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ETIOLOGY AND PREVALENCE OF DEPRESSION

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ABSTRACT

Along with the dementias and anxiety disorders, depression is the most common psychiatric disturbance in late life. The evidence of studies conducted so far is that depression is associated with declines in well being and levels of daily functioning and a higher risk of functional impairment, mortality and use of health services. Despite this long-standing interest, there is no consensus on basic issues, such as the influence that age and age-related developments may have on the prevalence of depression. Studies using a broader definition of clinically relevant depression have yielded much higher prevalence rates. The prevalence of minor depression was measured using the Center for Epidemiologic Depression Scale (CES-D). This is a 20-item self-report scale developed to measure depressive symptoms in the community. In the United States the prevalence of PPD ranges from 7 to 20%, but most studies suggest rates between 10 to 15%. Many psychosocial stressors may have an impact on the development of postpartum depression. The prevalence of depression appears to vary by the site of the cancer: 50% prevalence in pancreatic cancer, 22-40% prevalence in oropharyngeal cancers, 10-26% prevalence in breast cancer, 13-32% prevalence in colon cancer, 23-25% prevalence in gynaecologic cancers, 17% prevalence in lymphomas and 11% prevalence in gastric cancers. Major depression in the elderly is more often the exacerbation of a chronic mood disturbance, with roots in long-standing personal vulnerability; while minor depression is more often a reaction to the stresses encountered in later life. Depression is associated with certain disease conditions including diabetes mellitus, cancer etc and may also caused due to the adverse effect of certain drugs. The depressive symptoms can be cured by psychological treatment with drugs (eg; antidepressants) and patient counselling. Thereby counselling centres can be developed to prevent further risk of depressive disorder and it's associated complications.

INTRODUCTION

Along with the dementias and anxiety disorders, depression is the most common psychiatric disturbance in late life [1]. The evidence of studies conducted so far is that depression is associated with declines in well being and levels of daily functioning and a higher risk of functional impairment, mortality and use of health services. From a public health perspective, depression may be one of the more prevalent and potentially amenable factors involved in the pathways leading to utilization of the health services by older people [2]. Despite this long-standing interest, there is no consensus on basic issues, such as the influence that age and age-related developments may have on the prevalence of depression. Since first studied over 30 years ago by Holmes and Rahe (3), stressful life events have been a major focus of psychiatric epidemiology (4). In particular, numerous investigations have found a correlation between the occurrence of stressful life events and the subsequent onset of an episode of major depression (5). Less certain, however, is the nature of the relationship between major depression and stressful life events. In particular, it remains unclear to what extent stressful life events cause subsequent onsets of depression and to what extent the occurrence of stressful life events and onsets of depression are correlated for other reasons. Anti-stigma organizations, like the National Alliance for the Mentally Ill (NAMI), suggest that acceptance of a biological model of mental illness will help reduce the stigma that those who are mentally ill encounter. To this end, NAMI uses strictly medical and biological terminology in their informational brochure about depression (NAMI, 2002). Pharmaceutical companies are also pedaling the view of depression as a biological disease. For example, the website for Zoloft explains the biological mechanism by which the antidepressant works (Zoloft, 2002). The hope is that by understanding the biology behind depression, one would seek a biological solution (a pill) to treat depression. A final pressure is coming from insurance companies.

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Etiology of Depression

Over the past few years, two major brain areas have been implicated in major depression the prefrontal cortex/anterior cingulate and the hippocampus. Lockwood *et al*. report on significant neuropsychological impairment in depressed patients, based on comparisons with healthy subjects. Elderly subjects demonstrated impairment in performance of tasks of executive function (suggestive of prefrontal/anterior cingulate dysfunction), and particular impairment was seen in elderly depressed subjects. These findings support a number of other observations involving the prefrontal region in depression (6). Taken together, these data at first glance point to a major consequence or correlate of depression, namely marked cognitive dysfunction involving the prefrontal cortex/anterior cingulate, but also suggest that a performance decrement in the elderly involving this region could be a risk factor for becoming severely depressed.

Excessive glucocorticoid activity

One possible common thread affecting both regions is excessive glucocorticoid activity. Administration of cortisol to healthy subjects results in dysfunction in both regions (6, 7). Frodl *et al*. note that although in rats high cortisol levels can relatively rapidly reduce hippocampal size, excessive glucocorticoids probably do not explain their hippocampal findings in the first-episode patients. On the basis of the time period involved, this seems sensible. However, they also cite the work of Sanchez *et al*. (8) in which type II glucocorticoid receptors were not appreciably found in the hippocampus of the rhesus monkey, although they were found heavily in the prefrontal cortex, as another reason for not invoking glucocorticoids. A human glucocorticoid receptor probe was used in that study. In contrast, Patel *et al*. (9), using specific probes for glucocorticoid receptors in the squirrel monkey, found glucocorticoid receptors in the hippocampus as well as in the frontal cortex. Thus, glucocorticoids could play a key role in the findings of both studies. A smaller hippocampus could be associated with higher glucocorticoid levels during stressful times, which could lead to cognitive dysfunction associated with effects in the prefrontal cortex/anterior cingulate and the hippocampus. In turn, excessive glucocorticoids could result in "atrophy" of specific brain regions. This area is worthy of further investigation and will require longitudinal study.

Stressful life events

Dependent stressful life events and onsets of depression might be associated because such stressful life events directly increase the risk of major depression (i.e., a causal relationship) and/or because some third set of factors such as genetic vulnerability predisposes to both stressful life events and major depression (i.e., a noncausal relationship).[10]

Prevalence of Depression

Depression in Geriatrics

Studies using a broader definition of clinically relevant depression have yielded much higher prevalence rates. Here, definition of caseness was determined by the level of depressive symptomatology and the likelihood that intervention would be deemed necessary by clinicians. This divergence of findings has prompted discussion as to the validity of current diagnostic criteria for depressive disorders among the elderly [11]. Within the older population, prevalence rates of both minor and major depression appear to shift. The prevalence of major depression tends to decrease among the older-old, while the prevalence of minor depression seems to be highest in the oldest age groups. Within the older population, the prevalence of most established risk factors for depression changes. For example, among the older-old, there is an increased probability of physical illness, loss of loved ones and cognitive decline. On the other hand, through excess mortality among those depressed at younger ages, the least vulnerable individuals may be expected to reach the older-old age strata [12]. The net effect may be either an increase, decrease or stability of the rates of depression with age. Clearly, age-related changes in the patterns of risk factors should be taken into account in studies of the prevalence of late-life depression. Minor depression was defined as all clinically relevant depressive syndromes, not fulfilling rigorous diagnostic criteria for major depression.

Measuring depression

The prevalence of minor depression was measured using the Center for Epidemiologic Depression Scale (CES-D). This is a 20-item self-report scale developed to measure depressive symptoms in the community. Major depression was defined according to DSMIII criteria, and operationalised using an adapted version of the Diagnostic Interview Schedule (DIS). The DIS is a criterion instrument which was also designed specifically for use in epidemiological studies. It has been used widely in elderly samples.

Prevalence of Comorbid Depression in Adults with Diabetes

Recent meta-analyses link depression in diabetes with hyperglycemia [13] and with an increased risk for complications of the metabolic disorder [14]. There is also evidence from three controlled trials to suggest that treatment of depression improves glycemic control [15-17]. An accurate estimate of depression prevalence is needed to help gauge the potential impact of depression management in patients with comorbid diabetes. Several studies concluded that the presence of diabetes doubles the odds of comorbid depression. Prevalence estimates are affected by several clinical and methodological variables that do not affect the stability of the odds ratio.

Stress Induced Depression

Stress cause brain disturbances that may cause depression or particular components of the depressive syndrome. Focussing on 5-hydroxytryptamine (5-HT) and the stress hormones, this question was answered in the affirmative, based on the following two considerations:

- Changes in the 5-HT and stress hormone systems produced by sustained stress mimic to a substantial extent the disturbances in these systems that may be observed in depression.
- Substantial evidence indicates that the 5-HT and stress hormone disturbances in depression are of pathophysiological significance and not merely a consequence of the depressed state or a product of stress generated by the depressed state [18].

A decrease of 5-HT metabolism has been ascertained in the brain of a subgroup of depression: this phenomenon is trait-

related and associated with heightened anxiety and increased aggression, both inwardly and outwardly directed aggression across diagnoses.

In addition 5-HT receptor disturbances may be observed in this type of patients. The data are most firm for the 5- HT1A receptor that was found to be downregulated in a trait related fashion. Based on animal data, one can assume this phenomenon to be associated with increased anxiety and aggression [19].

IHD Associated Depression

Major depression has been associated with mortality from ischemic heart disease (IHD). Affective states such as hostility [20, 21] and major depression [22-25] have been associated with mortality from IHD. It is unlikely that the association between major depression and IHD is due to cardiotoxicity of antidepressants, since adequate treatment of depression appears to lower the risk of IHD [26]. The National Health Examination Follow-up Study (NHEFS), [27] a longitudinal study of U.S. adults, included a four-item scale to assess depressed affect. Although this scale does not permit the assignment of a diagnosis of major depression, it has been shown to correlate with interviewer ratings of depression.

Postpartum Depression

Postpartum depression (PPD) is a mood disorder that is often unrecognized and undertreated and that affects 10 to 15% of new mothers.[28] In the United States the prevalence of PPD ranges from 7 to 20%, but most studies suggest rates between 10 to 15%. Many psychosocial stressors may have an impact on the development of postpartum depression. The greater risk of postpartum depression is a history of major depression and those who have experienced depression during past pregnancies. Untreated maternal illness disrupts the early mother-infant relationship and also contributes to short and long-term adverse child outcomes.[29] It also has negative effects on children including increased risk of impaired mental and motor development, infant cognitive competence, poor self-regulation, and low self-esteem and behavior problems.[30, 31] The DSM-IV-TR defines PPD not as a discrete disorder but a sub-category of major depressive disorder. Postpartum depression is characterized by sadness or loss of interest, including poor concentration, appetite disturbance, sleep deficit beyond that required for care of the baby, lack of or excessive concern for the baby, constant fatigue, and anxiety or irritability. It begins within four weeks after delivery.

Etiology: The etiology of PPD remains unclear. It is sometimes thought that postpartum depression is caused by lack of vitamins. No specific cause of PPD has been documented, but studies tend to show that more likely causes are the significant changes in a woman's hormones during pregnancy.[32] Levels of the hormones estrogen, progesterone, and cortisol drop dramatically within 48 hours after delivery. Women who develop postpartum depression may be more sensitive to these hormonal changes and drops in hormone levels after delivery.[33, 34] Management of postpartum depression is a vital part of adequate medical care. The obstetrician and pediatrician can serve important roles in screening for and treating postpartum depression. To prevent adverse outcomes associated with depression and its impact on the child, it is important that all health care professionals and nurse practitioners are aware of specific signs and symptoms, appropriate screening methods, and proper treatment.

Drug Induced Depressive Disorders

Drug-induced depressive disorders are classified in the DSM-III-R as organic mood syndrome, depressed type. The ability of certain drugs to cause depression is of clinical relevance because organic mood syndrome is a component of the differential diagnosis of depressive symptoms [35]. Consequently, psychiatric textbooks often provide different lists of drugs thought to be capable of causing depression. Strong evidence supporting the existence of causal associations is often lacking. There is no specific drug for which there is definitive evidence of a causal association with depressive symptoms or depressive disorders. Nevertheless, for a number of drugs, the evidence is suggestive, although not conclusively, of a causal association. Despite this, rational decisions about the continuation or discontinuation of drugs can often be made [36].

Examples of certain drugs that causes depression

5-blocker (propranolol)
Corticosteroids
Digitalis
H2 blockers
Metoclopramide
Sedative hypnotics
Methyldopa
Clonidine
Oral contraceptives
Anabolic steroids
Levodopa

Depression Associated With Cancer

Approximately 16-25% of newly diagnosed cancer patients experience depression or an adjustment disorder with depressed mood [37]. Depression has also been associated with functional limitations in cancer survivors [38] and both anxiety and depression can independently contribute to functional and overall health [39, 40]. Several factors have been suggested to explain the observed variations in cancer mortality, including unequal access to cancer screening and treatment, lifestyle factors (i.e., diet, exercise, alcohol use and smoking), socioeconomic status and tumor biology. The prevalence of depression appears to vary by the site of the cancer: 50% prevalence in pancreatic cancer, 22-40% prevalence in oropharyngeal cancers, 10-26% prevalence in breast cancer, 13-32% prevalence in colon cancer, 23-25% prevalence in gynaecologic cancers, 17% prevalence in lymphomas and 11% prevalence in gastric cancers. Effective long-term management of these problems remains a challenge [41]. Cognitive Behavior therapy (CBT) with cancer survivors typically includes stress management and problem solving [42] although other approaches are considered CBT interventions as long as they are based on the assumptions that cognitions can be monitored and altered, and in turn may facilitate behaviour change [43]. The investigation of short- and longer-term follow-up indicated that individual CBT has short-term effects

on both depression and anxiety. Long-term effects were not observed.

Obesity and Depression

Obesity is associated with an increased risk of mental illness; however, evidence linking body mass index (BMI)-a measure of overall obesity, to mental illness is inconsistent. Among overweight and obese U.S. adults, waist circumference or abdominal obesity was significantly associated with increased likelihoods of having major depressive symptoms or moderate-to-severe depressive symptoms. Thus, mental health status should be monitored and evaluated in adults with abdominal obesity, particularly in those who are overweight. In addition to the broad range of obesity-related physiologic outcomes, obesity is associated with an increased risk for a number of mental disorders (i.e., depression, bipolar disorder, panic disorder, anxiety, or many others) [44] that have a substantial impact on public health (e.g., associated with great burden of diseases and increased mortality, disability, and reduced quality of life) [45]. However, other studies showed that BMI was not or was even inversely associated with some forms of mental illness. A possible explanation for these inconsistent results is that BMI as a measure of overall obesity does not account for varying proportions of muscle mass, bone, and fat, or the distribution of body fat. The continuing increases in BMI and waist circumference in the United States and the projected increases in the prevalence of overweight and obesity [45] suggest that mental health status should be screened, monitored, and evaluated especially in people with abdominal obesity.

CONCLUSION

A tempting conclusion is that major depression in the elderly is more often the exacerbation of a chronic mood disturbance, with roots in long-standing personal vulnerability; while minor depression is more often a reaction to the stresses encountered in later life. Depression is associated with certain disease conditions including diabetes mellitus, cancer etc and may also caused due to the adverse effect of certain drugs. The depressive symptoms can be cured by psychological treatment with drugs (eg; antidepressants) and patient counselling. Thereby counselling centres can be developed to prevent further risk of depressive disorder and it's associated complications.

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