Original Article

# Frequency Of Depression In Patient Coming To Medical Outpatient Department

**Dr. SHALEE TAJ[1] , Dr. Azra Tabassum[2] , Dr. Hazrat Ali[3] , PROF. DR. GHULAM RASOOL[4] , Dr. Mir Usman[5]**

[1]MD PSYCHIATRY DEPARTMENT OF PSYCHIATRY DEPARTMENT, BALOCHISTAN INSTITUTE OF PSYCHIATRY AND BEHAVIORAL SCIENCES QUETTA
[2]DEPARTMENT OF PSYCHIATRY DEPARTMENT, BALOCHISTAN INSTITUTE OF PSYCHIATRY AND BEHAVIORAL SCIENCES QUETTA
[3]DEPARTMENT OF PSYCHIATRY DEPARTMENT, BALOCHISTAN INSTITUTE OF PSYCHIATRY AND BEHAVIORAL SCIENCES QUETTA
[4]MBBS, FCPS DEPARTMENT OF PSYCHIATRY DEPARTMENT, BALOCHISTAN INSTITUTE OF PSYCHIATRY AND BEHAVIORAL SCIENCES QUETTA
[5]DEPARTMENT OF PSYCHIATRY DEPARTMENT, BALOCHISTAN INSTITUTE OF PSYCHIATRY AND BEHAVIORAL SCIENCES QUETTA
**DOI:** 10.47750/pnr.2022.13.S08.472

## Abstract

Behavioral and mental diseases account for 12% of the worldwide disease burden. Adults are expected to account for 10% of the global population, with behavioral and mental disorders accounting for four of the ten top causes of disability. Depression is the most frequent mental illness, resulting in years lived with disabilities (YLDs). It manifests itself in a variety of ways, including a loss of interest, a depressed mood, sleep disturbances, a lack of energy, poor concentration, and a sense of guilt. It is estimated that 350 million people worldwide suffer from depression, which claims the lives of 850,000 individuals each year. According to the World Health Organization, depression will rank second among YLDs by 2020, and by 2030, it will be the major cause of disability. Depression causes significant handicap and can make it more difficult to recover from any medical condition. Furthermore, because most cases of depression are not identified, it goes undetected and untreated. According to a rough estimate, more than a third of the patients in Pakistan had undiagnosed comorbid depression. There is a scarcity of data on the prevalence of undiagnosed depression among Pakistani patients who attend the medical outpatient department (OPD). The purpose of the study was to determine the frequency of depression in medical outpatient department. The frequency of depression in patients visiting to medical outpatient department in our study was found to be 64% respectively that was assessed by diagnostic and statistical manual of mental disorders (DSM–5).Furthermore, relationship of patients with depression was assessed with multiple variables such as gender, marital status, chronic health illness, employment status and distribution of age. Although clinically significant; none of them were found to have significant relationship with depression with pvalue >0.05 respectively. Patients who visited the medical outpatient department had a higher incidence of depression. Primary health care research and people must acknowledge this as a significant issue and concentrate on detecting undiagnosed depression, which may be attributed to a lack of awareness among patients and providers. Therefore, development of an efficient mental health strategy and the implementation of mental health programs in the country's rural primary healthcare institutions are necessary to reduce the burden of this illness.

**KEYWORDS:** Depression, Severity, Pattern, Co-Morbidities, Outpatient department.

## Introduction:

In 2010, the Global Burden of Disease survey identified mental and drug use disorders as the largest cause of years lived with disability (YLDs). The most common cause of YLDs linked to mental problems is depression. Because suicide and self-harm are regarded separate categories, the full burden of depression is underestimated, and the role

of depression to mortality caused by metabolic disorders is underappreciated. The prevalence of depression throughout time varies by country (Whiteford, Ferrari, Degenhardt, Feigin, & Vos, 2015). Depression is a mental illness marked by low mood, loss of interest in activities, unexpected weight gain, sleep problems, psychomotor agitation or slowness, decreased energy, feelings of worthlessness and hopelessness, difficulties making decisions, and recurring thoughts of self-harm or suicide. In general medical practice, depression is just second to hypertension as the most common chronic disease. Half of patients with psychiatric illnesses who visit medical and surgical outpatient departments have depression and anxiety disorders, while the majority have somatoform disorder, which may lead to referral to medical outpatient departments (Seifu, Yigzaw, Haile, Reshid, & Asfaw, 2021). In our country, psychiatric morbidity, particularly depressive illness, is on the rise as a result of rising insecurity, terrorism, political instability, socioeconomic challenges, food shortages, global economic slowdown, rising unemployment, and other issues. In 5 studies from industrialized countries, higher rates are recorded, whereas poorer and middle income countries report lower rates. A population-based study in Colombo found a lifetime prevalence of depression of 6.6 percent. Females, divorced people, and people from lower socioeconomic backgrounds have higher rates of depression (Somasundaram, 2007). A common comorbidity of physical sickness is depression. According to the World Health Survey, the prevalence of International Classification of Diseases (ICD)-10 depressive episodes was 4.5 percent in angina, 4.1 percent in arthritis, 3.3 percent in asthma, and 2% in diabetes over the course of a year. In the presence of a chronic illness, the chance of depression is much higher than in the absence of a chronic medical ailment. Patients with Parkinson's disease (37.5%), chronic renal failure (27.9%), and elderly patients in a Sri Lankan medical ward have all been found to have a high prevalence of depression (Han et al., 2019). Because of the high morbidity and mortality associated with depression, early detection and treatment are critical. In this case, the primary health care provider is crucial. GPs, on the other hand, have been proven to under-diagnose depression and treat only a small percentage of patients who are diagnosed with it (Katon, 2011). Only 47.3 percent of patients were accurately detected in a meta-analysis of 41 studies assessing the accuracy of GPs' unsupported diagnosis of depression. The manifestation of depression symptoms as somatic complaints is a regular occurrence. This wide range of symptoms, as well as the stigma associated with mental illness, may lead to a lack 6 of detection of the condition in basic care. Increased sensitivity in the detection and diagnosis of depression will result from a greater understanding of the prevalence and features of depressive symptoms (Mitchell, Rao, & Vaze, 2011). In Sri Lanka, there is a scarcity of data on the prevalence of depression in primary care settings and outpatient departments (OPD). The aim of conducting this study is to determine the frequency of depression in medical outpatient department. Depression is although common however increasing in the severity may cause lethal outcomes. In our society, it puts a huge burden on our population. Patients who use to visit in medial outpatient department have suffered from this illness due to chronic health care issues. Therefore, the aim of this study is to determine the magnitude of problem in our society and its trend. With the help of results of this study, we would be able to determine the exact burden of this illness as well as the factors contributing to cause this illness in term of social, financial or relationship issues. An integrated approach of early assessment with our PHQ-9 scale scores and proper management in terms of treatment and extensive counseling, we believe that we would be able to reduce the tendency of depression and its complications and improve the outcome of patient by timely recognition and prompt treatment in our society.

## Literature Review

Depression is a common ailment that goes undetected and untreated far too often. Despite this, depression is more likely to be diagnosed today than in previous decades, because the social stigma associated with the condition has faded, 9 available treatments are effective and well tolerated, and primary care physicians, to whom most patients first present and from whom most patients receive initial therapy, have become more willing to diagnose and treat this condition (Dennis & Chung-Lee, 2006). Dysthymic disorder, major depressive disorder (MDD), seasonal affective disorder, episodic depression, or an indication of an associated mood, bipolar, or psychotic condition may all be signs of depression. Depression can also be episodic, as a result of a loss or a significant life change. Furthermore, it may be important in some therapeutic situations, such as during pregnancy or the postpartum period, adolescence, or at the end of life. This sort of depression is not covered in this article (Uher, Payne, Pavlova, & Perlis, 2014). SYMPTOMS

Loss of interest in formerly pleasurable activities, sadness, anger, feelings of worthlessness, hopelessness, guilt or anxiety, fears about death, or suicide thinking are all common signs of depression. Changes in appetite, weight loss or gain, sleep difficulties, psychomotor activity, decreased energy, indecisiveness, or diverted attention are all possible signs. Patients with depression are more likely to have somatic problems and use primary care, urgent care, emergency, and 10 inpatient services often. Patients with depression may also be labeled as "difficult" to treat by professional staff (Burns & Beck, 1978). Patients who are exposed to psychosocial stressors but may not satisfy the criteria for a major depressive episode may have an adjustment disorder, often known as posttraumatic stress disorder. Profound depressive disorder is a condition marked by recurrent episodes of major depression and is associated with a high death rate. Patients with severe MDD have a high suicide rate, and epidemiologic studies show that patients with MDD over the age of 55 have a 4-fold increased risk of death. Bipolar disorder is defined as a combination of depressive and manic episodes (Casey, 2001). HISTORY AND PHYSICAL EXAMINATION Because depression is recurrent, it's critical to note the severity and length of symptoms, as well as any past bouts, when completing a medical history. Both major depression and bipolar disorder are heritable illnesses, so it's important to find out whether there's a family history of depression or other psychiatric disorders. Patients who show signs of manic or psychotic behavior may need to be referred to a psychiatrist sooner. A history of suicidal thoughts, plans, or actions must be established, and this may identify patients who require more immediate assistance (Neff & Marzani, 2012). 11 A thorough physical examination is important to identify contributing or causal illnesses, such as chronic liver or renal disease, endocrine disease (hypothyroidism or adrenal insufficiency), congestive heart failure, dementia, or other conditions, even though the physical examination is relatively insensitive for detecting depression (Baldessarini, Vieta, Calabrese, Tohen, & Bowden, 2010). The point prevalence of serious depression among people in primary care settings ranges from 5% to 9%, with up to 50% of depressed patients going undiagnosed. In primary care settings, the associated conditions of dysthymia (chronic lowgrade depression) and minor depression are as common as major depression. Depressive disorders afflict 0.8 percent to 2.0 percent of children and 4.5 percent of adolescents, according to estimates (Schramm, Klein, Elsaesser, Furukawa, & Domschke, 2020). Inadequate knowledge of diagnostic criteria, competing priorities and concomitant conditions, time constraints, stigma associated with "labeling" a patient as depressed and poor reimbursement processes are some of the factors that may contribute to these percentages (McCarter, 2008). A number of case-finding instruments have been developed and validated as a result of these barriers, including the Beck Depression Inventory, the Center for Epidemiological Studies-Depression Scale, the Zung Self-Assessment Depression 12 Scale, the General Health Questionnaire, and the Hopkins Symptom Checklist, among others (Williams Jr, Pignone, Ramirez, & Stellato, 2002).

## CLASSIFICATION, PREVALENCE AND COURSE OF DEPRESSION

According to the Diagnostic and Statistical Manual (DSM) of Mental Health, Fourth Edition, the essence of major depressive disorder is a clinical course marked by one or more major depressive episodes without a history of manic, mixed, or hypomanic episodes (DSM-IV). Five of the following nine DSM-IV symptoms must be present for a minimum of two weeks for an accurate diagnosis: 13 (i) a depressed mood; (ii) a loss of interest or pleasure; (iii) a significant change in weight or appetite; (iv) insomnia or hyposomnia; (v) psychomotor agitation or retardation; (vi) fatigue or loss of energy; (vii) feelings of worthlessness; (viii) diminished ability to think or concentrate or indecisiveness; and (ix) suicidal ideation (Akiskal, 1996). The origins and classification of depression has been the subject of much debate in the past. At the turn of the century, the two opposing points of view were Emil Kraepelin's conceptualization of depression as a sickness and Sigmund Freud's understanding of depression as an expression of repressed rage and loss. Sir Martin Roth and the Newcastle Group made a significant contribution to our understanding of depression by categorizing clinical symptoms of depression (ranging from mild to severe psychosis) and dividing them into discrete groupings of "endogenous" and "reactive" subtypes of depression (Whybrow, Akiskal, & McKinney Jr, 2012). This notion has been employed in biological psychiatric research for decades to discover different etiological subtypes of the condition. DSM-IV and the International Statistical Classification of Diseases, 10th Revision (ICD-10) are new versions that distinguish unipolar (depression) from bipolar (manic depressive) illness, based on findings from collaborative efforts in the United States and the United Kingdom (Moller, 2003). 14 In the general population,

the lifetime prevalence of depression is as high as 20%, with a female to male ratio of roughly 5:2. We must suppose that only approximately a third of patients are receiving therapy, perhaps not owing to ignorance, but rather because symptoms may be qualitatively similar to those encountered in ordinary life. The disease is usually recurrent, and the majority of individuals recover from major depressive episodes. However, a significant proportion of patients develop chronic depression, and after 5 and 10 years of prospective follow-up, 12 percent and 7% of patients are still depressed, respectively (Steers, Wickham, & Acitelli, 2014). There is a significant rate of recurrence in people who successfully recover, with around 75% of patients experiencing more than one episode of serious depression within ten years. Suicide is a significant cause of death in those who are depressed, and the suicide rate is particularly high between the ages of 15 and 24. Multiple lines of evidence point to a strong link between depression and cardiovascular disease, as well as higher mortality rates. Depression has been shown in certain studies to raise the chance of developing cardiac disease, particularly coronary artery disease, and to impair the prognosis after a heart attack. Depression appeared to raise the risk of cardiac mortality regardless of baseline cardiac state; additionally, the extra mortality risk for serious depression 15 was found to be significantly higher (Borentain, Nash, Dayal, & DiBernardo, 2020). The high rate of comorbidity with other psychiatric disorders is another essential feature of depression. Anxiety, particularly panic disorder, is frequently linked to affective disorders, but the link between alcohol and drug usage is weaker. Surprisingly, the development of anxiety usually precedes that of depression, but the onset of depression is equally likely to occur before or after the onset of alcohol abuse (Merikangas et al., 1996). RISK **FACTORS FOR**

## DEPRESSION THE IMPACT OF LIFE EVENTS

Numerous studies have been undertaken on the impact of chronic stress and traumatic life experiences on the development of depression, with research on the somatic and endocrine consequences of stress in animals serving as a foundation for the work. Despite widespread criticism of the methodology (for example, the choice of instruments to obtain life event information, the exclusion of events that are the result of physical illness, or the quantification of stress), the majority of findings show an excess of severely threatening events prior to onset, particularly for exit events or undesirable events (Picard & McEwen, 2018). 16 The existence of events triggering the beginning of endogenous or nonendogenous depression is variable and likely unrelated to the symptom pattern, implying that there is no clear-cut distinction between the presence of events provoking the onset of endogenous or non-endogenous depression. The role of events on depressed outcome is still being debated, since pleasant events have been proven to improve outcome while stressful situations have been shown to decrease progress and raise the risk of relapse. The fact that females are more prone than males to suffer from serious depression cannot be explained by differences in rates or sensitivity to stressful life events (Strauss et al., 2018). Although women reported more interpersonal and men reported more legal or work-related stressful life events, this cannot be linked to females' higher rates of severe depression (Kendler, Thornton, & Prescott, 2001).

## GENETIC INFLUENCES

Genetic variables have a crucial role in the etiology of emotional disorders, according to evidence from family, twin, and adoption studies. There is considerable epidemiological evidence for a genetic contribution, particularly in bipolar disorders, with heritability estimates as high as 80%. However, the inheritance does not follow the conventional mendelian pattern, implying that a single major gene locus may not - or only in a few families be responsible for the 17 increased intra-familial risk of the condition. A complex disorder hypothesis, in which numerous genes with little effects interact with each other or with a range of environmental circumstances to raise familial predisposition to the disorder, is more likely (Faraone, Glatt, & Tsuang, 2003).

## BIOCHEMICAL BASIS OF DEPRESSION

In the twentieth century, great development in the field of neuroscience provided us with intriguing insights into the nature of mental processes. Beginning with neuroanatomy and electrophysiology at the turn of the twentieth century, neuroscience has evolved into an interdisciplinary field that encompasses a wide range of biological investigations,

from molecular studies of cell and gene function to brain-imaging techniques, expanding our understanding of the cellular and molecular machinery that controls behavior. For a long time, especially in the field of psychiatry, little was known about the biological substrates of the disorders, and the work of Julius Axelrod, Arvid Carlsson, and other Nobel Laureates has significantly contributed to our understanding of brain function, and investigations of psychiatric disorders are now fully based in neuroscience (Brigitta, 2002).

## SYNAPTIC TRANSMISSION

The pioneering study of Otto Loewi and other scientists, namely, that chemical transmission is the primary way by which nerves interacts with one another, was 20 one of the most significant discoveries in neuroscience. Today, it is widely accepted that pre- and postsynaptic events are tightly regulated and serve as the foundation for central nervous system (CNS) plasticity and learning. Chemical transmission entails several steps, including neurotransmitter synthesis, storage in secretory vesicles, and regulated release into the synaptic cleft between pre- and postsynaptic neurons, as well as the termination of neurotransmitter action and induction of final cellular responses via various steps in the signal transduction cascade (Colangelo, Shichkova, Keller, Markram, & Ramaswamy, 2019). Figure-1 shows a synapse for traditional neurotransmitters in schematic form. The first step in the synthesis process is the facilitated transport of amino acids from the bloodstream to the brain, where precursors are converted into transmitters via enzymatic reactions, which are then stored in synaptic vesicles before being released into the synaptic cleft via a Ca2+ dependent process. The rate of neurotransmitter release is determined by the firing rate of the neurons, therefore situations or medicines that alter the firing rate affect the transmitter's release. Somatodendritic auto receptors are another significant regulator of release, because binding of released transmitter molecules reduces synthesis or prevents future release from the presynapse. The synaptic effects are stopped when transmitters attach to certain transporter proteins and are reabsorbed 21 into the presynapse, where they are degraded by enzymes such monoamine oxidase (MAO) or stored in vesicles anew.

## MONOAMINE HYPOTHESIS

The first major hypothesis of depression was proposed about 30 years ago, claiming that depression is caused by a functional deficiency of the brain monoaminergic transmitters norepinephrine (NE), 5-HT, and/or dopamine (DA), whereas mania is caused by a functional excess of monoamines at critical synapses in the brain (Hamon & Blier, 2013). Clinical findings and animal experiments supported this idea, demonstrating that the antihypertensive medication reserpine, which depletes presynaptic reserves of NE, 5-HT, and DA, creates a depression-like condition. In contrast to the effects of reserpine, some patients receiving iproniazid, a tuberculosis drug that boosted brain concentrations 23 of NE and 5-HT by blocking the metabolic enzyme MAO, experienced euphoria and hyperactive behavior (Chopra, Kumar, & Kuhad, 2011). It is clear that monoaminergic systems are responsible for many behavioral symptoms, such as mood, vigilance, motivation, fatigue, and psychomotor agitation or retardation, given the origins of noradrenergic, serotonergic, and dopaminergic neurons in the brain and their projections into many areas of the brain. Alterations in neurotransmitter synthesis, storage, or release, as well as disrupted sensitivity of their receptors or subcellular messenger activities, can cause abnormal function and behavioral implications in either depressive or manic states (Šimić et al., 2017).

## ENDOCRINE PROCESSES IN DEPRESSION

Hormonal abnormalities such as changes in Cortisol, growth hormone (GH), or thyroid hormone levels suggest the presence of endocrine disturbances, particularly dysfunctions in the hypothalamuspituitary-adrenal (HPA) axis and/or thyroid function control. The persistent finding that a significant subset of depressed people secretes excessive amounts of Cortisol when depressed but not after recovery prompted extensive research and analysis of the HPA system. In response to exogenous glucocorticoid injection, hypersecretion of hypothalamic corticotropin-releasing hormone (CRH) and insufficient glucocorticoid feedback, elevated Cortisol levels, and poor regulation of the HPA

axis were seen. Improved corticosteroid receptor signaling has recently been proposed as a critical factor in the etiology of depression, according to more refined research (Tsigos, Kyrou, Kassi, & Chrousos, 2020).

## NEUROTROPHINS AND DEPRESSION

Adaptation or plasticity of neural systems is one theory for the pathogenesis and therapy of depression. Antidepressants may act by repairing this dysfunction or by directly causing the proper adaptive responses to stress or other aversive stimuli, while depression may be caused by an inability to create the appropriate adaptive responses to stress or other aversive stimuli (Kupferberg, Bicks, & Hasler, 2016). 37 Neurotrophic factors are among the growth factors whose role in the adult nervous system has been investigated. Brain derived neurotrophic factor (BDNF) and neurotrophin-3 (NT-3) are two endogenous proteins that have been proven to increase the function and proliferation of 5-HT-containing neurons in the adult brain. Chronic infusions of these drugs, but not acute infusions, had a significant impact on serotonergic neuron development and regeneration, as well as triggered 5-HT nerve terminal sprouting (Binder & Scharfman, 2004). BDNF reduced learned helplessness in animals, which is a common side effect of antidepressant therapy. Treatment with antidepressants, such as particular inhibitors of 5-HT or NE absorption as well as MAOIs, boosts BDNF mRNA levels in the rat hippocampus via the 5-HT2A and adrenoceptor subtypes, and protects stressinduced declines in BDNF messenger RNA (mRNA), according to further research. This impact was noticeable after three weeks of treatment, but not after a single dose, evoking the treatment response delay (Hayley, Poulter, Merali, & Anisman, 2005). A recent postmortem finding of enhanced BDNF expression in antidepressant-treated patients corroborated the findings of these animal tests. Exogenous neurotrophins, which are relatively big lipophobic proteins that do not pass the blood-brain barrier, cannot increase BDNF levels, therefore understanding the mechanism by which these medications enhance BDNF mRNA could be particularly relevant. Small compounds that cross through the blood- 38 brain barrier and raise endogenous neurotrophin levels, on the other hand, could represent a new class of antidepressants (Maes et al., 2009).

In Bielefeld, Germany, a city of about 340.000 inhabitants, an integrated care model contract was established in 2007 (and is still in effect), in which our psychiatric clinic's outpatient service, regional unions of physicians and psychotherapists working in private practises ("Medi-OWL" and "APP"), and a work group of health insurances covering about 30% of the region's population ("ARGE BKK OWL") all participate. The Bielefeld Intensive Outpatient Depression Program (BID)", is a newly designed integrated care model that focuses on the treatment of individuals with depressive disorders (Roth & Fonagy, 2006). BID provides an outpatient, intense and multimodal treatment programme that lasts six weeks and is detailed below. The clinic, on the other hand, provides a regular 7-day per week inpatient complicated treatment programme for patients 47 with depression that is not limited to a specific number of weeks but often lasts about 6 weeks (Mulrow et al., 2000). We anticipated that both programmes are clinically beneficial in pre-post analyses after 6 weeks of therapy because prior studies have indicated that outpatient treatments are effective in (chronic) depressive illness. However, we assumed that inpatient treatment is more effective than outpatient treatment because it provides significantly more specific and non-specific therapies over the course of seven days per week, as well as permanent availability of specialist personnel and immediate assistance in the event of a crisis (Liebherz & Rabung, 2013).

## Research Methodology:

OPERATIONAL DEFINTIONS: - 1. Depression:-

It was defined as a state of mood disorder that can affect a person's daily life. It may be described as feelings of sadness, loss and/or anger. It will be assessed with the help of diagnostic and statistical manual of mental disorders (DSM–5), symptoms of depression. At least 5 symptoms must be present in an individual in order to confirm depression 2. Medical outpatient department: - It was defined as a routine visit of patients to medical outpatient department for their health problems in the form of diagnosis and treatment like hypertension, diabetes, arthritis and/or any autoimmune etc.

MATERIAL AND METHOD: Study design: Cross sectional study (Descriptive study). Setting: Balochistan institute of psychiatry and behavioral sciences Quetta. Duration of study: The study took six months from 1st July 2021 to 31st December 2021. Sample size: For the objective of the study, sample size was calculated by using WHO sample size calculator with Confidence level (1-α) is taken as 95%, with desired precision (d) of 8 % and approximate population estimation (frequency of depression in medical outpatient department) of 54.9 % taken from the parent study (Ofori & Adiukwu, 2018). By putting all the values, the largest sample size calculated was 148. n= Z1-α/2 2 * P (1-P)/d2 where; p= estimated prevalence= 0.549 q= 1-p= 0.451 d= margin of error 0.08 α= probability of type 1 error 0.05 (2-sided) = 1.96

Sampling technique: non-probability consecutive sampling.

**SAMPLE SELECTION:** - Inclusion Criteria: - 1. All individuals age 21 years- 65 years who were not admitted however use to visit in medical outpatient department for their chronic health problems. 2. Either gender. Exclusion Criteria: - 1. Patient visit in outpatient department for their chronic health illnesses however had been suffered complications related to either condition like stroke, congestive heart failure, kidney failure, or limb amputation. 2. Individual with remarkable history of any substance abuse problems, known neuropsychiatric disorders, or previous treatment by a clinical psychologist or psychiatrist. All above mentioned cases were effect modifier, and if would have included in the sample, would introduce bias in the study results.

**DATA COLLECTION TECHNIQUE:** -The study was conducted after the approval from Dean/ Director of Postgraduate Medical Institute, Balochistan institute of psychiatry and behavioral sciences Quetta. All individuals who were visiting medical outpatient department for their health problems were included. Written consent taken from patient before conducting the study. Demographic details were collected on a demographic proforma, including age, gender, education, occupation, area of residence, monthly income, duration of illness, treatment options and duration of treatment. Comorbid illnesses were noted like hypertension, hyperlipidaemias, ischemic heart disease and smoking. The 9-items Patient Health Questionnaire (PHQ-9) was used to assess the presence and severity of depressive symptoms (see annexure-C). PHQ-9 had been shown to be both a reliable and valid assessment tool. Patients were rating the frequency of experiencing the listed depressive symptoms on a scale of 0-3. A total score of 0 to 27 correlating with the severity of depression was calculated. Depression severity was grouped into none (score 0-4), minimal symptoms (score 5-9), and ≥ 10 as major depression. This was subcategorized as mild depression (score 10-14), moderately severe depression (score 15-19), and severe depression (score of greater than 20). Later, results were analyzed by senior resident who was a part of team of conducting this study which was checked by consultant before putting the data onto proforma. Afterwards, data analysis procedure was started as soon as required sample data was achieved.

**DATA ANALYSIS PROCEDURE:** - Data will be compiled and analyzed using statistical package for social sciences (SPSS) version 26.0. Mean ± Standard deviation (SD) will be calculated for quantitative variables like age, duration of illness and duration of treatment. Frequencies with percentages will be presented for qualitative variables and categorical variables like stratification of age, marital status (single/married), chronic health illness (hypertension/diabetes/ischemic heart disease/none), employment status (employed/unemployed/retired), depression (yes/no) and depression severity [PHQ-9 scale scores] (none/mild/moderate/moderately severe/ severe) (yes/no). Effect modifier will be controlled through stratification of age, marital status and chronic health illness to see the effect of these on outcome variables by applying P value of 0.05 was considered as statistically significant.

## Results:

There were 148 subjects enrolled in the study. Of them, 119 were males and 29 were females that constitute about 80% and 20 % respectively. The mean age of subjects was 45.24 ± 10.21 years (Figure-3). While the mean duration of illness was found to be 16.94 ± 8.80 months and the mean duration of treatment was found to be 12.33 ± 7.68 months respectively. Individuals were assessed on the basis of their marital status. Out of 148 subjects, 68 were single

while 80 were married constituting about 46% and 54% respectively. In our study, the frequency of depression in patients visiting to medical outpatient department was found to be 64% respectively. That means out of total 148 patients visiting to outpatient department, 94 were found to have depression. Depression was assessed by diagnostic and statistical manual of mental disorders (DSM–5) . Moreover, Out of 94 patients who were found to have depression; 14 had mild degree, 27 had moderate degree, 33 had moderately severe degree and 20 were found to have severe degree of depression that comprises about 15%, 29%, 35% and 21% respectively . We have also evaluated subjects with their history of chronic illness present. Out of 148 subjects, 46 had hypertension, 45 had diabetes, 37 had ischemic heart disease and remaining 20 were found to have others chronic illness with mixed 56 type of comorbidities. That constitutes about 31%, 30%, 25% and 14% respectively. Subjects were also assessed on the basis of their employment status. Out of 148 individuals, 34 were employed, 45 were found retired and 69 were found unemployed that comprises of 23%, 30%, and 47% respectively. Moreover, individuals were also analyzed with respect to distribution in age groups. Out of 148 subjects, 31 were found in age groups 21-35 years old, 73 in 36-50 years old and 44 in 51-65 years old respectively that constitute about 21%, 49% and 30% respectively. Furthermore, relationship of patients with depression was assessed with multiple variables. With respect to gender, no significant relationship was found statistically between gender and depression. Although, depression was mostly found in males compared to females, however; there was less number of female patients enrolled in our study. Similarly, there was no significant relationship was found statistically between depression and marital status of patients. Therefore it was demonstrated that whether the patient was single or married, it was not associated with depression . Likewise, relationship of patient with depression was also evaluated with respect to chronic health illness, employment status and distribution of age. However, none of them were found to have significantly associated statistically, with p-value >0.05 respectively.

## Discussion:

The frequency of depression in patients visiting to medical outpatient department in our study was found to be 64%. Similar results were seen in study conducted by El-Rufaie's study who reported a prevalence rate of 55% (El-Rufaie & Absood, 1993). On the other hand, some studies showed less percentage like study conducted by Molla et al who reported the frequency of depression about 17.5% respectively (Molla et al., 2016). Moreover, in our study, the severity of depression was mostly moderate in intensity. The fact that we conducted a crosssectional study while the lower report was a review of studies, the measurement tool we used was also not the same, and we only included adult outpatients while the review study included study participants from various age groups could be an explanation for the higher prevalence of depressive episodes in our study. Depression was more prevalent in the age group of 20-49 years in study conducted by Mahdi et al (Abumadini, 2003) while in our study it was found that the most common age group that was affected by depression was patients in 36-50 years old respectively. Males were mostly affected by depression in our study. Our findings were consistent with the study conducted by Alemu et al who also reported that the males compared to females were most likely to had depression (Lemma, Mulat, Nigussie, & Getinet, 2021). Contrary to this, study conducted by Mahdi et al showed that female was highly prone to develop depression than males (Abumadini, 2003). 74 In our study, marital study was also assessed of all subjects with depression. There was high proportion of married people who developed depression than those who were single, although the relationship was statistically insignificant between the two. Almost similar results were found in study conducted by Kowser et al respectively (Kowser, Zohra, Parveen, Islam, & Uddin, 2021). The current investigation found no evidence that depressive illnesses are more common among divorced people (Paykel, 1992). Marriage does not appear to provide much protection against developing depression. Comorbidities were although clinically significant however statistically insignificantly associated with depression in our study. Unemployed people are more likely to suffer from depression; however this was not clearly linked to socioeconomic factors. In our study, 69 patients were formally unemployed; 34 were employed, 45 were found retired respectively.

## Conclusion:

Patients who visited the medical outpatient department had a higher incidence of depression. As a result, patients who present to such OPDs should be screened for depression. Primary health care research and people must acknowledge

this as a significant issue and concentrate on detecting undiagnosed depression, which may be attributed to a lack of awareness among patients and providers. Male gender, married status, and unemployment are all predictors of depression in OPD patients. Furthermore, depression is a common affliction that continues to be costly in terms of both human and financial resources. More emphasis should be paid to its diagnosis and care, and collaboration and possible integration of various medical and social services could help a lot.

## References:


- Abhilash, M., Alex, M., Mathews, V. V., & Nair, R. H. (2014). Chronic effect of aspartame on ionic homeostasis and monoamine neurotransmitters in the rat brain. International journal of toxicology, 33(4), 332-341.
- Abou-haila, A., & Tulsiani, D. R. (2009). Signal transduction pathways that regulate sperm capacitation and the acrosome reaction. Archives of biochemistry and biophysics, 485(1), 72-81.
- Abumadini, M. S. (2003). Depressive disorders in psychiatric outpatient clinic attendees in eastern Saudi Arabia. Journal of family & community medicine, 10(2), 43.
- Akiskal, H. S. (1996). The prevalent clinical spectrum of bipolar disorders: beyond DSM-IV. Journal of clinical psychopharmacology, 16(2), 4S-14S.
- Baldessarini, R. J., Vieta, E., Calabrese, J. R., Tohen, M., & Bowden, C. L. (2010). Bipolar depression: overview and commentary. Harvard review of psychiatry, 18(3), 143-157.
- Binder, D. K., & Scharfman, H. E. (2004). Brain-derived neurotrophic factor. Growth factors (Chur, Switzerland), 22(3), 123.
- Blier, P. (2001). Crosstalk between the norepinephrine and serotonin systems and its role in the antidepressant response. Journal of psychiatry & neuroscience: JPN, 26(Suppl), S3. 78
- Blume, J., Douglas, S. D., & Evans, D. L. (2011). Immune suppression and immune activation in depression. Brain, behavior, and immunity, 25(2), 221-229.
- • Bondy, B. (2005). Pharmacogenomics in depression and antidepressants. Dialogues in clinical neuroscience, 7(3), 223.
- Booij, L., Tremblay, R. E., Szyf, M., & Benkelfat, C. (2015). Genetic and early environmental influences on the serotonin system: consequences for brain development and risk for psychopathology. Journal of psychiatry & neuroscience: JPN, 40(1), 5.
- Borentain, S., Nash, A. I., Dayal, R., & DiBernardo, A. (2020). Patientreported outcomes in major depressive disorder with suicidal ideation: a real-world data analysis using PatientsLikeMe platform. BMC psychiatry, 20(1), 1-11.
- Brigitta, B. (2002). Pathophysiology of depression and mechanisms of treatment. Dialogues in clinical neuroscience, 4(1), 7.
- Brust, P., Hesse, S., Muller, U., & Szabo, Z. (2006). Neuroimaging of the serotonin transporter: possibilities and pitfalls. Current psychiatry reviews, 2(1), 111-149.
- Burns, D. D., & Beck, A. T. (1978). Cognitive behavior modification of mood disorders. In Cognitive behavior therapy (pp. 109-134): Springer.
- Cairney, J., & Krause, N. (2005). The social distribution of psychological distress and depression in older adults. Journal of aging and health, 17(6), 807-835.
- Camacho, M. B., Vijitbenjaronk, W. D., & Anastasio, T. J. (2019). Computational Analysis of Therapeutic Neuroadaptation to Chronic Antidepressant in a Model of the Monoaminergic Neurotransmitter and Stress Hormone Systems. Frontiers in pharmacology, 10, 1215.
- Casey, P. (2001). Adult adjustment disorder: a review of its current diagnostic status. Journal of Psychiatric Practice®, 7(1), 32-40.
- Chopra, K., Kumar, B., & Kuhad, A. (2011). Pathobiological targets of depression. Expert opinion on therapeutic targets, 15(4), 379-400. 79
- Coccaro, E. F. (1989). Central serotonin and impulsive aggression. The British Journal of Psychiatry, 155(S8), 52-62.
- Cohen, S., Kessler, R. C., & Gordon, L. U. (1995). Strategies for measuring stress in studies of psychiatric and physical disorders. Measuring stress: A guide for health and social scientists, 3-26.
- Colangelo, C., Shichkova, P., Keller, D., Markram, H., & Ramaswamy, S. (2019). Cellular, synaptic and network effects of acetylcholine in the neocortex. Frontiers in neural circuits, 13, 24.
- Contreras, F., Menchon, J. M., Urretavizcaya, M., Navarro, M. A., Vallejo, J., & Parker, G. (2007). Hormonal differences between psychotic and non-psychotic melancholic depression. Journal of affective disorders, 100(1-3), 65-73.
- Dadds, M. R., Spence, S. H., Holland, D. E., Barrett, P. M., & Laurens, K. R. (1997). Prevention and early intervention for anxiety disorders: a controlled trial. Journal of consulting and clinical psychology, 65(4), 627.
- Debonnel, G., Saint-André, É., Hébert, C., De Montigny, C., Lavoie, N., & Blier, P. (2007). Differential physiological effects of a low dose and high doses of venlafaxine in major depression. International Journal of Neuropsychopharmacology, 10(1), 51-61.
- Delgado, P. L. (2000). Depression: the case for a monoamine deficiency. Journal of Clinical Psychiatry, 61(6), 7-11.
- El-Rufaie, O. E., & Absood, G. H. (1993). Minor psychiatric morbidity in primary health care: prevalence, nature and severity. International journal of social psychiatry, 39(3), 159-
- Farooq, R. K., Asghar, K., Kanwal, S., & Zulqernain, A. (2017). Role of inflammatory cytokines in depression: focus on interleukin-1β. Biomedical reports, 6(1), 15-20.
- Feighner, J. P. (1999). Mechanism of action of antidepressant medications. Journal of Clinical Psychiatry, 60(4), 4-13.

- Goldstein, J. M., Holsen, L., Cherkerzian, S., Misra, M., & Handra, R. (2017). NEUROENDOCRINE MECHANISMS OF DEPRESSION. Charney & Nestler's Neurobiology of Mental Illness, 365.
- Gorman, J. M., & Sullivan, G. (2000). Noradrenergic approaches to antidepressant therapy. Journal of Clinical Psychiatry, 61, 13-16.
- Gunn, J., Diggens, J., Hegarty, K., & Blashki, G. (2006). A systematic review of complex system interventions designed to increase recovery from depression in primary care. BMC health services research, 6(1), 1- 11. 81
- Hamon, M., & Blier, P. (2013). Monoamine neurocircuitry in depression and strategies for new treatments. Progress in NeuroPsychopharmacology and Biological Psychiatry, 45, 54-63.
- Han, K.-M., Kim, M. S., Kim, A., Paik, J.-W., Lee, J., & Ham, B.-J. (2019). Chronic medical conditions and metabolic syndrome as risk factors for incidence of major depressive disorder: A longitudinal study based on 4.7 million adults in South Korea. Journal of affective disorders, 257, 486-494.
- Harro, J., & Oreland, L. (2001). Depression as a spreading adjustment disorder of monoaminergic neurons: a case for primary implication of the locus coeruleus. Brain Research Reviews, 38(1-2), 79-128. •
- Hayley, S., Poulter, M., Merali, Z., & Anisman, H. (2005). The pathogenesis of clinical depression: stressor-and cytokine-induced alterations of neuroplasticity. Neuroscience, 135(3), 659-678.
- Mulrow, C. D., Williams Jr, J. W., Chiquette, E., Aguilar, C., HitchcockNoel, P., Lee, S., . . . Stamm, K. (2000). Efficacy of newer medications for treating depression in primary care patients. The American journal of medicine, 108(1), 54-64.
- • Neff, A. P., & Marzani, G. (2012). Bipolar disorders: a review. American family physician, 85(5), 483-493. •
- Nemeroff, C. B. (2020). The state of our understanding of the pathophysiology and optimal treatment of depression: Glass half full or half empty? American Journal of Psychiatry, 177(8), 671-685. •
- Newton-Cheh, C., & Hirschhorn, J. N. (2005). Genetic association studies of complex traits: design and analysis issues. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis, 573(1-2), 54-69. •
- Ofori, S. N., & Adiukwu, F. N. (2018). Screening for depressive symptoms among patients attending specialist medical outpatient clinics in a tertiary hospital in southern Nigeria. Psychiatry journal, 2018. • Owens, M. J., & Nemeroff, C. B. (1994). Role of serotonin in the pathophysiology of depression: focus on the serotonin transporter. Clinical chemistry, 40(2), 288-295. •
- Palazidou, E. (2012). The neurobiology of depression. British medical bulletin, 101(1), 127-145.
- Pévet, P. (2003). Melatonin in animal models. Dialogues in clinical neuroscience, 5(4), 343.
- Picard, M., & McEwen, B. S. (2018). Psychological stress and mitochondria: a systematic review. Psychosomatic medicine, 80(2), 141. 85
- Rebecchi, M., & Pentyala, S. (2002). Anaesthetic actions on other targets: protein kinase C and guanine nucleotide-binding proteins. British journal of anaesthesia, 89(1), 62-78.
- Reul, J. M., & Holsboer, F. (2002). On the role of corticotropin-releasing hormone receptors in anxiety and depression. Dialogues in clinical neuroscience, 4(1), 31.
- Rizzoli, R., Cooper, C., Reginster, J.-Y., Abrahamsen, B., Adachi, J. D., Brandi, M. L., . . . Ferrari, S. (2012). Antidepressant medications and osteoporosis. Bone, 51(3), 606-613.
- Russo, S. J., & Nestler, E. J. (2013). The brain reward circuitry in mood disorders. Nature Reviews Neuroscience, 14(9), 609-625.
- Schramm, E., Klein, D. N., Elsaesser, M., Furukawa, T. A., & Domschke, K. (2020). Review of dysthymia and persistent depressive disorder: history, correlates, and clinical implications. The Lancet Psychiatry, 7(9), 801-812.
- Šimić, G., Leko, M. B., Wray, S., Harrington, C. R., Delalle, I., JovanovMilošević, N., . . . Di Giovanni, G. (2017). Monoaminergic neuropathology in Alzheimer's disease. Progress in neurobiology, 151, 101-138.
- Somasundaram, D. (2007). Collective trauma in northern Sri Lanka: a qualitative psychosocial-ecological study. International journal of mental health systems, 1(1), 1-27. 86
- Steers, M.-L. N., Wickham, R. E., & Acitelli, L. K. (2014). Seeing everyone else's highlight reels: How Facebook usage is linked to depressive symptoms. Journal of Social and Clinical Psychology, 33(8), 701-731.
- Stepto, N. K., Moreno-Asso, A., McIlvenna, L. C., Walters, K. A., & Rodgers, R. J. (2019). Molecular mechanisms of insulin resistance in polycystic ovary syndrome: unraveling the conundrum in skeletal muscle? The Journal of Clinical Endocrinology & Metabolism, 104(11), 5372- 5381•
- Vizi, E. S. (2000). Role of high-affinity receptors and membrane transporters in nonsynaptic communication and drug action in the central nervous system. Pharmacological Reviews, 52(1), 63-90. 87
- Wang, W. Y., Barratt, B. J., Clayton, D. G., & Todd, J. A. (2005). Genome-wide association studies: theoretical and practical concerns. Nature Reviews Genetics, 6(2), 109-118.
- Whiteford, H. A., Ferrari, A. J., Degenhardt, L., Feigin, V., & Vos, T. (2015). The global burden of mental, neurological and substance use disorders: an analysis from the Global Burden of Disease Study 2010. PloS one, 10(2), e0116820.
- Whybrow, P. C., Akiskal, H. S., & McKinney Jr, W. T. (2012). Mood disorders: Toward a new psychobiology: Springer Science & Business Media.
- Wiles, N., Thomas, L., Abel, A., Barnes, M., Carroll, F., Ridgway, N., . . . Odondi, L. o. (2014). Clinical effectiveness and cost-effectiveness of cognitive behavioural therapy as an adjunct to pharmacotherapy for treatment-resistant depression in primary care: the CoBalT randomised controlled trial. Health Technology Assessment (Winchester, England), 18(31), 1.
- Wilson, C. J., Finch, C. E., & Cohen, H. J. (2002). Cytokines and cognition—the case for a head-to-toe inflammatory paradigm. Journal of the American Geriatrics Society, 50(12), 2041-2056.

- Wittchen, H. U., & Pittrow, D. (2002). Prevalence, recognition and management of depression in primary care in Germany: the Depression 2000 study. Human Psychopharmacology: Clinical and Experimental, 17(S1), S1-S11.