INTRODUCTION

Psychiatric hospitals, also known as mental hospitals are hospitals specializing in the treatment of serious mental disorders, such as depression, schizophrenia, and bipolar disorder. Psychiatric hospitals vary widely in their size and grading. Some hospitals may specialize only in short-term or outpatient therapy for low-risk patients. Others may specialize in the temporary or permanent care of residents who, as a result of a psychological disorder, require routine assistance, treatment, or a specialized and controlled environment. Patients are often admitted on a voluntary basis, but people whom psychiatrists believe may pose a significant danger to themselves or others may be subject to involuntary commitment.

Undoubtedly psychiatric Hospitals are benificial for mental health of the majority of patients but for some patients, these often stressful environment can be far from therapeutic ***(Drinkwater, 2011).***

In psychiatric practice, some mentally ill patients spend their life in continuous or prolonged hospitalization, that is, as long stay patients ***(Priebe, 2004).***

And the long-stay status of inpatients has different definitions across studies. Several reports have described stays between 60 and 90 days as long stays ***(Glick et al., 1975),*** whereas another study defined a long stay as lengh of stay (LOS)

of one year or longer the sample for the study was taken from the population of all patients admitted to the Berne psychiatric clinic over a 12-month period ***(Jakubaschk et al., 1993)***.

Psychiatrists from 59 UK mental health services returned data on 905 patients, aged 18-64 on admission, who had been in hospital for between six months and three years, used LOS criterion of greater than six months ***(Lelliott et al., 1994).***

Daly and Walsh (2009) defined of long-stay as those patients who have been in hospital continuously for between one and five years,

In Australia, new long-stay patients, who had been in hospital for over a year and less than 3 years ***(Richards et al., 1997).***

McCrone and Phelan found that psychiatric diagnosis predicted only 3% of the variation in length of psychiatric hospitalization ***(McCrone et al., 1994),*** so the duration of hospitalization was shown to depend not only on the clinical characteristics of the patient but also on his or her personality and socio-psychological characteristics both in the process of illness and in the premorbid state ***(AIa et al., 1984)***.

And as aconsequence of socio-environmental studies of psychosis emanating from the 1960s has been the belief that patients with psychosis benefit from a rapid return to the community, thus minimizing social isolation and the adverse

effects of lengthy stay in psychiatric hospitals including poor motivation, poor self-management ***(Wing et al., 1986)***. And a further defining characteristic among long-stay mental health clients is a high level of dependency. Becoming "institutionalized" is a common result of a lengthy stay, which forces inpatients to rely on both hospital staff and services and prevents them from assuming more responsibility for themselves and loss of basic daily living skills. Also these individuals typically lack social support networks of their own ***(Dale et al., 2009)***. Also they in high risk of developing physical illnesses by high rates of smoking, with central weight distribution and excessive weight gain ***(Cormac et al., 2004).***

Studies of patients returning to the community compared with those remaining in institutions show not only better quality of life and larger friendship networks, but also reductions in dependence on pharmacotherapy and lower mortality rates ***(Ryu et al., 2006)***.

Also hospitalization is among the most costly health care services and is projected as the most costly in 2014 ***(Truffer et al., 2010),*** so attention has focused on using less and less inpatient treatment, replacing hospital stay with treatment in the community. In fact, longer hospital stay may nowadays imply poor mental health care and support in the community. As a consequence, during the last two decades there has been an increased interest by administrators and governments responsible for financing mental health services in reducing the

money spent on inpatient services and consequently in length of stay reduction. Reduction of length of stay is associated with less expenditure as Longer admission, of course, means a greater financial burden incurred and reducing length of stay is considered to be a sign of successful treatment in the community ***(Douzenis et al., 2012).***

Now inpatient psychiatric care in the 21st century is defined by ultrashort lengths of stay. In the last two decades of the 20th century, length of stay for psychiatric inpatient care decreased from months to days. The sole focus of psychiatric inpatient treatment has become safety and crisis stabilization ***(Sharfstein et al., 2008).***

RATIONALE OF THE STUDY

The need for this study stems from the large number of mental inpatients that have been lengthly admitted in mental health hospitals in our country, trying in this study to understand main factors leading to the phenomena as a step to solve it.

AIM OF THE WORK

- To review of the literature about lengthy stay in mental health hospitals and factor which could affect this stay.
- To assess type of illness, disease severity and social factors among a sample of inpatients admitted for more than 1 year in Helwan mental Health hospital.
- To assess type of illness, disease severity and social factors among a sample of outpatients who had been admitted for less than 3 months in Helwan mental hospital then receiving their treatment at the outpatient clinic.
- To study the role of social factors that determines length of stay in mental hospital.

HYPOTHESIS

Our hypothesis is that lengthystay is more correlative to social factors rather than clinical or other disease variables.

Chapter 1
LENGTHY STAY IN MENTAL HOSPITALS AND SCHIZOPHRENIA

Schizophrenia:

Schizophrenia is a serious mental illness that interferes with a person s ability to think clearly, manage emotions, make decision and relate to others, researches has linked schizophrenia to changes in brain chemistry and structure ***(Kenduckworth, 2013).*** It is likely caused by distributed brain dysconnectivity ***(Stephan et al., 2006).*** Studies of its pathophysiology initially focused on characterizing striatal dopaminergic hyperactivity ***(Kegeles et al., 2010).*** This focus is now complemented by studies that have characterized altered glutamate neurotransmission in schizophrenia ***(Anticevic et al., 2012).*** An influential mechanistic hypothesis proposes a possible disruption in the balance of excitation and inhibition in the cortical microcircuitry resulting from hypofunction of the N-methyl-D-aspartate glutamate (NMDA) receptor ***(Krystal et al., 2003)***.

Like diabetes schizophrenia is a complex, long term medical illness that affect everybody differently the course of the illness is unique for each person ***(Kenduckworth, 2013)***.

It tends to be a chronic and relapsing disorder with generally incomplete remissions, variable degrees of functional impairment and social disability ***(Tandon et al., 2009)***.

It is a complex neuropsychiatric syndrome that profoundly affects perception, belief, and cognition ***(Lewis et al., 2006)***, we can divide symptoms of schizophrenia into positive symptoms including hallucinations (perception in absence of any stimulus) and delusions (fixed or falsely held beliefs), and negative symptoms (such as emotional apathy, lack of drive, poverty of speech, social withdrawal and self-neglect) ***(APA, 2013)***.

Typically there is a prodromal period, which precedes a first episode of psychosis and can last from a few days to around 18 months. The prodromal period is often characterised by some deterioration in personal functioning. Changes include the emergence of transient (of short duration) and/or attenuated (of lower intensity) psychotic symptoms, memory and concentration problems, unusual behaviour and ideas, disturbed communication and affect, and social withdrawal, apathy and reduced interest in daily activities. The prodromal period is usually followed by an acute episode marked by hallucinations, delusions and behavioural disturbances, usually accompanied by agitation and distress. Following resolution of the acute episode, usually after pharmacological, psychological and other interventions, symptoms diminish and often disappear for many people, although sometimes a number of negative symptoms

remain. This phase, which can last for many years, may be interrupted by recurrent acute episodes that may need additional pharmacological, psychological and other interventions, as in previous episodes. Others have no prodromal period, the disorder beginning suddenly with an acute episode ***(NICE, 2014).***

Schizophrenia remains a leading cause of disability worldwide ***(Murray et al., 1996)*** and is inadequately treated by available therapies ***(Krystal et al., 2003)***. This is especially true for disturbances in motivation and cognition that often accompany schizophrenia ***(Barch et al., 2012)***.

Epidemiology of schizophrenia:

Schizophrenia occurs throughout the world. The prevalence of schizophrenia (ie, the tend number of cases in a population at any one time point) approaches 1 percent internationally. The incidence (the number of new cases annually) is about 1.5 per 10,000 people ***(McGrath et al., 2008).***

Schizophrenia affects around 0.3–0.7% of people at some point in their life ***(Van et al., 2009)***. Or 24 million people worldwide as of 2011 (about one of every 285) ***(WHO, 2011)***.

Slightly more men are diagnosed with schizophrenia than women (on the order of 1.4:1) ***(Abel et al., 2010)***. And women to be diagnosed later in life than men.

Dopamine hypothesis of schizophrenia:

The dopamine hypothesis emerged from the discovery of antipsychotic drugs and the seminal work of Carlsson and Lindqvit who identified that these drugs increased the metabolism of dopamine when administered to animals.Further evidence came from observations that reserpine, which is effective for treating psychosis, was found to block the reuptake of dopamine and other monoamines, leading to their dissipation ***(Howes et al., 2009).*** Studies showing that amphetamine, which increases synaptic monoamine levels, can induce psychotic symptoms ***(Lieberman et al., 1987)*** provided additional evidence. It was not until the 1970s, however, that the dopamine hypothesis was finally crystallized with the finding that the clinical effectiveness of antipsychotic drugs was directly related to their affinity for dopamine receptors ***(Howes et al., 2009)***. The focus at the time was on excess transmission at dopamine receptors and blockade of these receptors to treat the psychosis ***(Seeman et al., 2000)***.

It was seen as a hypothesis of schizophrenia as a whole without a clear articulation of its relationship to any particular dimension (eg, positive vs negative symptoms) and no link was made to genetics and neurodevelopmental deficits and there was little clear indication of where the abnormality was in the living brain, and there was no framework for linking the dopaminergic abnormality to the expression of symptoms.

In 1991, Davis et al. published a landmark article describing what they called "a modified dopamine hypothesis of schizophrenia" that reconceptualized the dopamine hypothesis in the light of the findings available at the time. The main advance was the addition of regional specificity into the hypothesis to account for the available postmortem and metabolite findings, imaging data, and new insights from animal studies into cortical-subcortical interactions. It was clear by this stage that dopamine metabolites were not universally elevated in the cerebrospinal fluid (CSF) or serum of patients with schizophrenia.

Dopamine receptors show different brain distributions—characterized as D1 predominantly cortical and D2 predominantly subcortical—to provide a basis for suggesting that the effects of abnormalities in dopamine function could vary by brain region. However, it was PET studies showing reduced cerebral blood flow in frontal cortex that provided the best evidence of regional brain dysfunction in schizophrenia. "Hypofrontality" in these studies was directly correlated with low CSF dopamine metabolite levels. Because CSF dopamine metabolite levels reflect cortical dopamine metabolism, they argued that the relationship between hypofrontality and low CSF dopamine metabolite levels indicates low frontal dopamine levels. Thus, the major innovation in version II was the move from a one-sided dopamine hypothesis explaining all facets of schizophrenia to a regionally specific prefrontal

hypodopaminergia and a subcortical hyperdopaminergia. Lesions of dopamine neurons in the prefrontal cortex result in increased levels of dopamine and its metabolites and D2 receptor density in the striatum, ***(Pycock et al., 1980)*** while the application of dopamine agonists to prefrontal areas reduced dopamine metabolite levels in the striatum ***(Scatton et al., 1982).*** This provided a mechanism to propose that schizophrenia is characterized by frontal hypodopaminergia resulting in striatal hyperdopaminergia. Furthermore, ***Davis et al. (1991)*** hypothesized that negative symptoms of schizophrenia resulted from frontal hypodopaminergia, based on the similarities between the behavior exhibited by animals and humans with frontal lobe lesions and the negative symptoms of schizophrenia. Positive symptoms were hypothesized to result from striatal hyperdopaminergia, based on the findings that higher dopamine metabolite levels are related to greater positive symptoms and response to antipsychotic drug treatment.

Although there are a number of weaknesses in "version II" of the dopamine hypothesis, Much of the evidence for the hypothesis relied on inferences from animal studies or other clinical conditions. There was no direct evidence for low dopamine levels in the frontal cortex and limited direct evidence for elevated striatal dopaminergic function. It was unclear how the dopaminergic abnormalities were linked to the clinical phenomena—there was no framework describing how striatal

hyperdopaminergia translates into delusions or how frontal hypodopaminergia results into blunted affect, for example.

Then finally the hypothesis of the final common pathway. This hypothesis seeks to be comprehensive in providing a framework that links risk factors, including pregnancy and obstetric complications, stress and trauma, drug use, and genes, to increased presynaptic striatal dopaminergic function. It explains how a complex array of pathological, positron emission tomography, magnetic resonance imaging, and other findings, such as frontotemporal structural and functional abnormalities and cognitive impairments, may converge neurochemically to cause psychosis through aberrant salience and lead to a diagnosis of schizophrenia ***(Howes et al., 2009)***.

Etiology of schizophrenia:

1. Genetic factors:

Estimates of heritability vary because of the difficulty in separating the effects of genetics and the environment ***(O'Donovan et al., 2008)***, The greatest risk for developing schizophrenia is having a first-degree relative with the disease, more than 40% of monozygotic twins of those with schizophrenia are also affected ***(Picchioni et al., 2007)***. If one parent is affected the risk is about 13% and if both are affected the risk is nearly 50% ***(Herson, 2011).***

2. ***Environmental Factors***

Environmental factors associated with the development of schizophrenia include the living environment, drug use and prenatal stressors ***(Van et al., 2009)***. Childhood trauma, separation from ones families, and being bullied or abused increase the risk of psychosis ***(Sideli et al., 2012)***. Living in an urban environment during childhood or as an adult has consistently been found to increase the risk of schizophrenia ***(Van et al., 2009),*** Other factors that play an important role include social isolation and immigration related to social adversity, racial discrimination, family dysfunction, unemployment, and poor housing conditions ***(Picchioni et al., 2007)***. Parenting style seems to have no major effect, although people with supportive parents do better than those with critical or hostile parents ***(Picchioni et al., 2007)***.

Other environmental factors, such as pregnancy/obstetric complications, act in early life to increase the subsequent risk of schizophrenia ***(Cannon et al., 2002)***. There is now substantial evidence from animal models that pre- and perinatal factors can lead to long-term overactivity in mesostriatal dopaminergic function ***(Boksa et al., 2003; Boksa, 2004)***. For example, neonatal lesions affecting the hippocampus ***(Lipska, 2002)*** or frontal cortex ***(Flores et al., 1996)*** increase dopamine-mediated behavioral responses in rats, as does prenatal stress, whether induced by corticosterone administration ***(Diaz et al., 1995)*** or maternal handling ***(Henry et al., 1995).*** Neonatal