UNIT 1 SCHIZOPHRENIA: ETIOLOGY, NEUROCOGNITIVE FUNCTIONING AND INTERPERSONAL ASPECTS

Structure

1.0 INTRODUCTION

This unit deals with schizophrenia and focuses on neuropsychological aspects. The unit begins with the concept and description of schizophrenia and provides the incidence and prevalence rate of schizophrenia in India and abroad. It explains the characteristics of schizophrenia and indicates the onset of the disorder to be in the teens and adolescent years to young adulthood. The comorbidity of certain other disorders along with schizophrenia are discussed. Then we present a few important tests to clearly diagnose the schizophrenic disorder. The next section deals with the etiology of schizophrenia in which we discuss the role of genetics, prenatal factors, fetal growth abnormalities, lack of oxygen andsome of the important childhood antecedents. The causes then present the role of substance euse in schizophrenia and tin this we discuss the role of cannabis, amphetamines, hallucinogens, tobacco etc. Social fac\tors, urbanicity, relationships within family and personality types are also considered as causes in the onset of schizophrenia. Then we discuss the neurocognitive functioning of schizophrenia followed by treatment of schizophrenic disorder. The treat ment includes hospitalisation, medicines, cognitive behaviour therapy and family therapy.

1.1 OBJECTIVES

On completing this unit, you will be able to :

- Define schizophrenia and describe the characteristic features;
- Explain the etiology of schizophrenia;
- Describe how substance use cause schizophrenic symptoms;
- Explain the neurocognitive functioning aspects of schizophrenia;
- Elucidate the treatment of schizophrenia; and
- Analyse the importance of various psychological therapies.

1.2 CONCEPT AND DESCRIPTION OF SCHIZOPHRENIA

Schizophrenia is a severe, psychotic disorder. People who have it may hear voices, see things that are not there or believe that others are reading or controlling their minds. In men, symptoms usually start in the late teens and early 20s. They include hallucinations, such as visual hallucinations (seeing things which are not there), and auditory hallucinations (hearing things which are not present), and delusions such as false beliefs that others are plotting or conspiring against them while actually there is no such thing.

1.2.1 Incidence of Schizophrenia

The incidence of schizophrenia is estimated to be one percent to one and a half percent of the U.S. population being diagnosed with it over the course of their lives. In India, according to NIMH, it is estimated that 4.3 to 8.7 million people (a rough estimate based on the population) suffer from schizophrenia. According to Barua et al (2006), the prevalence rate of schizophrenia in India is 1%.

1.2.2 Characteristics of Schizophrenia

While there is no known cure for schizophrenia, it is a treatable disorder. Most of those afflicted by schizophrenia respond to drug therapy, and many are able to lead productive

and fulfilling lives. It is characterised by a constellation of distinctive and predictable symptoms. The symptoms that are most commonly associated with the disease are called positive symptoms, that denote the presence of grossly abnormal behaviour. These include thought disorder, delusions, and hallucinations.

Thought disorder is the diminished ability to think clearly and logically. Often it is manifested by disconnected and nonsensical language that renders the person with schizophrenia incapable of participating in conversation, contributing to the person's alienation from his family, friends, and society.

Delusions are common among individuals with schizophrenia. An affected person may believe that he is being conspired against (called "paranoid delusion"). Broadcasting, describes a type of delusion in which the individual with this illness believes that his thoughts can be heard by others.

Hallucinations are perceptual disorder, in which one could suffer from auditory hallucination, visual hallucination and tactile hallucination. Sometime the voices that the schizophrenic hears may describe the person's actions, warn him of danger or tell him what to do. At times the individual may hear several voices carrying on a conversation.

Less obvious than the "positive symptoms" but equally serious are the deficit or negative symptoms that represent the absence of normal behaviour. These include flat or blunted affect (i.e. lack of emotional expression), apathy, and social withdrawal).

Schizophrenia is a mental disorder characterised by a disintegration of thought processes and of emotional responsiveness. It most commonly manifests as auditory hallucinations, paranoid or bizarre delusions, or disorganised speech and thinking, and it is accompanied by significant social or occupational dysfunction.

1.2.3 Onset of Schizophrenia

It can affect anyone at any point in life, it is somewhat more common in those persons who are genetically predisposed to the disorder. The first psychotic episode generally occurs in late adolescence or early adulthood. The probability of developing schizophrenia as the offspring of two parents, neither of whom has the disease, is 1 percent. The probability of developing schizophrenia as the offspring of one parent with the disease is approximately 13 percent. The probability of developing schizophrenia as the offspring of both parents with the disease is approximately 35%. Persons with schizophrenia develop the disease between 16 and 25 years of age.

This disorder has its onset around adolescent years to 20s to early 30s. This disorder makes the person behave in the weirdest manner that persons with this disorder are also stigmatized. As generally thought to be, schizophrenia is not a split personality, it is a rare and very different disorder. Like cancer and diabetes, schizophrenia has a biological basis. It is not caused by bad parenting or personal weaknesses.

Onset is uncommon after age 30, and rare after age 40. In the 16-25 year old age group, schizophrenia affects more men than women. In the 25-30 year old group, the incidence is higher in women than in men.

The onset of symptoms typically occurs in young adulthood, with a global lifetime prevalence of about 0.3–0.7%. Diagnosis is based on observed behaviour and the patient's reported experiences.

1.2.4 Neurocognitive Explanations of Schizophrenia

Increasingly, neuro cognitive paradigms are used to study patients with schizophrenia.

With such paradigms, the cognitive abnormalities in schizophrenia are characterised by means of experimental and clinical tests. These techniques have indicated that some types of cognitive impairment are not only reliably present in schizophrenia, but are also central and enduring features of the disorder. This focuses on certain recent advances in

i) characterising the precise nature of cognitive impairments in schizophrenia,

ii) understanding the implications of these for treatment, given the course and relationship to outcome of these variables, and

iii) on novel applications of neuro cognitive approaches to the genetics of schizophrenia.

1.2.5 Comorbidity

Genetics, early environment, neurobiology, and psychological and social processes appear to be important contributory factors; some recreational and prescription drugs appear to cause or worsen symptoms. Current research is focused on the role of neurobiology, although no single isolated organic cause has been found.

The many possible combinations of symptoms have triggered debate about whether the diagnosis represents a single disorder or a number of discrete syndromes. Despite the etymology of the term from the Greek roots *skhizein* (to split) and *phrēn, phren-* (mind), schizophrenia does not imply a "split mind" and it is not the same as dissociative identity disorder, also known as "multiple personality disorder" or "split personality" a condition with which it is often confused in public perception.

The disorder is thought mainly to affect cognition, but it also usually contributes to chronic problems with behaviour and emotion. People with schizophrenia are likely to have additional (comorbid) conditions, including major depression and anxiety disorders. The lifetime occurrence of substance abuse is almost 50%. Social problems, such as long-term unemployment, poverty and homelessness, are common. The average life expectancy of people with the disorder is 12 to 15 years less than those without, the result of increased physical health problems and a higher suicide rate (about 5%).

It is possible that nearly every cognitive function of a schizophrenic patient is impaired, and to an equivalent degree three functions play a role that is early descriptions of the clinical phenomenology of schizophrenia emphasized impairment of volitional attention. This clinical observation has been amply supported by many years of experimental study with the use of a wide variety of tasks.

1.2.6 Tests for Schizophrenia

Recent models have sharpened the lines between selective attention, shifting attention, and biasing for and encoding relevant target information. We investigate some of these functions by examining three tasks, viz.,

i) the Continuous Performance Test (CPT),

ii) the Covert Visual Orienting test, and the

iii) Stroop Test.

The classic test of selective attention is the Stroop color word task, in which a word (e.g., red) can be printed in incongruent colors (e.g., green). Depending on instructions, the task is either to name the actual word or name the ink color in which the word is written.

The attentional task requires the subject to focus selectively on one dimension of the stimulus and ignore or inhibit contextually inappropriate response tendencies. Normal

subjects are slowed when they have to name a color of ink that is incongruent with the word because they have to inhibit their over learned tendency of reading the word.

Schizophrenic patients may have differential problems on this task in reaction time or accuracy, a finding that has been taken to suggest that they have disproportionate difficulty in inhibiting over learned tendencies (of reading the word), and may be susceptible to failure in conditions of cognitive conflict more generally, because they are unable to use the contextual information appropriately.

Secondly Memory impairment is often the most striking feature of neuro cognitive impairment in schizophrenia. Newer work has sought to determine if patients with schizophrenia have qualitative abnormalities in specific stages of mnemonic processing. Toward this end, Elvevaag and colleagues conducted an encoding study in which subjects had to state whether the letter *a* was present in a word (shallow level)or make a decision as to whether the word represented a living thing or not (deep level).

Much previous work has demonstrated that words are recalled better when they are encoded deeply. Preliminary results indicated that although patients' performance was worse than that of controls, they showed the same benefit of deep encoding. In other words, although impairment in any given cognitive process may exact only a small cost in social and vocational functioning, a constellation of impairments may be disabling and result in the emergence of psychosis. Thus, understanding the genetic architecture of individual processes may well be critical.

Self Assessment Questions

1) Define schizophrenia and bring out its important features.

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2) What is the prevalence and incidence rate of schizophrenia?

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3) Describe the onset of schizophrenia.

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4) Give the neurocognitive explanations of schizophrenia.

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5) What is meant by comorbidity? What are the disorders associated with schizophrenia?

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6) Describe the tests for schizophrenia.

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1.3 ETIOLOGY OF SCHIZOPHRENIA

The causes of schizophrenia have been the subject of much debate, with various factors proposed and discounted or modified. The language of schizophrenia research under the medical model is scientific. Such studies suggest that genetics, prenatal development, early environment, neurobiology and psychological and social processes are important contributory factors.

Current psychiatric research into the development of the disorder is often based on a neurodevelopmental model (proponents of which see schizophrenia as a syndrome. However, schizophrenia is diagnosed on the basis of symptom profiles. Neural correlates do not provide sufficiently useful criteria "Current research into schizophrenia has remained highly fragmented, much like the clinical presentation of the disease itself"

1.3.1 Genetics

Genetic vulnerability and environmental factors can act in combination to result in diagnosis of schizophrenia. Research suggests that genetic vulnerability to schizophrenia is multi factorial, caused by interactions of several genes.

Both individual twin studies and meta analyses of twin studies estimate the heritability of risk for schizophrenia to be approximately 80%. Concordance rates between monozygotic twins was close to 50%, whereas dizygotic twins was 17%. Adoption studies have also indicated a somewhat increased risk in those with a parent with schizophrenia even when raised apart. Studies suggest that the phenotype is genetically influenced but not genetically determined. Also the variants in genes are generally within the range of normal human variation and have low risk associated with them each individually. Some interact with each other and with environmental risk factors and that they may not be specific to schizophrenia.

1.3.2 Prenatal

It is well established that obstetric complications or events are associated with an increased chance of the child later developing schizophrenia, although overall they constitute a non specific risk factor with a relatively small effect.

Obstetric complications occur in approximately 25 to 30% of the general population and the vast majority do not develop schizophrenia, and likewise the majority of individuals with schizophrenia have not had a detectable obstetric event.

Nevertheless, the increased average risk is well replicated, and such events may moderate the effects of genetic or other environmental risk factors. The specific complications or events most linked to schizophrenia, and the mechanisms of their effects, are still under examination.

One epidemiological finding is that people diagnosed with schizophrenia are more likely to have been born in winter or spring. However, the effect is not large. Explanations have included a greater prevalence of viral infections at that time, or a greater likelihood of vitamin D deficiency. A similar effect (increased likelihood of being born in winter and spring) has also been found with other, healthy populations, such as chess players.

1.3.3 Fetal Growth

Lower than average birth weight has been one of the most consistent findings, indicating slowed fetal growth possibly mediated by genetic effects. Almost any factor adversely affecting the fetus will affect growth rate, however, so the association has been described as not particularly informative regarding causation. In addition, the majority of birth cohort studies have failed to find a link between schizophrenia and low birth weight or other signs of growth retardation.

1.3.4 Hypoxia

It has been hypothesized since the 1970s that brain hypoxia (low oxygen levels) before, at or immediately after birth may be a risk factor for the development of schizophrenia.

Hypoxia is now being demonstrated as relevant to schizophrenia in animal models, molecular biology and epidemiology studies. One study in Molecular Psychiatry was able to differentiate 90% of schizophrenics from controls based on hypoxia and metabolism.

Hypoxia has been recently described as one of the most important of the external factors that influence susceptibility, although studies have been mainly epidemiological. Such studies place a high degree of importance on hypoxic influence. Fetal hypoxia, in the presence of certain unidentified genes, has been correlated with reduced volume of the hippocampus, which is in turn correlated with schizophrenia.

Although most studies have interpreted hypoxia as causing some form of neuronal dysfunction or even subtle damage, it has been suggested that the physiological hypoxia that prevails in normal embryonic and fetal development, or pathological hypoxia or ischemia, may exert an effect by regulating or deregulating genes involved in neurodevelopment.

1.3.5 Other Factors

There is an emerging literature on a wide range of prenatal risk factors, such as prenatal stress, intrauterine (in the womb) malnutrition, and prenatal infection. Increased paternal age has been linked to schizophrenia, possibly due to “chromosomal aberrations and mutations of the aging germline.”

Maternal-fetal rhesus or genotype incompatibility has also been linked, via increasing the risk of an adverse prenatal environment. Also, in mothers with schizophrenia, an increased risk has been identified via a complex interaction between maternal genotype,

maternal behaviour, prenatal environment and possibly medication and socio-economic factors.

There may be an association between celiac disease (gluten intolerance) and schizophrenia in a small proportion of patients, though large randomized controlled trials and epidemiological studies will be needed before such an association can be confirmed.

Withdrawal of gluten from the diet is an inexpensive measure which may improve the symptoms in a small ($\leq$3%) number of schizophrenic patients.

1.3.6 Infections

Numerous viral infections, in utero or in childhood, have been associated with an increased risk of later developing schizophrenia.

Influenza has long been studied as a possible factor. A 1988 study found that individuals who were exposed to the Asian flu as second trimester fetuses were at increased risk of eventually developing schizophrenia. This result was corroborated by a later British study of the same pandemic, but not by a 1994 study of the pandemic in Croatia. A Japanese study also found no support for a link between schizophrenia and birth after an influenza epidemic.

Polio, measles, varicella-zoster, rubella, herpes simplex virus type 2, maternal genital infections, Borna disease virus, and more recently *Toxoplasma gondii*, have been correlated with the later development of schizophrenia. Psychiatrists E. Fuller Torrey and R.H. Yolken have hypothesized that the latter, a common parasite in humans, contributes to some, if not many, cases of schizophrenia.

1.3.7 Childhood Antecedents

In general, the antecedents of schizophrenia are subtle and those who will go on to develop schizophrenia do not form a readily identifiable subgroup, which would lead to identification of a specific cause. Average group differences from the norm may be in the direction of superior as well as inferior performance.

Overall, birth cohort studies have indicated subtle nonspecific behavioural features, some evidence for psychotic like experiences (particularly hallucinations), and various cognitive antecedents. There have been some inconsistencies in the particular domains of functioning identified and whether they continue through childhood and whether they are specific to schizophrenia.

A prospective study found average differences across a range of developmental domains, including reaching milestones of motor development at a later age, having more speech problems, lower educational test results, solitary play preferences at ages four and six, and being more socially anxious at age 13.

1.4 SUBSTANCE USE

The relationship between schizophrenia and drug use is complex, meaning that a clear causal connection between drug use and schizophrenia has not been found. There is strong evidence that using certain drugs can trigger either the onset or relapse of schizophrenia in some people. It may also be the case, however, that people with schizophrenia use drugs to overcome negative feelings associated with both the commonly prescribed antipsychotic medication and the condition itself, where negative emotion, paranoia and anhedonia are all considered to be core features.

The rate of substance use is known to be particularly high in this group. In a recent study, 60% of people with schizophrenia were found to use substances and 37% would be diagnosable with a substance use disorder.

1.4.1 Cannabis

There is some evidence that cannabis use can contribute to schizophrenia. Some studies suggest that cannabis is neither a sufficient nor necessary factor in developing schizophrenia, but that cannabis may significantly increase the risk of developing schizophrenia and may be, among other things, a significant causal factor. Nevertheless, some previous research in this area has been criticised as it has often not been clear whether cannabis use is a cause or effect of schizophrenia. To address this issue, a recent review of studies from which a causal contribution to schizophrenia can be assessed has suggested that cannabis statistically doubles the risk of developing schizophrenia on the individual level, and may, assuming a causal relationship, be responsible for up to 8% of cases in the population.

1.4.2 Amphetamines and other Stimulants

As amphetamines trigger the release of dopamine and excessive dopamine function is believed to be responsible for many symptoms of schizophrenia (known as the dopamine hypothesis of schizophrenia), amphetamines may worsen schizophrenia symptoms. In addition, amphetamines are known to cause a stimulant psychosis in otherwise healthy individuals that superficially resembles schizophrenia, and may be misdiagnosed as such by some healthcare professionals.

1.4.3 Hallucinogens

Drugs such as ketamine, PCP, and LSD have been used to mimic schizophrenia for research purposes. Using LSD and other psychedelics as a model has now fallen out of favour with the scientific research community, as the differences between the drug induced states and the typical presentation of schizophrenia have become clear. The dissociatives ketamine and PCP, however, are still considered to produce states that are remarkably similar however, and are considered to be even better models than stimulants since they produce both positive and negative symptoms.

1.4.4 Tobacco Use

People with schizophrenia tend to smoke significantly more tobacco than the general population. The rates are exceptionally high amongst institutionalised patients and homeless people. In a UK census from 1993, 74% of people with schizophrenia living in institutions were found to be smokers A 1999 study that covered all people with schizophrenia in Nithsdale, Scotland found a 58% prevalence rate of cigarette smoking, to compare with 28% in the general populatio

Despite the higher prevalence of tobacco smoking, people diagnosed with schizophrenia have a much lower than average chance of developing and dying from lung cancer. While the reason for this is unknown, it may be because of a genetic resistance to the cancer, a side effect of drugs being taken, or a statistical effect of increased likelihood of dying from causes other than lung cancer.

A 2003 study of over 50,000 Swedish conscripts found that there was a small but significant protective effect of smoking cigarettes on the risk of developing schizophrenia later in life. While the authors of the study stressed that the risks of smoking far outweigh these minor benefits, this study provides further evidence for the 'self-medication' theory

of smoking in schizophrenia and may give clues as to how schizophrenia might develop at the molecular level.

1.4.5 Social Adversity

The chance of developing schizophrenia has been found to increase with the number of adverse social factors (e.g. indicators of socio-economic disadvantage or social exclusion) present in childhood. Stressful life events generally precede the onset of schizophrenia. A personal or recent family history of migration is a considerable risk factor for schizophrenia, which has been linked to psychosocial adversity, social defeat from being an outsider, racial discrimination, family dysfunction, unemployment and poor housing conditions.

Childhood experiences of abuse or trauma are risk factors for a diagnosis of schizophrenia later in life. Recent large-scale general population studies indicate the relationship is a causal one, with an increasing risk with additional experiences of maltreatment although a critical review suggests conceptual and methodological issues require further research There is some evidence that adversities may lead to cognitive biases and/or altered dopamine neurotransmission, a process that has been termed "sensitisation".

Specific social experiences have been linked to specific psychological mechanisms and psychotic experiences in schizophrenia. In addition, structural neuroimaging studies of victims of sexual abuse and other traumas have sometimes reported findings similar to those sometimes found in psychotic patients, such as thinning of the corpus callosum, loss of volume in the anterior cingulate cortex, and reduced hippocampal volume.

1.4.6 Urban City

A particularly stable and replicable finding has been the association between living in an urban environment and the development of schizophrenia, even after factors such as drug use, ethnic group and size of social group have been controlled for.[115] A recent study of 4.4 million men and women in Sweden found a 68%–77% increased risk of diagnosed psychosis for people living in the most urbanized environments, a significant proportion of which is likely to be described as schizophrenia

1.4.7 Close Relationships

Evidence is consistent that negative attitudes from others increase the risk of schizophrenia relapse, in particular critical comments, hostility, authoritarian, and intrusive or controlling attitudes (termed 'high expressed emotion' by researchers).

1.4.8 Environment

Pollack and Malzberg studied 175 patients of this disease and reached to the conclusion that environment plays a bigger part in creating this disease than does heredity, and so psychologist today refutes the importance of environment in causing of schizophrenia.

1.4.9 Instinct for Self-respect

According to McDougall, when the patient is unable to find proper and desirable expression for his instincts of self-respect, he becomes a prey to schizophrenia.

1.4.10 Personality Type

It is the opinion of some psychologists that only a certain personality type is susceptible to schizophrenic tendencies, primarily the introverted type of individual. But this concept

of the personality type being more prone to schizophrenia has also not found much of the following among the thinkers.

1.5 NEUROCOGNITIVE FUNCTIONING ASPECTS IN SCHIZOPHRENIA

In neuropsychology, that is, inferring regional brain dysfunction based on poor performance on putatively localising neuropsychological tests. On the basis of such an approach, various authors have concluded that schizophrenia is characterised by cognitive test profiles indicative of dysfunction of the frontal lobe, temporal lobe, left or right hemisphere, basal ganglia, etc. This lack of consensus may reflect the heterogeneity of schizophrenia, and may also be a result of the relatively poor localising ability of many standard neuropsychological instruments. A variety of brain regions and associated cognitive functions have thus been implicated in the psychopathology that characterises schizophrenia.

In general, the strongest camps to emerge have been those that claim a disproportionate impairment of memory functioning and relatively selective executive dysfunction. Others have reported more widespread neuropsychological dysfunction. An extreme case is put by Meehl who stated that impaired cognitive test performance in patients with schizophrenia may be an epiphenomenon, for example, reflecting lack of motivation or distraction by hallucinations. In order to convince skeptics that the neuropsychological impairment is important, one would have to demonstrate a clear relationship between cognitive test performance and 'real-life' functional outcome.

An important review of this area was published by Green (1996), who evaluated studies that used cognitive measures as predictors and correlates of functional outcome. The most consistent finding to emerge was that verbal memory functioning was associated with all types of functional outcome. It was observed that verbal memory showed the greatest impairment in the meta-analysis whereas sustained attention or vigilance was found to be related to social problem solving and skill acquisition.

Interestingly, psychotic symptoms were not significantly associated with outcome measures in any of the studies that were reviewed. Green (1996) concluded that deficiencies in verbal memory and vigilance may prevent patients from attaining optimal adaptation and hence may act as rate limiting factors in terms of rehabilitation. It is interesting to observe that where the patients showed symptomatic improvement with clozapine treatment, there was no associated improvement in neuropsychological functioning.

Velligan et al (1997) confirmed a poor correlation between symptomatology and ability to perform daily living tasks. However, cognitive impairment predicted over 40% of the variance in scores on a functional needs assessment rating scale.

Addington & Addington (1999) used a novel video taped measure of interpersonal problem solving skills. In a study of 80 out patients with schizophrenia, they found that better cognitive flexibility and verbal memory were positively associated with interpersonal problem solving ability.

In summary all these studies taken together, strongly support the view that cognitive impairment in schizophrenia is directly related to social deficits and functional outcome for many patients.

Schizophrenia symptoms may more clearly relate to disordered patterns of information processing. Liddle & Morris (1991) conducted a seminal study in this area where they

assessed a group of patients with chronic schizophrenia using a battery of neuropsychological tests allegedly sensitive to frontal lobe dysfunction.

Signs and symptoms were clustered into three syndromes:

- psychomotor poverty,
- disorganisation and
- reality distortion.

Scores for the disorganisation syndrome were associated with impairment on tests that required the subject to inhibit a well established but inappropriate response. Ratings for the psychomotor poverty syndrome were found to be associated with slowness of mental activity.

More recently, Baxter & Liddle (1998) confirmed that the psychomotor poverty syndrome was associated with psychomotor slowing, and disorganisation was associated with impaired performance on the Stroop Attentional Conflict task, but not with other tests of cognitive inhibition.

This led the authors to conclude that the disorganisation syndrome might be associated with a specific difficulty in suppressing irrelevant verbal responses. This approach is appealing, because it tries to integrate neuropsychology with the clinical features of schizophrenia. Pursuing this approach to a more specific level would result in an attempt to explain specific signs or symptoms in terms of aberrant information processing.

As an illustration of this approach, McKenna (1991) proposed that delusions may arise as a consequence of a dysfunctional semantic memory system. Again, this hypothesis has intuitive appeal, as delusions by definition must represent false belief, knowledge. However, efforts to try to provide convincing evidence of a causal relationship between a specific neuropsychological abnormality and a particular sign or symptom have, as yet, been disappointing.

The resulting cognitive data were subjected to cluster analysis and five cognitive clusters emerged:

- selective executive dysfunction;
- normative function;
- executive and motor deficits;
- dementia/multi-focal disturbance; and
- relatively selective motor deficits.

Heinrichs & Awad (1993) proposed that cluster analysis of cognitive test data may thus have promise in reducing and clarifying the heterogeneity of schizophrenia, and concluded that several patterns of neurocognitive dysfunction may underlie schizophrenia, thus contributing to the heterogeneity of the illness and its variable functional outcome.

Frith (1992) has also proposed a fascinating theoretical model, where he relates specific signs and symptoms to particular information processing abnormalities. For example, he proposes that the inability to generate spontaneous (willed) intentions can lead to poverty of action, perseveration and inappropriate action. In contrast, the inability to monitor the beliefs and intentions of others can lead to delusions of reference, paranoid delusions, certain kinds of incoherence and third-person hallucinations.

Self Assessment Questions

1) Discuss the etiology of schizophrenia.

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2) What are the childhood antecedents that cause schizophrenia?

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3) Discuss social adversity and urbanicity as causes of schizophrenia.

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4) Discuss etiology of schizophrenia in terms of substance use.

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5) Describe the neurocognitive functioning aspects of schizophrenia.

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1.6 TREATMENT OF SCHIZOPHRENIA

1.6.1 Hospitalisation

Hospitalisation may occur with severe episodes of schizophrenia. This can be voluntary or (if mental health legislation allows it) involuntary (called civil or involuntary commitment). Long term inpatient stays are now less common due to the policy of deinstitutionalisation, yet we still have large number of patients admitted to institutions for longer period of stay .

Following (or in lieu of) a hospital admission, support services available can include drop in centers, visits from members of a community mental health team or Assertive Community Treatment team, supported employment and patient led support groups.

1.6.2 Medication

The mainstay of psychiatric treatment for schizophrenia is an antipsychotic medication. These can reduce the "positive" symptoms of psychosis. Most antipsychotics take around 7–14 days to have their main effect. Risperidone (trade name Risperdal) is a common atypical antipsychotic medication.

Treatment was revolutionized in the mid 1950s with the development and introduction of the first antipsychotic chlorpromazine. Others such as haloperidol and trifluoperazine soon followed.

Though expensive, the newer atypical antipsychotic drugs are usually preferred for initial treatment over the older typical antipsychotics; they are often better tolerated and associated with lower rates of tardive dyskinesia, although they are more likely to induce weight gain and obesity-related diseases. Of the atypical antipsychotics, olanzapine and clozapine are the most likely to induce weight gain. The effect is more pronounced if high doses of olanzapine are used.[11] Smaller amounts of weight gain are induced by risperidone and quetiapine. Ziprasidone and aripiprazole are considered to be weight neutral antipsychotics.

It remains unclear whether the newer antipsychotics reduce the chances of developing neuroleptic malignant syndrome, a rare but serious and potentially fatal neurological disorder most often caused by an adverse reaction to neuroleptic or antipsychotic drugs. In combination with drug treatment, Psychosocial and Psychotherapy are also widely recommended and used in the treatment of schizophrenia.

1.6.3 Cognitive Behavioural Therapy (CBT)

CBT is used to target specific symptoms and improve related issues such as the therapy advanced from its initial applications in the mid 1990s, more recent reviews clearly show CBT is an effective treatment for the psychotic symptoms of schizophrenia.

Another approach is cognitive remediation therapy, a technique aimed at remediating the neurocognitive deficits sometimes present in schizophrenia. Based on techniques of neuropsychological rehabilitation, early evidence has shown it to be cognitively effective, resulting in the improvement of previous deficits in psychomotor speed, verbal memory, nonverbal memory, and executive function, such improvements being related to measurable changes in brain activation as measured by fMRI.

A similar approach known as cognitive enhancement therapy, which focuses on social cognition as well as neurocognition, has shown efficacy. CBT, an evidenced based practice, is now offered in community mental health agencies and hospitals.

1.6.4 Metacognitive Training

In view of a many empirical findings suggesting deficits of metacognition (thinking about one's thinking, reflecting upon one's cognitive process) in patients with schizophrenia, metacognitive training (MCT) is increasingly adopted as a complementary treatment approach.

MCT aims at sharpening the awareness of patients for a variety of cognitive biases (e.g. jumping to conclusions, attributional biases, over-confidence in errors), which are implicated in the formation and maintenance of schizophrenia positive symptoms (especially delusions), and to ultimately replace these biases with functional cognitive strategies.

1.6.5 Family Therapy or Education

This addresses the whole family system of an individual with a diagnosis of schizophrenia, has been consistently found to be beneficial, at least if the duration of intervention is longer term. Aside from therapy, the impact of schizophrenia on families and the burden on careers has been recognised, with the increasing availability of self help books on the subject.

There is also some evidence for benefits from social skills training, although there have also been significant negative findings. Some studies have explored the possible benefits of music therapy and other creative therapies.

1.7 UNIT END QUESTIONS

1) Define the etiology for schizophrenia from genetic, hereditary and biological point of view?

2) What are the environmental factors that cause schizophrenia?

3) How neurofunctioning deficts affects the life of individual and cause schizophrenia?

4) Discuss some of the treatment approaches to schizophrenia.

5) Discuss hospitalisation and medication as treatment techniques for schizophrenia.

6) Describe family therapy and education as important treatment programme for schizophrenia.

1.8 LET US SUM UP

In the lay imagination, schizophrenic patients experience problems in living because they are divided against themselves, out of touch with reality, and disorganised. The view of scientists, once not altogether different, has changed.

Not only have the symptoms been defined and codified, but the neurobiological underpinnings of the disorder have begun to be described. Emerging also is a view in which cognitive impairments may be a relatively central feature of the disorder.

Cognitive impairments are involved in the genetic etiology of schizophrenia. They seem enduring in that they are present for much of the clinical history and are associated with outcome. Cognitive impairments also may have a relatively well delineated profile in which executive, memory, and attentional deficits are prominent.

As explained, schizophrenia is very disabling. But as research progresses treatment is slowly but surely becoming more and more effective. Fewer patients have to be kept in hospitals and damage to the brain is not as severe.

Scientists discovered the effects of oestrogen, and learned it could be used as a medicine (though long term medications using oestrogen have side effects). They discovered age and gender differences, and learned that there were structural changes even at a cellular level.

In conclusion, though schizophrenia is disabling and sometimes even deadly, modern science has made many medical breakthroughs, and perhaps, if it is even possible, scientists may discover a complete or partial cure.

1.9 SUGGESTED READINGS

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