சேர்த்துக் கொள்ளப்படுவார்கள்.

அந்தரங்கத் தன் மை:
உங்கள் / உங்கள் மனைவி/கணவரின் மருத்துவப் பதிவேடுகள் மிகவும் அந்தரங்கமாக வைத்துக் கொள்ளப்படும்
மற்றும் இன்ன பிற மருத்துவர்கள்/விஞ்ஞானிகள்/இந்த ஆய்வின் தணிக்கையாளர்கள் அல்லது ஆராய்ச்சி
ஆதரவாளர்களின் பிரதிநிதிகள் ஆகியோரிடமும் அவை வெளிப்படுத்தப்படும். இந்த ஆய்வின் முடிவுகள்
அறிவியல் பத்திரிக்கைகளில் பிரசுரிக்கப்படலாம். ஆனால் பெயரை வெளியிடுவதன்மூலம் நோயாளிகளின் அடையாளம்

காட்டப்பட மாட்டார்கள்.
ஆய்வில் உங்கள் பங்கேற்பு மற்றும் உங்கள் உரிமைகள்
இந்த ஆய்வில் உங்கள்/உங்கள் உறவினரின் பங்கேற்பு முழுவதும் உங்களுடைய விருப்பத்தைச் சார்ந்தது.
இதில் நீங்கள் பங்கேற்க மறுக்கவோ, பாதியில் வெளி யேறிவிடவோ அல்லது குறிப்பிட்ட கேள்விகளுக்கு
விடையளிக்க மறுக்கவோ, உங்களுக்கு முழு உரிமை உண்டு. எப்படி இருந்தாலும் உங்கள்/உங்கள் உறவினரின்
உடல்நிலைக்கேற்ப, உங்களுக்கு/உங்கள் உறவினருக்கு பொருத்தமான சிகிச்சை தொடர்ந்து அளிக்கப்படும்.
தாங்கள் இது குறித்து வேறு விபரங்கள் தெரிந்து கொள்ள விரும்பினால், எங்களிடம் கேட்டுத்
தெரிந்துகொள்ளலாம்.
மேலும் விபரங்கள் அறிய கீழ் கண்ட நபரை அணுகவும்:

மரு. ஜெனிபர் சங்கீதா 098402 22308

(தனியாகப் பிரித்தெடுத்து, ஆய்வில் பங்கேற்பவரிடம் தரப்பட வேண்டும்)

சுய ஒப்புதல் படிவம் -நோயாளி

ஆய்வின் பெயர் : : சிறிய உடல் முரண்பாடுகள் மற்றும் மென்மையான நரம்பியல் அறிகுறிகள் மனச்சிதைவு நோயாளிகளிடையே ஓர் ஒப்புமை ஆய்வு .

ஆராய்ச்சி நிலையம் பிரிவு, : மனநலப்புறநோயாளிகள்

அரசு ஸ்டான்லி மருத்துவமனை, சென்னை -6 00 001.

பங்கு பெறுபவரின் பெயர் :
பங்கு பெறுபவரின் எண் :

நோயாளி இதனை (√) குறிக்கவும்.

மேலே குறிப்பிடப்பட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கங்களைப் பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும் எந்த கட்டத்திலும் எந்த சட்டச்சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்று அறிந்துகொள்கிறேன். ☐

இந்த ஆய்வு சம்பந்தமாகவும், இதைச் சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும், இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளைப் பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும், மற்றும் சிகிச்சை தொடர்பான தகவல்களையும், மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் / பதிப்பிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்படும் அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதி அளிக்கிறேன். என் உடல்நலம் பாதிக்கப்பட்டாலோ அல்லது

எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க் குறி தென்பட்டாலோ, உடனே அதனை மருத்துவ அணிக்கு தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.

நோயாளி/பங்கேற்பவரின் கையொப்பம் _____
இடம் _____தேதி ____

கட்டை விரல் ரேகை _

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்

_____

_____

_____

ஆய்வாளரின் கையொப்பாம்

_____ இடம் _____தேதி ___

ஆய்வாளரின் பெயர் _____

நோயாளியின் பெயர் _____ பாலினம் : ஆண் _____.
பெண் _____.

வயது _____ஆண்டுகள் அல்லது பிறந்த தேதி
_____

நோயாளியை தொடர்பு கொள்ளும் முகவரி

_____

_____

_____

நோயாளியின் தொலைபேசி எண்.

நோயாளியின் உறவினர் பெயர் _____

		பங்கேற்பவரின் கையொப்பம்/

		பெருவிரல் பதிப்பு
1	மேலே குறிப்பிடப்பட்டுள்ள மருத்துவ ஆய்வின் ..... தேதியிட்ட நோயாளிகளுக்கான செய்தி நான் படித்திருக்கிறேன் மற்றும் புரிந்திருக்கிறேன்/ விவரிக்கப்பட்டுள்ளேன். கேள்விகள் கேட்கவும் அனுமதி வழங்கப்பட்டுள்ளேன் என நான் உறுதி செய்கிறேன்.	
2	இந்த ஆய்வில் பங்கேற்பது என் சொந்த விருப்பப்படியே என நான் அறிந்திருக்கிறேன்.. மேலும் என் மருத்துவ சிகிச்சை கவனிப்பு அல்லது சட்டபூர்வ உரிமைகளுக்கு பாதிப்பு ஏற்படாமல் நான் எந்த நேரத்திலும் விலகிக் கொள்ளலாம் என்பதை அறிந்திருக்கிறேன்.	
3	எத்திக்ஸ் கம்மிட்டி மற்றும் ரெகுலேட்டரி அத்தாரிட்டீஸ்-க்கும் நான் இந்த ஆய்விலிருந்து விலகினாலும் தற்போதைய மற்றும் எதிர்கால இந்த ஆய்வு சார்ந்த என் உடல்நல குறிப்புகளை என் அனுமதியின்றி பார்க்க முடியும் என நான் அறிகிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	
4	இந்த ஆய்வின் மூலம் கிடைக்கப்பெறும் குறிப்புகளையும் தகவல்களையும் மற்றும் பரிசோதனை முடிவுகளையும், உபயோகப்படுத்த தடை செய்ய மாட்டேன் என சம்மதிக்கிறேன். அதனால் அவைகள் விஞ்ஞானம், ஆராய்ச்சிக் கட்டுரைகள் போன்ற சம்மந்தப்பட்டவைகளுக்கு பயன் உள்ளதாக இருக்க வேண்டும். இக்குறிப்புகள், அதன் விளக்கங்கள், ஆய்வுக் கட்டுரைகள் ஆகியவற்றை பிரசுரிக்கவும் / பதிப்பிக்கவும் நான் முழு மனதுடன் சம்மதிக்கிறேன்.	
5	மேற்கூறிய ஆய்வில் என் சுய விருப்பத்தின்படி பங்கு கொள்ள நான் சம்மதிக்கிறேன்.	

ஆய்வில் பங்கேற்பவர் கையொப்பம் அல்லது பெரு விரல் பதிவு

சுய ஒப்புதல் படிவம் - நோயாளியின் உறவினர்

ஆய்வின் பெயர் : : சிறிய உடல் முரண்பாடுகள் மற்றும் மென்மையான நரம்பியல் அறிகுறிகள் மனச்சிதைவு நோயாளிகளிடையே ஓர் ஒப்புமை ஆய்வு .

ஆராய்ச்சி நிலையம் பிரிவு, : மனநலப்புறநோயாளிகள்

அரசு ஸ்டாண்ளி மருத்துவமனை, சென்னை -600 001.

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் எண் :

நோயாளியின் உறவினர் இதனை (√) குறிக்கவும்.

மேலே குறிப்பிடப்பட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கங்களைப் பெரவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் / என் உறவினர் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும் எந்த கட்டத்திலும் எந்த சட்டச்சிக்கலுக்கும் உட்படாமல் நான் / என் உறவினர் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்று அறிந்துகொள்கிறேன். ☐

இந்த ஆய்வு சம்பந்தமாகவும், இதைச் சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும், இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய உறவினர் மருத்துவ அறிக்கைகளைப் பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் / என் உறவினர் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், என் உறவினர் நேர்முக

பரிசோதனை முடிவுகளையும், மற்றும் சிகிச்சை தொடர்பான தகவல்களையும், மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் / பதிப்பிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்படும் அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதி அளிக்கிறேன். எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க் குறி தென்பட்டாலோ, உடனே அதனை மருத்துவ அணிக்கு தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.

பங்கேற்கும் உறவினரின் கையொப்பம் _____
இடம் _____தேதி ____

கட்டை விரல் ரேகை _

/பங்கேற்பவரின் காப்பாளரின் கையொப்பம்
_____ இடம் _____தேதி ____

கட்டை விரல் ரேகை _

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்
_____

_____

_____

ஆய்வாளரின் கையொப்பாம்
_____ இடம் _____தேதி ___

ஆய்வாளரின் பெயர் _____

நோயாளியின் உறவினர் பெயர் _____ பாலினம் :
ஆண் _____; பெண் _____

வயது _____ஆண்டுகள் அல்லது பிறந்த தேதி _____

நோயாளியின் உறவினர் தொடர்பு கொள்ளும் முகவரி

_____

_____

_____

நோயாளியின் உறவினர் தொலைபேசி எண்.

நோயாளியின் பெயர் _____

		பங்கேற்பவரின் கையொப்பம்/ பெருவிரல் பதிப்பு
1	மேலே குறிப்பிடப்பட்டுள்ள மருத்துவ ஆய்வின் _____ தேதியிட்ட நோயாளிகளுக்கான செய்தி நான் படித்திருக்கிறேன் மற்றும் புரிந்திருக்கிறேன்/ விவரிக்கப்பட்டுள்ளேன். கேள்விகள் கேட்கவும் அனுமதி வழங்கப்பட்டுள்ளேன் என நான் உறுதி செய்கிறேன்.	
2	இந்த ஆய்வில் பங்கேற்பது என் / என் உறவினரின் சொந்த விருப்பப்படியே என நான் அறிந்திருக்கிறேன்.. மேலும் என் / என் உறவினரின் மருத்துவ சிகிச்சை கவனிப்பு அல்லது சட்டபூர்வ உரிமைகளுக்கு பாதிப்பு ஏற்படாமல் நான் எந்த நேரத்திலும் விலகிக் கொள்ளலாம் என்பதை அறிந்திருக்கிறேன்.	
3	எத்திக்ஸ் கம்மிட்டி மற்றும் ரெகுலேட்டரி அத்தாரிட்டிஸ்-க்கும் நான் இந்த ஆய்விலிருந்து விலகினாலும் தற்போதைய மற்றும் எதிர்கால இந்த ஆய்வு சார்ந்த என் / என் உறவினர் உடல்நல குறிப்புகளை என் அனுமதியின்றி பார்க்க முடியும் என நான் அறிகிறேன். நான் / என் உறவினர் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	
4	இந்த ஆய்வின் மூலம் கிடைக்கப்பெறும் குறிப்புகளையும் தகவல்களையும் மற்றும் முடிவுகளையும், உபயோகப்படுத்த தடை செய்ய மாட்டேன் என சம்மதிக்கிறேன். அதனால் அவைகள் விஞ்ஞானம், ஆராய்ச்சிக் கட்டுரைகள் போன்ற சம்மந்தப்பட்டவைகளுக்கு பயன் உள்ளதாக இருக்க வேண்டும். இக்குறிப்புகள், அதன் விளக்கங்கள், ஆய்வுக் கட்டுரைகள் ஆகியவற்றை பிரசுரிக்கவும் / பதிப்பிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன்.	

5	மேற்கூறிய ஆய்வில் என் சுய விருப்பத்தின்படி பங்கு கொள்ள நான் சம்மதிக்கிறேன்.	

ஆய்வில் பங்கேற்பவர் /
சட்டபூர்வமாக ஏற்கப்பட்ட நபர்
கையொப்பம் அல்லது பெரு விரல்
பதிவு

ANNEXURES

PROFORMA

NAME:

AGE:

SEX: MALE –1, FEMALE-2

RESIDENCE:URBAN/SEMIURBAN/RURAL

EDUCATION: ILLITERATE/ < VSTD / VI TO VIII STD/ IX TO X STD/XI TO XII STD DIPLOMA/GRADUATE/PG /PROFESSIONAL

OCCUPATION:

UNEMPLOYED/UNSKILLED/SEMISKILLED/SKILLED/CLERICAL SHOP OWNER FARMER/SEMIPROFESSIONAL /PROFESSIONAL

MARITAL STATUS:

UNMARRIED/MARRIED/WIDOW/DIVORCED/SEPERA TED

SOCIO-ECONOMIC STATUS:

UPPER /UPPERMIDDLE/LOWERMIDDLE /UPPERLOWER /LOWER

FAMILY HISTORY OF SCHIZOPHRENIA:

FIRST DEGREE RELATIVE/SECOND DEGREE RELATIVE/NO FAMILY HISTORY

AGE OF ONSET:

DURATION OF ILLNESS:

DELUSIONS: PRESENT / ABSENT

HALLUCINATIONS:PRESENT / ABSENT

PANSS:

1. POSITIVE SCALE
2. NEGATIVE SCALE
3. GENERAL PSYCHOPATHOLOGY SCALE

WALDROP SCALE: FOR MINOR PHYSICAL ANOMALIES

CAMBRIDGE NEUROLOGICAL INVENTORY:
FOR SOFT NEUROLOGICAL SIGNS

M.I.N.I.

MINI INTERNATIONAL NEUROPSYCHIATRICINTERVIEW

English Version5.0.0 DSM-IV

USA: **D. Sheehan, J. Janavs, R. Baker, K. Harnett-Sheehan, E. Knapp, M.Sheehan**

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DISCLAIMER

Our aim is to assist in the assessment and tracking of patients with greater efficiency and accuracy. Before action is takenon any data collected and processed by this program, it should be reviewed and interpreted by a licensedclinician.

This program is not designed or intended tobe use d in the place of a full medical and psychiatric valuation by a qualified licensed physician psychiatrist. It is intended only as a tool to facilitate accurate data collection and processing of symptoms elicited by trainedpersonnel.

M.I.N.I. 5.0.0 (July 1,2006)

L. PSYCHOTIC DISORDERS AND MOOD DISORDER WITHPSYCHOTIC FEATURES

Ask for an example of each question answered positively. Code **yes** only if the examples clearly show a distortion of thought or of perception or if they are not culturally appropriate before coding, investigate whether delusions qualify as"bizarre".

Delusions are" bizarre" if: clearly implausible, absurd, not understandable, and cannot derive from ordinary life experience.

Hallucinations are scored "bizarre" if: avoice comments on the person's thoughts or behavior, or when two or more voices are conversing with each other.

		Now I am going to ask you about unusual experiences that some peoplehave.		
L1	a	was plotting against you, or trying to hurtyou? **NOTE**: ASK FOR EXAMPLES TO RULE OUT ACTUALSTALKING.	NO	YES
	b	**IF YES OR YES BIZARRE:** do you currently believe thesethings?	NO	YES
L2	a	Have you ever believed that someone was reading your mind or couldhear your thoughts, or that you could actually read someone's mind or hearwhat another person was thinking?	NO	YES
	b	**IF YES OR YES BIZARRE:** do you currently believe thesethings?	NO	YES
L3	a	Have you ever believed that someone or some force outside ofyourself put thoughts in your mind that were not your own, or made you act ina way that was not your usual self? Have you ever felt that youwere possessed? CLINICIAN: ASK FOR EXAMPLES AND DISCOUNT ANY THAT ARE NOTPSYCHOTIC.	NO	YES
	b	**IF YES OR YES BIZARRE:** do you currently believe thesethings?	NO	YES
L4	a	Have you ever believed that you were being sent special messagesthrough the TV, radio, or newspaper, or that a person you did not personallyknow was particularly interested inyou?	NO	YES
	b	**IF YES OR YES BIZARRE:** do you currently believe thesethings?	NO	YES
L5	a	Have your relatives or friends ever considered any of your beliefsstrange or unusual? INTERVIEWER:ASKFOREXAMPLES.ONLYCODE**YES**IFTHEEXAMPLESARE**CLEARLY** DELUSIONALIDEASNOTEXPLOREDINQUESTIONSL1TOL4,FOREXAMPLE,SOMATICORRELIGIOUS DELUSIONSORDELUSIONSOFGRANDIOSITY,JEALOUSY,GUILT,RUINORDESTITIUTION,ETC.	NO	YES
	b	**IF YES OR YES BIZARRE:** do they currently consider your beliefsstrange?	NO	YES
L6	a	Have you ever heard things other people couldn't hear, such asvoices? HALLUCINATIONSARESCORED"BIZARRE"ONLYIFPATIENTANSWERS**YES**TOTHEFOLLOWING.	NO	YES
		IF YES: Did you hear a voice commenting on your thoughts or behavioror did you hear two or more voices talking to eachother?	NO	
	b	**IF YES OR YES BIZARRE TO L6a:** have you heard these things in the HALLUCINATIONSARESCORED"BIZARRE"ONLYIFPATIENTANSWERS**YES**TOTHEFOLLOWING Did you hear a voice commenting on your thoughts or behavioror did you hear two or more voices talking to eachother?	NO	YES

L7 a Have you ever had visions when you were awake or have you ever seenthings other people couldn'tsee? NO YES

b **IF YES:** have you seen these things in the pastmonth? NO YES

CLINICIAN'SJUDGMENT

L8 b IS THE PATIENT CURRENTLY EXHIBITING SPEECH,ORMARKEDLOOSENINGOFASSOCIATIONS? NO YES

L9 b IS THE PATIENT CURRENTLY EXHIBITING DISORGANIZED BEHAVIOR? NO YES

L10 b ARENEGATIVESYMPTOMSOFSCHIZOPHRENIA,E.G.SIGNIFICANTAFFECTI FLATTENING,POVERTYOFSPEECH(ALOGIA)ORANINABILITYTOINITIATE ORPERSISTINGOALDIRECTEDACTIVITIES(AVOLITION), PROMINENTDURING THE INTERVIEW? NO YES

L11 a ARE1ORMORE«a»QUESTIONSFROML1aTOL7aCODED**YESORYESBIZAR** AND ISEITHER:
MAJOR DEPRESSIVE EPISODE, (CURRENT ORRECURRENT)
OR
MANICORHYPOMANICEPISODE,(CURRENTORPAST)CODED**YES**? NO YES
IFNOTOL11a,CIRCLENOINBOTH'MOODDISORDERWITHPSYCHOTIC FEATURES' DIAGNOSTIC BOXES AND MOVE TOL13. **{L13**

b You told me earlier that you had period(s) when you felt (depressed/high/persistently irritable).

Were the beliefs and experiences you just described (SYMPTOMSCODED**YES**FROM**L1**aTO**L7**a) restricted exclusively to times when you were feelingdepressed/high/irritable?

IF THE PATIENT EVER HAD A PERIOD OF ATLEAST 2 WEEKS OF HAVING THESE BELIEFS OR EXPERIENCES (PSYCHOTIC SYMPTOMS) WHEN THEY WERE NOT DEPRESSED / HIGH/ IRRITABLE, CODE NOT THIS DISORDER.

IF THE ANSWER IS NOT THIS DISORDE, ALSO CIRCLE NOTOL12 AND MOVE TOL13

NO YES

MOOD DISORDERWITH PSYCHOTICFEATURES

LIFETIME

L12 aARE 1 OR MORE « b » QUESTIONS FROM L1b TO L7b CODED **YES OR YESBIZARRE**
AND ISEITHER:

MAJOR DEPRESSIVE EPISODE,(CURRENT)
OR
MANIC OR HYPOMANIC EPISODE, (CURRENT) CODED**YES**?

NO YES

MOOD DISORDERWITH PSYCHOTICFEATURES

CURRENT

IF THE ANSWER IS YES TO THIS DISORDER (LIFETIME ORCURRENT), CIRCLE NO TO L13 AND L14 AND MOVE TO THE NEXTMODULE.

L13
ARE1ORMORE«b»QUESTIONSFROML1b TOL6b,CODEDYESBIZARRE**?**
OR

ARE 2 OR MOR QUESTIONS FROM L1bTOL10b, CODED**YES** (RATHERTHAN **YESBIZARRE**)?

AND DIDATLEASTT WOOF THEPSYCHOTIC SYMPTOMS OCCURDURINGTHE SAME 1 MONTH PERIOD?

NO **YES**

PSYCHOTICDISORDER
CURRENT

L14 IS **L13** CODED**YES**
OR

ARE1ORMORE«a»QUESTIONSFROML1aTOL6a,CODED**YESBIZARRE**?

OR

ARE 2 OR MORE « a » QUESTIONS FROM L1a TO L7a, CODED **YES** (RATHERTHAN **YESBIZARRE**)

AND DID ATLEAST TWO OF THEPSYCHOTIC SYMPTOMS OCCURDURING THE SAME 1 MONTH PERIOD?

NO **YES**

PSYCHOTICDISORDER
LIFETIME

Psychiatric University Hospital Zurich, P.O. Box 1931, CH-8032 Zurich, Switzerland *27.12.2007*

Psychiatric University Hospital Zurich, Division of Clinical Psychiatry

POSITIVE AND NEGATIVE SYNDROME SCALE

P A N S S

S.R. Kay, A. Fiszbein, L.A. Opler

STUDY STUDY	[_ _ _ _] [	1-4
GROUP	[_ _]	5-6
PATIENT	[_ _ _]	7-9
RATING DAY	[_ _ _]	10-12
CARD NUMBER	[_ _]	13-14
Sex (1=male, 2=female)	[_]	15
Birthday (dd.mm.yy)	[_ _ : _ _ : _ _]	16-21
Date of hospitalization (dd.mm.yy)	[_ _ : _ _ : _ _]	22-27
First diagnosis	[_ _ _ . _ _]	28-32
Second diagnosis	[_ _ _ . _ _]	33-37
Diagnostic system (1=ICD9, 2=ICD10, 3=DSM3-R, 4=DSM4)	[_]	38
Age at onset	[_ _]	39-40
Course (1=first manifestation, 2=intermittent, 3=progredient, 4=chronic)	[_]	41
Duration of Current Episode Prior to Hospitalization (days)	[_ _ _]	42-44
Medication Prior to Hospitalization (0=none, 1=antidepr., 2=neuroleptics, 3=other)	[_]	45
Current Medication (cf. list of codes)	[_ _ _]	46-48
Educational level (1=remedial, 2=junior high, 3=high, 4=college)	[_]	49
DATE (dd.mm.yy)	[_ _ : _ _ : _ _]	50-55
INTERVIEWER	[_ _ _]	56-58
HOSPITAL	[_ _]	59-60
PATIENT ID (the hospital's internal PID)	[_ _ _ _ _ _ _ _ _ _ _ _]	61-72

0=Absent 1=Minimal 2=Mild 3=Moderate 4=Moderate severe 5=Severe 6=Extreme

1-12 dupl

CARD NUMBER [_ _] 13-14

POSITIVE SCALE (P)

P1 Delusions [_] 15
Beliefs which are unfounded, unrealistic, and idiosyncratic. Basis for rating: Thought content expressed in the interview and its influence on social relations and behavior.

P2 Conceptual disorganization [_] 16
Disorganized process of thinking characterized by disruption of goal-directed sequencing, e.g., circumstantiality, tangentiality, loose associations, non sequiturs, gross illogicality, or thought block. Basis for rating: Cognitive-verbal processes observed during the course of interview.

P3 Hallucinatory behavior [_] 17
Verbal report or behavior indicating perceptions which are not generated by external stimuli. These may occur in the auditory, visual, olfactory, or somatic realms. Basis for rating: Verbal report and physical manifestations during the course of interview as well as reports of behavior by primary care workers or family.

P4 Excitement [_] 18
Hyperactivity as reflected in accelerated motor behavior, heightened responsivity to stimuli, hypervigilance, or excessive mood lability. Basis for rating: Behavioral manifestations during the course of interview as well as reports of behavior by primary care workers or family.

P5 Grandiosity [_] 19
Exaggerated self-opinion and unrealistic convictions of superiority, including delusions of extraordinary abilities, wealth, knowledge, fame, power, and moral righteousness. Basis for rating: Thought content expressed in the interview and its influence on behavior.

P6 Suspiciousness/persecution [_] 20
Unrealistic and exaggerated ideas of persecution, as reflected in guardedness, a distrustful attitude, suspicious hypervigilance, or frank delusions that others mean one harm. Basis for rating: Thought content expressed in the interview and its influence on behavior.

P7 Hostility [_] 21
Verbal and nonverbal expressions of anger and resentment, including sarcasm, passive-aggressive behavior, verbal abuse, and assaultiveness. Basis for rating: Interpersonal behavior observed during the interview and reports by primary care workers or family.

NEGATIVE SCALE (N)

N1 Blunted affect [_] 22
Diminished emotional responsiveness as characterized by a reduction in facial expression, modulation of feelings, and communicative gestures. Basis for rating: Observation of physical manifestations of affective tone and emotional responsiveness during the course of interview.

N2 Emotional withdrawal [_] 23
Lack of interest in, involvement with, and affective commitment to life's events. Basis for rating: Reports of functioning from primary care workers or family and observation of interpersonal behavior during the course of interview.

N3 Poor rapport [_] 24
Lack of interpersonal empathy, openness in conversation, and sense of closeness, interest, or involvement with the interviewer. This is evidenced by interpersonal distancing and reduced verbal and nonverbal communication. Basis for rating: Interpersonal behavior during the course of interview.

0=Absent 1=Minimal 2=Mild 3=Moderate 4=Moderate severe 5=Severe 6=Extreme

N4 Passive/apathetic social withdrawal [_] 25
Diminished interest and initiative in social interactions due to passivity, apathy, anergy, or avolition. This leads to reduced interpersonal involvements and neglect of daily activities.

N5 Difficulty in abstract thinking [_] 26
Impairment in the use of the abstract-symbolic mode of thinking, as evidenced by difficulty in classification, forming generalizations, and proceeding beyond concrete or egocentric thinking in problem-solving tasks. Basis for rating: Responses to questions on similarities and proverb interpretation, and use of concrete vs. abstract mode during the course of interview.

N6 Lack of spontaneity and flow of conversation [_] 27
Reduction in the normal flow of communication associated with apathy, avolition, defensiveness, or cognitive deficit. This is manifested by diminished fluidity and productivity of the verbal-interactional process. Basis for rating: Cognitive-verbal processes observed during the course of interview.

N7 Stereotyped thinking [_] 28
Decreased fluidity, spontaneity, and flexibility of thinking, as evidenced in rigid, repetitious, or barren thought content. Basis for rating: Cognitive-verbal processes during the course of interview.

GENERAL PSYCHOPATHOLOGY SCALE (G)

G1 Somatic concern [_] 29
Physical complaints or beliefs about bodily illness or malfunctions. This may range from a vague sense of ill being to clear-cut delusions of catastrophic physical disease. Basic for rating: Thought content expressed in the interview.

G2 Anxiety [_] 30
Subjective experience of nervousness, worry, apprehension, or restlessness, ranging from excessive concern about the present or future to feelings of panic. Basis for rating: Verbal report during the course of interview and corresponding physical manifestations.

G3 Guilt feelings [_] 31
Sense of remorse or self-blame for real or imagined misdeeds in the past. Basis for rating: Verbal report of guilt feelings during the course of interview and the influence on attitudes and thoughts.

G4 Tension [_] 32
Overt physical manifestations of fear, anxiety, and agitation, such as stiffness, tremor, profuse sweating, and restlessness. Basis for rating: Verbal report attesting to anxiety and, thereupon, the severity of physical manifestations of tension observed during the interview.

G5 Mannerisms and posturing [_] 33
Unnatural movements or posture as characterized by an awkward, stilted, disorganized, or bizarre appearance. Basis for rating: Observation of physical manifestations during the course of interview as well as reports from primary care workers or family.

G6 Depression [_] 34
Feelings of sadness, discouragement, helplessness, and pessimism. Basis for rating: Verbal report of depressed mood during the course of interview and its observed influence on attitude and behavior.

G7 Motor retardation [_] 35
Reduction in motor activity as reflected in slowing or lessening of movements and speech, diminished responsiveness to stimuli, and reduced body tone. Basis for rating: manifestations during the course of interview as well as reports by primary care workers or family.

G8 Uncooperativeness [_] 36
Active refusal to comply with the will of significant others, including the interviewer, hospital staff, or family, which may be associated with distrust, defensiveness, stubbornness, negativism, rejection of authority, hostility, or belligerence. Basis for rating: Interpersonal behavior observed during the course of interview as well as reports by primary care workers or family.

G9 Unusual thought content [_] 37
Thinking characterized by strange, fantastic, or bizarre ideas, ranging from those which are remote or atypical to those which are distorted, illogical, and patently absurd. Basis for rating: Thought content expressed during the course of interview.

G10 Disorientation [_] 38
Lack of awareness of one's relationship to the milieu, including persons, place, and time, which may be due to confusion or withdrawal. Basis for rating: Responses to interview questions on orientation.

G11 Poor attention [_] 39
Failure in focused alertness manifested by poor concentration, distractibility from internal and external stimuli, and difficulty in harnessing, sustaining, or shifting focus to new stimuli.
Basis for rating: Manifestations during the course of interview.

b

G12 Lack of judgment and insight [_] 40
Impaired awareness or understanding of one's own psychiatric condition and life situation. This is evidenced by failure to recognize past or present psychiatric illness or symptoms, denial of need for psychiatric hospitalization or treatment, decisions characterized by poor anticipation of consequences, and unrealistic short-term and long-range planning. Basis for rating: Thought content expressed during the interview.

G13 Disturbance of volition [_] 41
Disturbance in the willful initiation, sustenance, and control of one's thoughts, behavior, movements, and speech. Basis for rating: thought content and behavior manifested in the course of interview.

G14 Poor impulse control [_] 42
Disordered regulation and control of action on inner urges, resulting in sudden, unmodulated, arbitrary, or misdirected discharge of tension and emotions without concern about consequences. Basis for rating: Behavior during the course of interview and reported by primary care workers or family.

G15 Preoccupation [_] 43
Absorption with internally generated thoughts and feelings and with autistic experiences to the detriment of reality orientation and adaptive behavior. Basis for rating: Interpersonal behavior observed during the course of interview.

G16 Active social avoidance [_] 44

WALDROP'S LIST OF MPA'S(MINOR PHYSICAL ANOMALIES)

REGION	SCORE
HEAD	
Fine, electric hair	
Very fine hair that will not comb down	**2**
Fine hair that is soon awry after combing	**1**
Two or more hair whorls	**0**
Head circumference outside normal range:	
>1.5 SD	**2**
>1.0 SD <1.5 SD	**1**
EYES	
Epicanthus	
Where upper and lower lids join the nose, point of union is:	
Deeply covered	**2**
Partly covered	**1**
Hypertelorism	
Approximate distance between tear ducts:	
>1.5 SD	**2**
>1.0 SD <1.5 SD	**1**
EARS	
Low-seated ears	
Point where ear joins head, not in line with the corner of eye and nose bridge:	
Lower by > 0.5 cm	**2**
Lower by < 0.5 cm	**1**
Adherent ear lobes	
Lower edge of ears extend:	
Upward and back toward crown of head	**2**
Straight back toward rear of neck	**1**
Malformed ears	**1**
Asymmetrical ears	**1**
Soft and pliable ears	**0**
MOUTH	
High-steepled palate	
Roof of mouth:	
Definitely steepled	**2**
Flat and narrow at the top	**1**
Furrowed tongue (one with deep groves)	**1**

Tongue with smooth-rough spots	**0**
HANDS	
Curved fifth finger Markedly curved inward toward other fingers Slightly curved inward toward other fingers	**2** **1**
Single transverse palmar crease	**1**
FEET	
Third toe longer than second: Definitely longer than second toe Appears equal in length to second toe	**2** **1**
Partial syndactylia of two middle toes	**1**
Big gap between first and second toes	**1**

3. CAMBRIDGE NEUROLOGICAL INVENTORY

It is a bedside neurological evaluation checklist which includes subtle neurological signs in six categories.

Instruction:rate as follows: 0 = normal, 0.5 = subthreshold, 1 = definitely abnormal, 2 = grossly abnormal. The sum of all individual tests is rated as the total score.

- Motor (including casual gait, tandem gait, Romberg's test)
- Complex motor coordination (including fist- edge- palm, alternating fist palm, diadochokinesis, finger opposition, rhythm tapping)
- Extra-ocular movements (visual tracking, gaze persistence)
- Other motor signs (mirror movements, motor persistence, heel shin test, synkinesis, tremors, and choreo-athetosis)
- Primitivereflexes (glabellartap, palmar grasp, palmomental, snoutreflexes).

- Sensory Integration (stereognosis, graphaesthesia, R-L disorientation, face – hand extinction)

1. MOTOR:

A. GAIT:*W*alking down the hall at least five paces.

METHOD: The patient is asked to balance for 15 s on each leg in turn. the patient is instructed to walk a few steps, stop, and block.

- **GAIT (EXAGGERATED ASSOCIATED MOVEMENT)**:

  Excess or reduced arm, leg, or trunk movement observed during walking

  SCORE:0 – absent, 1 - definitely present, 2 - markedly or pervasively present

B. TANDEM WALKING:

METHOD*:*Tandem gait is a gait (method of walking or running) where the toes of the back foot touch the heel of the front foot at eachstep(heel to toe for 10 paces).

C. ROMBERGS TEST:

METHOD: Ask the subject to stand erect with feet together and eyes closed. Stand close by as a precaution in order to stop the person from falling over and hurting himself or herself. Watch the movement of the body in relation to a perpendicular object behind the subject. A positive sign is noted when a swaying, sometimes irregular swaying and even toppling over occurs. The essential feature is that the patient becomes more unsteady with eyes closed.

SCORE:0 - normally still or slight weaving, 1 - widened base to stay in place

2 - Unable to stand still with eyes closed

2. COMPLEX MOTOR COORDINATION:

A. FIST EDGE PALM TEST:

METHOD: The patient is shown the task and then asked to perform the following: using a smooth and steady rhythmic pattern, to touch the table with the side of his/her list, the edge of his/her hand, and the palm of his/her hand. The patient is to break contact with the surface of the table between each change in hand position, but not to bring the arm back in full flexion. The patient is to re- peat this sequence of position changes 10 times.

(***Examiner****:* "Watch me do this."[Demonstrate five times, without verbal instruction.] "Now see if you can do it."[Repeat demonstration once if patient fails to perform.])

SCORE:

0 – Normal

1 - One or two minor mistakes, slow (C l/s) or clumsy (e.g., gross presence of associated movements in other parts of the hand and forearm), but no major disruption of movements

2 - Major disruption (e.g., total loss of rhythm or precision) or repeated breakdowns of Sequence

B. ALTERNATING FIST PALM TEST:

METHOD : The patient is to place both hands on the table, one hand palm down and the other hand in the shape of a fist. The patient is then asked to simultaneously alternate the position of his/her hands in a smooth and steady motion. The patient is asked to repeat this motion 15 times. Synchrony in change of position is observed.

(Examiner: "Watch me do this."[Demonstrate five times.] "Now see if you can do it."[Repeat demonstration once only if patient fails to perform.])

SCORE:

1. - Normal
2. - Minor mistakes, but no major desynchronization of movements
2 - Total desynchronization or repeated breakdown of sequence

C. DIADOCHOKINESIS*:*

METHOD *:* The patient is asked to make a fist with one hand and pat the back of the fist with the other hand alternately using the palm and the dorsum. Demonstrate five times; rate as finger-thumb opposition

SCORE:

0 – Normal

1 - One or two minor mistakes, slow (< l/s) orclumsy (e.g., gross presence of associated movements in other parts of the hand and forearm), but no major disruption of movements

2 - Major disruption (e.g., total loss of rhythm or precision or repeated breakdown of sequence

D. FINGER THUMB OPPOSITION*:*

METHOD *:*

The patient is asked to place both hands palm up with lingers fully extended on his/her legs. The patient is to start with his/her dominant hand and is to touch the tip of his/her fingers with the tip of his/her thumb, from index finger to little finger, returning to index finger, for a total to 10 repetitions.

SCORE:

0 – Normal

1 - One or two minor mistakes, slow (< l/s) or clumsy (e.g., gross presence of associated movements in other parts of the hand and forearm), but no major disruption of movements

2 - major disruption(e.g., total loss of rhythm or precision or repeated breakdown of sequence)

E. RHYTHM-TAPPING TEST*:*

METHOD*:*Ask the patient to re- produce exactly the series of taps heard while the patient has eyes closed (live trials using stimulus sequence suggested).

(Examiner*: "I* am going to tap some sound on the table like this; some taps are louder than others [demonstrate]. Could you tap the same rhythm back to me? Now close your eyes and listen.")

SCORE :0 - No error, 1 - One error (either in loudness or rhythm), 2 - Two or more errors

3. **EXTRA-OCULAR MOVEMENTS**

A. VISUAL TRACKING:

METHOD: Patient is asked to focus on a slowly moving target example (a pencil or pen) at a distance that the patient can focus on. The target is moved slowly in a horizontal and then in a vertical direction.

(Examiner: "Could you follow the [e.g., pen] with your eyes, keeping the head still")

SCORE : Extent of smooth pursuit eye movements:

Rate as positive if range of movementisclearlyrestricted. Do not rate if there is obvious proptosis or unilateral ophthalmoplegia.

Smoothness of smoothpursuiteyemovements: Rate as positive if notice ably "catchy" or jerky; onlyclear instances are rated.

B. GAZE PERSISTENCE:

METHOD:Patient is asked to fixhis/her gaze on an object (e.g., a pen) at a 45 angle in the horizontal plane of the right and left visual fields for 15 s each. ***(Examiner:*** "Could you keep looking at this [pen] with your head still, until I tell you to stop").

SCORE :

0 - no deviation from fixation

1 - deviation from fixation on one or two occasions but able to resume gaze

2 - deviation from fixation repeatedly, unable to resume gaze

4. OTHER MOTOR SIGNS:

A. HEEL SHIN TEST:

A test of voluntary motor coordination in which a personis asked to slow ly touch the knee with the heel of the opposite leg, which is altered in cerebella r dysfunction

B. CHOREO-ATHETOSIS:

Choreoathetosis is the occurrence of involuntary movements in a combination of chorea (irregular migrating contractions) and athetosis (twisting and writhing).

C. SYNKINESIS:

Synkinesis manifestedthrough involuntarymusculармovementsaccompa nyingvoluntarymovements. For example, voluntarysmilingwillinduce an involuntary contraction of the eyemusclescausing the eye to squintwhen the

subjectsmiles. The two cases of synkinesismostcommonlystudiedinvolve the facial muscles and the extraocular muscles.

- Eye closure with volitional contraction of mouth muscles
- Midfacial movements with volitional eye closure
- Neck tightness (Platysmal contraction) with volitional smiling.

D. TREMOR (POSTURAL) :

METHOD :Ratedwithpatient'sarmsoutstretched. Typicalresting, lowfrequency, parkinsonian "pill-rolling" tremorrated.

SCORE :0- No tremor, 1- Mild or occasionaltremor, 2 - Gross or persistent tremor

- **TREMOR (RESTING) :**

METHOD :

Ratedwithpatient'sarms by the side. Typicalresting, low-frequency, parkinsonian "pill-rolling" tremorrated.

SCORE :0- No tremor, 1- Mild or occasionaltremor,2 - Gross or persistent tremor

E. MIRROR MOVEMENTS(I)

METHOD:During the test for disdiadochokinesia, the patient's resting hand, holding a fist, is observed for mirror movements(pronation and supination)

SCORE:

0-No observable movement

1- Minor pronation or supination movements

2 - Consistent, distinctive pronation and supina- tion movements of the forearm

MIRROR MOVEMENTS (II):

METHOD : Thepatient's hand, whichis not performing the finger-thumb opposition test, isobserved for mirrormovements (tendency for the resting hand to move in a waysymmetrical to the performing hand)

SCORE :

0- No observable movement

1- Minor movements of the fingers

2- Consistent, distinctive movements of the lingers

5. PRIMITIVE REFLEXES*:*

A. SNOUT REFLEX*:*

METHOD*:*After explanation, the patient is instructed to relax, and the examiner rests a tongue depressor against the patient'sphiltrum and taps gently with the index finger. ***(Examiner:*** "Could you close your eyes and relax.iam going to tap gently on your mouth

SCORE:

0 - No contraction of the orbicularis orris

1 - Any contraction of the orbicularis orris

B. **GRASP REFLEX*:***

METHOD *:*The patient is instructed to relax and the palm is stroked lightly with the examiner's index linger. The sign should be demonstrable at least twice on repetition

SCORE:0 - No movement of patient's hand, 1- Some flexion of fingers, 2 - Examiner'sfingergrasped.

C. PALMOMENTAL REFLEX*:*

METHOD *:*The patient is instructedto relax. Muscle activity around the lips isobserved. The thenareminance (of the leftand then right hand in turn) is then strokedvigorously with a blunt pointed object. induced movement of the mentalis muscle is observed. If a positive response is gained from either hand, then it is rated as positive. If elicited unilaterally, please indicate in the space

provided, the stimulus in which side of the hand led to response in which side of the face.

(Examiner: "I am going to stroke the palm. Could you close your eyes and relax.")

SCORE : 1. No movement observed, 2. movement of the mentalis muscle

D. GLABELLAR TAP:

METHOD : Patient is instructed to fix his/her gaze on a distant point across the room or outside the room. After explanation, the patient is approached from above the forehead outside of the visualfield, and the examiner taps the glabellar region 10 times with the index linger. If the spontaneous blink rate is high, the patient is asked to relax, and the blinking pattern is carefully observed before the taps are applied.

(***Examiner***: "I amgoing to tapyourforeheadgently. Just try to relax and look ahead at the [fixation point].")

SCORE :0 - One to threeblinks (include partial blinks), 1 - More thanthreeblinkswithsome habituation (reduction of tendency to blinkwhentapped, No habituation at all

B **SENSORY INTEGRATION :**

A. STEREOGNOSIS*:*

METHOD *:*The patient, with eyes closed, isasked to identify an object placed in his/her hand. The patient is instructed to feel the object with one hand and to take as much time as needed. If the patient cannot name the object, he/she is asked to describe for what purpose the object is used. The patient starts with the dominant hand. Five trials are conducted for each hand. Objects are placed between thumb and index fingers for patients with proper care being taken to ensure that the patient does not look at the object. Suggested objects are: paper clip, coin, rubber band, eraser, screw, small seashell, or match).

SCORE :0 - No error, 1 - One error, 2 - Two or more errors

B. GRAPHESTHESIA*:*

METHOD*:*The patient, with eyes closed, is asked to identify the number written on his/her palm with a blunt point, the number being orientated facing

the patient. Five trials for each hand. Stimulus can be repeated once upon request by the patient or when the patient gives a response other than a number.

(Examiner: "I am going to trace a number on your palm; for example, this would be a [number]."[Demonstrate.] "Could you tell me what the number is, with your eyes closed.")

SCORE :0 - No error, 1 - One error, 2 - Two or more errors

C. LEFT-RIGHT ORIENTATION*:*

METHOD*:*

The patient is asked to point to his/her right foot, left hand; place his/her right hand to left shoulder, left hand to right ear; point to the examiner's left knee, then right elbow; with examiner's arms crossed, point to examiner's left hand with his/her right hand, and with examiner recrossing arms, point to examiner's right hand with his/her left hand.

SCORE :0 - No error, 1 - Left/right disorientation confined to perception of another person

2 - Left/right disorientation in self-body space

D. EXTINCTION*:*

METHOD*:*

The patient is seated, with handsresting palm down, on his/her knees and with eyes closed. The patient is told that he/she will be touched on the cheek, the hand, or both and that he/she is to say where he/she has been touched. If the patient names just one touch, he/she is asked (the first time this occurs only) if a touch is felt anywhere else. Simultaneous touching is performed in the following order: right cheek-left hand, left cheek-right hand, right cheek-right hand, left cheek-left hand, both hands, and both cheeks. Intact sensation to touch is confirmed in each test area beforehand.

SCORE :0 - No error, 1 - One error. 2- Two or more errors

MASTER CHART

KEY TO MASTER CHART

NAME	**:**	
AGE	**:**	**<20-1/20-30-2/30-40—3/40-50-4**
SEX	**:**	MALE –1 FEMALE-2
RESIDENCE	**:**	URBAN-1/SEMIURBAN-2/RURAL-3
EDUCATION	:	ILLITERATE-1/ < VSTD-2 / VI TO VIII STD-3/ IX TO X STD-4/XI TO XII STD DIPLOMA-5 / GRADUATE/PG - 6/PROFESSIONAL-7
OCCUPATION	**:**	UNEMPLOYED-1/UNSKILLED-2/SEMISKILLED-3/SKILLED-4/CLERICAL SHOP OWNER FARMER-5/SEMIPROFESSIONAL-6/PROFESSIONAL-7
MARITAL STATUS	**:**	UNMARRIED-1/MARRIED-2/WIDOW-3/DIVORCED-4/ SEPERATED-5

SOCIOECNOMIC STATUS :

UPPER-1 /UPPERMIDDLE-2/LOWERMIDDLE-3/UPPERLOWER-4/LOWER -5

AGE OF ONSET OF ILLNESS IN YEARS:

DURATION OF ILLNESS:

FAMILY HISTORY OF SCHIZOPHRENIA:

FIRST DEGREE RELATIVE-1 / SECOND DEGREE RELATIVE-2 / NO FAMILY HISTORY-3

MENTAL STATUS EXAMINATION:

DIAGNOSIS (ICD-10):

PANSS:

1. POSITIVE SCALE
2. NEGATIVE SCALE
3. GENERAL PHYCHOPATHOLOGY SCAL

DELUSION : PRESENT/ ABSENT

HALLUCINATION : PRESENT / ABSENT

WALDROP SCALE :
HEAD/EYES/EARS/MOUTH/HANDS/FEET

CAMBRIDGE NEUROLOGICAL INVENTORY:

MOTOR/MOTOR COORDINATION/EOM/OTHERS/PRIMITIVE REFLEXES/SENSORY INTEGRATION

S.no	ID.no	NAME	AGE	SEX	EDUCATION	SES	OCCUPATION	MARITAL STATUS	RESIDENCE	AGE OF ONSET	DURATION OF ILLNESS	FAMILY HISTORY	DELUSION	HALLUCINATION	POSITIVE	NEGATIVE	GEN. PSY	HEAD	EYES	EARS	MOUTH	HANDS	FEET	MPA total	MOTOR	MOTOR COORDINATION	EOM	OTHERS	PRIMITIVE REFLEXES	SENSORY INTEGRATION	SNS total
1		WINSTON 26	2	1	3	5	4	1	1	1	1	3	5	2	18	14	35	1	2	0	-	-	-	3	-	-	-	-	-	1	-
2		EMROSE 40	4	2	2	5	1	2	1	2	10	3	1	2	9	12	26	0	1	1	1	-	0	5	-	5	2	2	2	-	11
3		SARAVANAN 32	3	1	3	5	4	2	1	1	8	3	3	3	20	8	23	1	-	2	1	-	-	4	-	3	-	1	1	-	5
4		MEENA 35	3	2	3	4	1	2	2	2	6	2	6	5	16	12	20	0	-	0	1	-	1	2	-	2	1	-	-	-	3
5		RAVI 28	2	1	3	4	3	1	2	2	I EPISODE	2	3	3	18	8	18	0	1	0	1	-	-	2	-	2	-	2	2	-	6
6		RAGUNATH 42	4	1	2	4	2	2	3	2	16	1	4	3	10	16	19	0	-	2	0	-	-	2	-	5	1	2	2	1	11
7		MALLIGA 40	3	2	2	5	2	2	1	1	20	3	3	2	9	10	18	1	1	0	1	-	-	3	-	2	-	3	2	-	7
8		MOSES 30	2	1	2	5	4	2	1	2	3	3	3	2	9	7	17	0	-	2	-	-	-	2	-	-	3	-	-	-	3
9		KANNAN 35	3	1	4	4	3	2	3	1	9	3	3	4	18	10	10	0	2	1	1	-	0	4	-	-	-	3	-	-	3
10		VIJAYALAKSHMI 32	3	2	4	5	1	2	1	1	9	2	1	4	15	15	32	0	2	1	-	-	0	3	-	1	-	4	1	-	6
11		MEHABOOBSHAS 28	2	1	3	5	4	2	1	1	4	3	2	3	14	12	18	0	1	2	1	-	-	4	-	-	-	-	-	-	-
12		SUNDAR 42	3	1	5	4	1	1	1	2	14	3	6	2	19	17	26	0	1	2	1	-	1	5	-	4	2	1	-	-	7
13		PREMKUMAR 32	2	1	2	5	1	1	2	1	10	3	4	4	15	18	27	2	1	2	0	-	1	6	-	4	-	3	3	-	10
14		KUMARAN 33	3	1	2	5	2	3	1	1	9	3	2	1	18		18	0	2	0	1	-	-	3	-	-	-	-	1	-	1
15		LATHA 26	2	2	2	5	1	2	2	2	I EPISODE	3	1	2	16	10	20	1	-	0	1	-	-	2	-	3	-	3	-	-	6
16		RAJESH 32	3	1	3	4	5	2	2	2	4	2	1	2	12	15	18	1	-	0	1	-	-	2	-	1	-	3	-	2	6
17		SARASWATHI 26	2	2	1	5	1	2	2	2	I EPISODE	2	4	1	16	12	17	1	1	2	-	-	-	4	1	-	-	-	-	-	1
18		KALA 28	2	2	2	5	2	2	2	2	2	1	2	2	9	17	23	0	0	0	-	-	-	0	-	-	-	-	-	-	-
19		MAGESH 29	2	1	3	5	3	1	1	2	I EPISODE	3	2	1	16	12	28	1	0	2	-	-	1	4	-	2	1	2	-	-	5
20		ESWARI 32	2	1	3	4	3	2	3	2	6	1	2	1	15	12	28	0	0	0	-	-	1	1	-	1	-	-	-	-	-

21		THENMOZHI 20	2	2	3	3	1	1	2	1	I EPISODE	3	2	1	9	12	27	0	0	0	-	-	-	0	-	1	-	-	-	-	1
22		VASUKI 30	2	2	2	5	1	4	1	2	I EPISODE	4	2	1	10	17	32	0	0	0	1	-	-	1	1	2	-	2	1	-	6
23		RAFIQ 34	3	1	5	4	4	2	2	2	8	4	1	2	7	18	18	1	0	2	1	-	-	4	-	2	2	-	1	1	6
24		RAVIDOSS 38	2	1	3	4	3	2	2	2	8	3	2	1	15	8	25	0	0	0	-	-	1	1	-	1	-	1	-	-	2
25		GAJALAKSHMI 22	2	2	4	5	3	1	1	1	1	3	1	2	11	19	22	0	0	2	1	-	-	3	-	1	-	-	-	1	2
26		SIDDIQ 35	3	1	3	4	2	4	1	1	11	3	4	6	8	20	20	0	0	1	-	-	-	1	-	1	-	1	-	1	3
27		ANNALAKSHMI 38	3	2	2	5	2	4	1	2	8	3	3	5	11	13	21	1	0	0	1	-	2	4	-	-	-	1	-	-	1
28		DHANAPANDIAN 27	2	2	2	5	3	1	3	1	I EPISODE	3	4	4	11	13	23	0	0	0	-	-	-	0	-	2	2	1	-	-	5
29		SHANKAR 29	2	2	3	5	3	2	2	1	6	3	2	2	14	15	32	0	0	0	2	-	-	2	-	-	-	-	-	-	-
30		GOWRI 23	2	2	3	5	4	1	1	1	I EPISODE	3	3	3	16	21	28	0	1	1	0	-	2	4	-	2	2	3	1	2	10
31		ESWARAN 39	3	1	2	5	3	2	2	2	10	3	2	1	20	23	25	0	0	0	0	-	-	0	-	-	-	0	-	-	0
32		GAJENDRAN 44	4	1	3	5	2	2	3	2	14	2	4	3	18	21	38	0	0	0	0	-	-	0	-	1	-	-	-	2	3
33		RAGAMADULLAH 26	2	1	2	5	2	2	1	1	1	3	5	3	21	22	24	0	1	1	0	-	-	2	1	2	2	-	1	-	6
34		RANI 23	2	2	4	5	4	1	2	1	I EPISODE	3	2	1	16	10	22	0	0	0	0	-	-	0	3	2	-	1	-	-	6
35		AGASTHI 32	3	2	2	5	1	3	2	2	4	3	4	2	14	20	23	2	0	0	0	1	-	3	-	-	-	-	-	-	-
36		SUGUMAR 30	2	1	2	5	2	1	3	2	I EPISODE	2	2	1	22	12	20	0	0	0	0	-	1	1	2	-	1	2	-	-	5
37		MALAR 33	3	2	4	5	4	2	3	1	9	3	1	1	11	13	21	0	0	0	0	1	-	1	2	-	1	2	-	-	5
38		RAJESHWARI 23	2	2	2	5	2	1	3	1	I EPISODE	1	2	1	16	10	22	2	0	0	-	2	-	4	-	3	1	2	-	-	6
39		THANGARAJ 38	3	1	3	5	3	2	2	2	10	3	1	2	9	17	23	0	0	0	-	-	-	0	-	-	-	-	1	-	1
40		DHANDAPANI 29	2	1	2	5	3	2	2	2	I EPISODE	4	1	2	14	16	20	0	1	0	-	-	-	1	1	-	1	-	-	-	2
41		VASUDEVAN 35	3	1	3	4	1	2	2	2	5	2	5	3	15	14	20	0	0	1	-	-	-	1	3	3	-	2	-	1	9
42		LOKESH 25	2	1	5	4	4	1	1	1	8	3	3	16	12	`8	-	0	0	-	-	-	-	0	-	-	-	-	-	-	-

43		DEVI 30	3	2	2	4	1	2	1	1	9	4	2	12	9	18	-	0	0	0	-	-	-	0	-	-	-	-	-	-	-
44		MURUVAMMAL 35	3	2	2	5	1	2	1	1	20	3	5	5	21	10	22	0	0	2	-	-	-	2	-	-	3	-	-	-	3
45		NISHANTHINI 22	1	2	2	4	1	2	1	1	I EPISODE	3	3	2	8	8	18	0	0	0	1	1	-	2	-	-	-	-	-	-	-
46		SASIKALA 24	2	2	4	4	3	1	2	1	I EPISODE	2	3	1	15	9	19	0	0	0	0	0	1	1	1	-	-	-	-	-	1
47		MURUGAN 36	3	1	2	5	2	2	3	2	6	3	2	3	12	10	19	0	0	0	0	0	0	0	0	-	-	0	-	-	0
48		GOKUL 29	2	1	3	4	3	2	3	2	1	3	2	3	13	12	20	0	0	1	0	0	0	1	-	-	-	-	-	-	-
49		SALEEM 30	2	1	2	4	1	2	1	2	I EPISODE	3	4	3	16	10	20	1	0	0	0	0	0	1	2	1	-	-	-	-	3
50		IRFAN 32	3	1	2	5	3	2	1	2	2	3	4	1	16	8	18	0	0	0	0	0	0	0	-	1	-	-	-	-	1
51		KAMALA 28	2	2	2	5	2	2	2	2	2	1	4	3	9	16	19	0	0	0	1	-	-	1	3	1	-	-	-	-	4
52		ESWAR 28	3	1	1	5	2	2	3	2	6	3	2	2	16	8	20	0	0	1	1	-	-	2	1	-	-	-	-	1	2
53		GAJALAKSHMI 28	2	2	2	3	4	1	2	2	2	3	5	4	12	16	18	0	0	0	1	-	1	2	1	-	-	-	-	-	1
54		KUMARAVEL 39	3	1	3	5	4	2	1	2	9	2	2	2	19	9	18	0	0	0	0	-	-	0	-	-	1	-	-	-	1
55		VANITHA32	3	2	4	4	3	2	3	1	9	3	3	4	12	10	10	0	0	0	0	-	-	0	-	-	-	-	-	-	-
56		RAJESH 28	2	1	3	4	3	1	2	2	I EPISODE	2	3	3	8	12	18	1	0	1	0	-	-	2	2	3	1	-	-	1	7
57		DAVID 35	3	1	3	4	1	2	2	2	5	3	5	3	20	14	20	0	0	2	0	-	-	2	2	1	-	2	-	1	6
58		DHARMAN 25	2	1	5	4	4	1	1	1	8	3	4	3	16	12	18	0	0	0	0	-	-	0	-	-	-	-	-	-	-
59		KALA23	2	2	4	5	4	1	2	1	I EPISODE	3	2	1	12	10	22	0	0	0	0	-	-	0	-	-	-	-	-	-	-
60		RAJKUMAR 38	3	1	3	5	3	2	2	2	10	3	5	4	12	17	23	0	2	0	0	-	1	3	2	1	-	-	-	-	3
																		17	20	34	21	5	14		28	66	27	49	19	15	202

Sno	ID no	Name	ag	sex	educat	ses	cupati	RES			Heac	Eye	Ear	Han	Feet	Mot	MOTC	EON	Others			MPA	SNS	T	
1		MURUGAN 40	4	1	3	5	2	2	2			1								1			1		
2		KUMARAVEL 26	2	1	3	5	2	1	1											1				1	
3		MALLIGA 32	3	2	2	3	2	2	3									1						1	
4		RAVI 34	3	1	2	4	4	4	3											1			1		
5		RAGUNATH 36	3	1	3	5	1	2	3											1				1	
6		DEVI 22	2	2	3	5	2	1	3									2						2	
7		MHD SAFI 41	4	1	3	4	3	2	1																
8		ISMAIL 20	2	1	2	5	2	1	3																
9		GOWRI 33	3	2	4	4	4	4	1			1							1				3	1	
10		PARAMOHWARI 26	2	2	3	4	2	2	1																
11		LOGANATHAN 34	3	1	2	4	4	4	3									1						1	
12		VICTOR RAJ 29	2	1	2	5	3	2	2											1				1	
13		THANDAPANI 38	3	1	3	5	3	2	2																
14		RANI 23	2	2	2	5	2	1	3							2							3		
15		MANJULA 33	3	2	4	5	4	2	3																
16		SIVANESAN 33	3	1	2	5	2	1	3				1							1			1	1	
17		ALAMENU 33	3	2	2	5	1	3	2																
18		RAJISHVARI 23	2	2	4	5	4	1	2																
19		ABDUL 26	2	1	2	5	2	2	1								2	1						3	
20		GAJELAKSHMI 23	2	2	3	5	4	1	1																
21		SHANMUYAM 29	2	2	3	5	3	2	2																
22		DANASEKARAN 28	2	2	2	5	3	1	3										1					1	
23		ANNALAKSHMI 37	3	2	2	5	2	4	1																
24		GOMATHY 22	2	2	4	5	3	3	3					1									1		
25		RAGHAVENDER 38	2	1	3	4	3	2	2														1		
26		DHADAPANI 34	3	1	5	4	4	2	2									1						1	
27		VASUNDHRA 30	2	2	2	5	1	4	1																
28		THENGALAKSHMI20	2	2	3	3	1	2	2																
29		ESWARI 32	2	1	3	4	3	2	3																
30		MAGENDRAN 29	2	1	3	5	3	1	1														1		

31		RAMESH 32	3	1	3	4	5	2	2																	
32		LOGANAYAGI26	2	2	2	5	1	2	2																	
33		KUMAR 33	3	1	2	5	2	3	1																	
34		SARALA 28	2	1	3	5	4	2	1									1							1	
35		KANNAN 36	3	1	4	4	3	2	3															1		
36		MEENA 40	4	2	2	5	2	2	1											1					1	
37		VIMALA 40	4	2	2	5	2	2	1																	
38		THANGAM 32	3	2	2	5	1	3	2																	
39		PREMKUMAR 29	2	1	3	5	3	1	1											1					1	
40		RAVI 30	2	1	2	5	2	1	3																	
41		VALLANAR 35	3	1	3	4	1	2	2			1												1		
42		LEVI 25	2	1	5	4	4	1	1																	
43		DURGA 32	3	2	2	4	1	3	1																	
44		MANGALA 35	3	2	2	5	1	2	1									2		1					3	
45		NEELA 23	1	2	3	4	2	2	1																	
46		SALEEM 30	2	1	2	4	4	1	2										1							
47		JAYANTHI 24	2	2	4	4	3	1	2				2						1					2	1	
48		KALA 22	1	2	2	4	2	2	1																	
49		KAMATCHI 36	3	2	3	5	1	2	3									1							1	
50		KUMUDHA 40	4	2	2	5	1	2	1			1	1	2										3		
51		DHARANI 34	3	1	2	4	4	3	3																	
52		LOKESH 27	2	2	3	5	1	2	2																	
53		PARAMESHWARI 40	4	2	3	2	2	2	1									1							1	
54		LATHA 33	3	2	3	4	4	1	3					2										2		
55		KANCHANA 22	2	2	4	5	3	2	1																	
56		THANGARAJ 29	2	1	4	4	3	2	3									1						1	1	
57		VANI 32	3	2	2	4	1	3	1																	
58		JOHNSON 41	4	1	3	4	3	2	1																	
59		ALAMELU 28	2	1	3	5	4	2	1											1					1	
60		RAVI 36	3	1	2	5	4	3	1			1		1										3		