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An Overview of Indian Research in Depression

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ABSTRACT: ***Researchers in India have long been interested in depression as a disease. A vast number of research from India have been published in the previous 50-60 years, covering various facets of this widely widespread disease. Epidemiology, demographic and psychosocial risk factors, neurobiology, symptomatology, comorbidity, evaluation and diagnosis, effect of depression, treatment-related difficulties, and depression prevention, as well as the efficacy and tolerability of various antidepressants, were all investigated. Depression is a prevalent and treatable illness that is nevertheless under-diagnosed in primary care settings. There is a need to mobilise additional resources to deal with depressive illnesses in India, which has a national shortfall of 77% for psychiatrists. The present subtyping of depression is based on the categorical classification of bipolar and depressive disorders in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition, Text Revision (DSM-IV-TR). However, current research supports a dimensional approach to depression, which is defined as a continuum/spectrum of overlapping illnesses that range from bipolar I depression to severe depressive disorder. In this article, we look at statistics from India on several elements of depression.***

KEYWORDS: ***Depression, Clinical Features, Mental Disorders, India***

1. INTRODUCTION

In terms of its prevalence, as well as the pain, dysfunction, morbidity, and economic cost it causes, depression is a serious public health concern. Women are more likely than males to suffer from depression. The point prevalence of unipolar depressive episodes is estimated to be 1.9 percent for men and 3.2 percent for women in the Global Burden of Disease study, with a one-year prevalence of 5.8 percent for men and 9.5 percent for women. If present demographic and epidemiological trends continue, the burden of depression is expected to rise to 5.7 percent of the overall burden of illness by 2020, making it the second greatest cause of disability-adjusted life years (DALYs), after only ischemic heart disease. Depression as a disease has long been a focus of study for researchers in India due to the high morbidity. Several writers have attempted to investigate its prevalence, nosological problems, psychosocial risk factors such as life events, symptomology in the cultural context, comorbidity, psychoneurobiology, therapy, outcome, prevention, impairment, and burden. Some of the research also attempted to address concerns affecting youngsters and the elderly. This review focuses on studies conducted in India on a variety of depressive illnesses. A thorough internet search was conducted for this purpose, including key terms such as depression, life events, prevalence, categorization, cultural difficulties, result, prevention, handicap, and burden, among others, in various combinations. Pubmed, Google Scholar, Sciencedirect, Search Medica, Scopus, and Medknow were among the search engines utilised. In addition, a comprehensive search of all issues of the Indian Journal of Psychiatry that are available online was carried out. A hand search of some of the lost issues resulted in the discovery of a few additional articles. Review papers that were deemed to not adequately portray the Indian situation or to not cover the available Indian data were eliminated. In this collection of comments to be published, we look at treatment problems (antidepressants)

individually. Until it was deemed essential, data from animal studies and origins in the form of case reports and short case series were not included. Epidemiology, demographic and psychosocial risk factors, neurobiology, symptomatology, comorbidity, evaluation and diagnosis, effect of depression, treatment-related concerns, and depression prevention were used to arrange the available data[1]–[3].

2. LITERATURE REVIEW

According to Chhabra and Kar, Mood disorders were the most prevalent diagnosis in senior mental inpatients, who investigated the profile of psychiatric illnesses in elderly psychiatric inpatients (46.5 percent). Depression was shown to be prevalent in older research from geropsychiatric clinics, ranging from 13 to 22.2 percent. A recent outpatient research that assessed psychiatric morbidity in 100 randomly selected senior patients attending a geriatric clinic discovered that 29% of the patients had psychiatric disease, with depressive disorders being the most frequent. Another study found that depression was the most prevalent mental diagnosis among 1586 senior people (aged 60 and up) who visited the All India Institute of Medical Sciences' Geriatric Clinic in New Delhi. Guha and Valdiya found that major depressive disorder (13.4 percent) was the most prevalent psychiatric diagnosis in this cohort in a survey of elderly people[1].

According to Srinath et al., In a community sample from south India, there was a 0.1 percent incidence of depression in children and adolescents aged 4 to 16, but no kid aged 0 to 3 was diagnosed with depression. In a community-based research on school students, another study from north India found an annual incidence rate of 1.61/1000 children. A prevalence rate of 1.2 to 9.2% for affective disorders has been recorded in clinic-based research, with unipolar depression being the most frequent group in the majority of the investigations[3].

According to Malhotra et al., the prevalence of affective disorders in children (0-14 years) visiting psychiatric outpatient clinics increased from 2% to 13.49 percent. Post-natal depression has been shown to affect 11% of women during and after pregnancy, according to studies[4].

3. DISCUSSION

3.1 Studies on Neurobiology

In comparison to schizophrenia, India has done very little study on the neuroscience of depression.

3.1.1 Neurochemicals:

The amounts of catecholamine metabolites in cerebrospinal fluid, urine, and blood have been studied. Depressed individuals had considerably greater urine 5-hydroxyindoleacetic acid (5HIAA), a metabolic end product of serotonin, than controls, according to studies, and these values reduced after effective therapy. One research, however, found no change in CSF 5HIAA levels between depressed patients and healthy controls. There is a substantial positive association between the degree of depression and the patient's urine 5HIAA values, as well as a negative correlation between suicidal ideations and the patient's urinary and cerebrospinal fluid (CSF) 5HIAA and homovanillic acid (HVA) values. Platelet serotonin (5HT) uptake is substantially reduced in individuals with depression compared to controls, which improves briefly after Electroconvulsive Therapy (ECT). Treatment with imipramine resulted in a

substantial decrease in platelet 5HT uptake, but ECT resulted in a considerable increase in uptake, according to another research by the same authors. In terms of dopamine metabolites, depressed people have lower HVA levels than healthy people, which corresponds with platelet monoamine oxidase (MAO) activity, erythrocyte adenosine deaminase activity, and 5-HIAA levels. In addition, as compared to healthy controls, the HVA/5-HIAA ratio was shown to be lower in depressed patients. Both ECT and imipramine have been demonstrated in studies to produce a substantial decrease in platelet MAO activity, which returns to normal after therapy. It has been shown that individuals with psychotic major depressive disorder have substantially lower serum dopamine-hydroxylase activity than healthy controls. Dopamine-hydroxylase activity, on the other hand, did not differ between healthy controls, acute schizophrenia patients, non-psychotic depression patients, or mania patients. Srinivasan also discovered that urine 5-vinyl mandelic acid (VMA) levels are low during the acute period of depression, and that they drop much more with imipramine therapy. The investigators also discovered a similar result in normal participants when it came to imipramine's impact. Depressed patients' levels return to normal following therapy, whereas normal subjects' levels return to normal when treatment is stopped[4]–[7].

3.1.2 Electrophysiology:

Many studies have compared depressed individuals' pre-treatment P300 amplitude and latency to healthy controls. P300 amplitude is consistently lower in sad patients, and this decreases with recovery, suggesting that it might be a status marker for depression. The results on pre-treatment P300 latency, on the other hand, are mixed. P300 latency shows a strong positive association with patient age and degree of depression, whereas P300 amplitude has a significant negative correlation with age and severity of depression, according to studies. P300 amplitude is smaller in depressive participants, but there is no difference between depressed and dysthymic people, according to research. However, the pre-treatment P300 amplitude does not indicate treatment response. There is also no change in the latency of the middle components of the evoked potential between patients with melancholic depression and the control group, according to studies. The amplitude and frequency of Bereitschafts potential (BP), a marker of frontal brain functioning, are lower in patients with melancholic melancholy than in healthy controls, according to studies[2], [3], [8].

3.1.3 Endocrinology:

Dexamethasone suppression test (DST) has a poor sensitivity but a high specificity as a diagnostic for melancholia, according to research. DST non-suppressors, compared to DST suppressors, are substantially more depressed, attempt suicide more frequently, have higher rates of prior and family history of depression, require electroconvulsive therapy more frequently, and exhibit greater treatment response. A similar outcome in terms of treatment response was also observed in another research. Another research of post-stroke depression patients found that DST reaction correlates with clinical course and therapy response. A study using absolute eosinophil counts as an indirect measure of adrenocortical function found that the initial level of eosinophils in depressed patients was greater than normal before therapy, with a varied response to treatment, indicating different degrees of decreased adrenal cortex activity. Depressed people have greater plasma cortisol levels than healthy people, according to studies, and the rise has little to do with the degree of depression. Studies have also shown that an aberrant plasma cortisol level may distinguish patients with depression from patients with schizophrenia and healthy controls with a confidence level of 60% -93.33%. The

strongest indication of distinguishing depressed individuals from healthy people and patients with schizophrenia was urinary free cortisol following dexamethasone suppression testing. Measurements of adrenocorticotrophic hormone (ACTH) in individuals with severe depression were found to be substantially higher at baseline and post-DST when compared to healthy controls. A research looked at the function of dexamethasone in therapy based on the idea that it would speed up recovery, but it found no difference between dexamethasone and placebo[3], [4], [8], [9].

3.1.4 Immunology:

When depressed people are compared to those who have neurological or surgical disorders, total CSF protein levels have been shown to be significantly higher. Immunoglobulin (Ig) M, IgG, and IgA levels were found to be greater in depressed and neurologically unwell patients than in surgically ill subjects. Furthermore, when immunoglobulin levels were examined between depressed and normal patients, IgG and IgA levels in depressed subjects were substantially greater. However, no viral indicators for rubella or CMV were found in the CSF of depressed people, according to the study[1], [5].

3.1.5 Extracellular-fluid

The extracellular fluid (ECF) volume of individuals with depression is smaller than normal controls, according to Verma and Wig, and normalisation of the same corresponds with clinical recovery[1], [2], [5], [10].

3.1.6 Melatonin

Urinary melatonin levels have been demonstrated in studies to help differentiate those with endogenous depression from those with neurotic depression. Subjects with endogenous depression had low nocturnal and 24-hour urine melatonin levels, but those with neurotic depression have greater than normal levels. Melatonin levels were also shown to be linked to suicide attempts, diurnal fluctuation, and psychomotor impairment[1]–[3], [5], [9], [10].

3.1.7 Neurocognitive functioning:

According to studies from India, clear cognitive deficits in the domains of intelligence and memory are evident in the depressed state (Bhatia's Battery test or the Weschler Adult Performance Intelligence Scale and PGI memory scale), but they do not remain after recovery. The Wisconsin Card Sorting Test (WCST) is also found to perform poorly in patients with depression as compared to controls, indicating cognitive inflexibility and prefrontal dysfunction. Furthermore, on the WCST, more severe sickness is linked to a higher impairment in executive functioning. Other studies examining various cognitive domains have found that when patients with depression are asked to discriminate emotional tone in terms of intensity of facial expression while presented in pairs, they are more evaluative of sadness and less evaluative of happiness than the general population. Other research has found that when individuals with depression are asked to distinguish emotional tone in terms of intensity of facial expression when presented in pairs, they are more evaluative of sorrow and less evaluative of happy than the general population[1]–[3], [5], [9], [10].

3.1.8 Genetics:

Genetic variables have an important role in therapy response, according to a recent study. Patients who were homozygous for the long allele of the serotonin transporter gene had a much greater response to escitalopram than patients who were homozygous for the short allele or heterozygous for both the short and long allele. Short variations of the D7S1875 marker in the LEP gene were found to be a risk factor for depression in another research[1], [3], [5].

3.1.9 Lipids:

Recent research compared depressed patients to normal controls and discovered that there is a substantial increase in blood total cholesterol in depressed patients compared to normal controls, which remains even after adjusting for confounders. Another study found that measuring blood cholesterol levels in sad people might actually indicate hypothyroidism[1], [3], [5].

3.2 Impact of depression

Depressive disorders have been proven in studies to cause considerable dysfunction, impairment, and poor quality of life in sufferers, as well as a significant load on carers. Relatives of patients with affective disorders and schizophrenia have been demonstrated to have a similar pattern of burden, which is mostly felt in the areas of family routine, leisure, interaction, and money. Caregivers of people with depression, on the other hand, are less burdened than those caring for people with schizophrenia or bipolar illness. According to another study, the prevalence of dysthymia is comparable to that of neurotic illnesses such as obsessive compulsive disorder and generalised anxiety disorder. In comparison to normal/medically sick controls, people with dysthymia exhibit considerable impairment on measures of quality of life, disability, social support, and marital adjustment. The study also discovered that the length of illness and the degree of depression are the most significant predictors of poor quality of life and disability. Although both depressive and somatic symptoms were unpleasant, perceived stigma was higher for depressive symptoms, according to a research examining the connection between stigma and depression and somatization in psychiatric patients in south India. When compared to somatization symptoms, depressive symptoms were seen as socially unfavourable[3], [5]–[7].

3.3 Course and Outcome

Only a few research have looked at the progression and prognosis of depression. 3-13 years after initial index diagnosis, Venkoba Rao and Nammalvar followed up on 109 of 122 instances of endogenous depression. In 28 instances, there was no recurrence. 42 patients were found to be bipolar, whereas 21 remained unipolar. Depressive episodes were outweighed by manic events. In some cases, the transformation from depression to mania occurred within three years after the original depression, whereas in others it took anything from three to twelve years. Before the beginning of mania, the number of episodes of depression ranged from one to three. While individuals who had depression before the age of 40 were more likely to have recurrences, those who started the condition beyond that age had a higher chance of chronicity. Gada looked examined 92 out of 100 instances of major depressive disorder five to ten years after the first diagnosis and found that 36.6 percent had no recurrence and 63.4 percent had recurrence. Bipolar disorders were identified in 37.8% of the patients, whereas recurrent depressive disorder was diagnosed in 25.6 percent. In 77 percent of patients, the diagnosis of major depressive disorder was changed to bipolar

disorder within three years after the original depression. Brown stated in another research that in their sample, all patients recovered completely within a year. At a year's follow-up, 71% of depressed individuals had no symptoms or social impairment. The average length of a depressive episode was 14.2 weeks, with an 18% relapse rate. The authors found that the overall outcome was much better than in comparable trials of affective disorders conducted in industrialised countries. All of these data show that depressive illnesses have a high recurrence rate, and many of the people who were first diagnosed with unipolar depression were later diagnosed with bipolar disorder. Avasthi et al. observed in another prospective research of seasonal affective disorder (SAD) participants that following the first diagnosis of SAD, the subjects did not show any modification in mood, behaviour, sleep pattern, or weight fluctuation during a five- to seven-year period. According to a study of older individuals with depression, complete recovery occurred in 58 percent of cases, partial recovery in 24 percent of cases, and relapses in 18 percent of cases. Sleep problems, agitation, depression depth, age at current onset, age at first onset, history over one year with no symptom-free interval, abrupt start of disease, sufficient premorbid personality, and adequate psychogenesis were all shown to be predictive variables by Sachdev et al[1], [2].

3.3 Prevention

Sethi et al. proposed a depression prevention model that focuses on improving social networks and educational programmes that educate the public about the risks associated with changing jobs, residences, and patterns of living, as well as how to protect against them and eliminate malnutrition and infections. According to a recent study, religion may protect against depression[1], [8], [9].

4. CONCLUSION

Depression is a persistent, widespread, and burdensome condition that can be caused by a variety of biological and psychological causes. People who have chronic medical illnesses are more prone to acquire depression, and untreated depression can have a negative influence on the course and prognosis of the medical condition. Early detection and treatment can help to lessen impairment and even the risk of suicide. There are therapies for depression that are both safe and effective. In the primary care/medical environment, trained general doctors should be able to detect and successfully manage mild to moderate instances of depression utilising a mix of pharmacological and psychosocial therapies. Most community-based research reveals that depression is the most frequent mental illness. It's also one of the most frequent mental disorders observed in outpatient clinics and in medical and surgical settings. In some contexts, it is also believed to be the most prevalent mental illness among the elderly. Life experiences in the time leading up to the beginning of depression also have a significant influence in depression, according to studies from India. Interpersonal problems, marital discord, and sexual coercion have all been identified as risk factors in women's studies. Cost, attitude toward therapy, adherence, compliance, and neurobiological correlations all need to be studied further. There is also a need to research the course of depressive illnesses in India so that the need for and duration of follow-up treatment may be determined. Studies should also look at cost-effective treatment approaches that may be easily implemented in primary care to successfully treat depression[1], [2], [6], [8]–[10].

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