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The Global Burden of Major Depressive Disorder: Prevalence, Diagnosis, and Impact: A Comprehensive Review

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Abstract

In this review, we will discuss the major depressive disorders on the basis of neurobiological changes. MDD, a prevalent psychiatric condition, manifests as a complex interplay of genetic, environmental, and physiological factors with a substantial impact on individuals and societies globally. Here, the clinical assessment is fully based on diagnosis and the statistical manual for mental disorders, 5th edition (DSM-5). The pathophysiology involves diagnosis alterations in neurotransmitter systems, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, and inflammatory responses. Serotonin, dopamine, and norepinephrine imbalances contribute to mood disturbances, with the monoamine hypothesis highlighting the role of neurotransmitter deficiencies. Abnormalities in the HPA axis lead to hypersecretion of corticotropin-releasing factor (CRF) and cortisol, influencing stress responses in MDD. Additionally, inflammatory cytokines play a role in neuroinflammation, affecting neurotransmitter function and contributing to depressive symptoms. Power spectral analysis of electroencephalogram (EEG) signals reveals distinctive patterns, including altered delta, theta, alpha, and beta waves, providing insights into neural activity changes in MDD. Spectral asymmetry, particularly in the frontal regions, further indicates neurobiological correlates of depression. Conventional treatments primarily include antidepressant medications and psychotherapy, although challenges in efficacy and tolerability exist. Understanding the multifaceted aspects of MDD is crucial for improving diagnosis, treatment, and overall mental health outcomes. Potential biomarkers and emerging therapies are considered treatment responses. By critically examining existing challenges and embracing innovative approaches, this review aims to contribute to the ongoing efforts to improves the treatment landscape for individuals grappling with major depressive disorder.

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INTRODUCTION

The most common cause of mental distress in later life, depression considerably lowers the quality of life of the people [1]. It significantly diminishes one's capacity to work and accounts for one-third of all disabilities experienced by people in both developed and developing nations [2].

Prevalence

Depression is a widespread mental health condition, impacting approximately 280 million individuals globally. According to the World

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Health Organization (2021), the prevalence rate of depression is 3.8%. Globally, it is estimated that 5% of adults and 5.7% older than 60 years suffer from depression. Depression is usually predominant in women. The danger of severe depression is marked by its increased suicidal tendency [3]. According to the WHO's predictions, MDD will overtake all other diseases as the third leading cause of disease burden globally by 2030 [4]. Around 7 million Bangladeshis struggle with anxiety and depression [5]. Depression frequently begins in adolescence or early adulthood, and it typically affects women more than men. Women of their reproductive age are the most severely affected, with 10–20% reporting postpartum depression [4].

Diagnosis

Several types of depressive disorders exist, such as nonspecific depressive disorder, disruptive mood dysregulation disorder, premenstrual dysphoric disorder, substance- or medication-induced depression, persistent depression, and depressive disorders brought on by other medical conditions [4]. Major depressive disorder (MDD) is a specific category of depression characterized by persistent feelings of unhappiness and reduced interest in activities throughout most of the day, nearly every day, for at least a two-week period. Those with MDD may struggle to focus, feel guilty or hopeless, have sleep issues, change their appetite, or feel exhausted [3].

A clinical diagnosis major depressive disorder is mostly determined by a clinical history and mental status evaluation. As per the Diagnostic and Statistical Manual for Mental Disorders, fifth edition (DSM-5), if an individual displays five or more symptoms within a two-week period, including at least one symptom of either persistent sadness or diminished interest, they may be diagnosed with depressive disorders.

- Experience of unhappiness, weeping, or an empty or hopeless feeling
- Outbursts of anger, irritability, or frustration
- Diminished interest and enjoyment in most or all usual activities, including sex, hobbies, or sports
- Disruption in sleep patterns
- Fatigue and a lack of energy
- Decreased appetite and weight loss
- Feelings of anxiety, restlessness, or agitation
- Slowed thought processes, speech, or bodily movement
- Sense of worthlessness or guilt
- Difficulty with thinking and concentration
- Persistent thoughts of death or suicidal ideation, planning, or attempts
- Unexplained physical issues, such as back pain or headaches

MDD can be classified into mild, moderate, severe, and extremely severe based on the appearance and severity of symptoms [6]. Deficits in frontal functioning measure the cognitive areas, including learning and memory, as well as executive processes like attention and focus, which are characteristics of MDD [7]. Recently, it has been found that the perceived lack of attention among patients with major depressive disorder (MDD) strongly correlates with reduced workplace productivity and diminished quality of life [8].

Evaluation

There is no objective test to diagnose depression. Individuals are incidentally diagnosed as patients with depression by health professionals. The state of the depression evaluation at every visit is very important. For screening, diagnosing, and monitoring treatment responses for MDD, there are some scales [4]. In primary settings, one commonly used tool is the Patient Health Questionnaire-9 (PHQ-9), a standardized self-report scale designed to evaluate psychological impairment related to depression. The PHQ-9 scores can range from 0-27. It is designed to screen for depressive symptoms.

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Through PHQ-9, the validity of the measure can be examined [9]. To evaluate depression, most frequently, the Hamilton Rating Scale for Depression (HAM-D) is used in hospital settings.

The scale contains 21 items; among them, first 17 items score for depression [4]. In Bangladesh, the Depression Anxiety Stress Scales 21 items (DASS-21) and the Beck Depression Inventory (BDI) are commonly used for the assessment of depression. DASS-21 is a multidimensional scale that was validated only among medical students. Its use among patients is uncertain [10].

There are some other scales for rating depression, like the Montgomery-Asberg Depression Rating Scale (MADRS), the Zung Self-Rating Depression Scale, the Ruskin Depression Rating Scale, and others [6]. In recent years, MADRS has been translated into a Bangla version. It was developed in the 1970s and since then has never been updated or modified [10].

The Beck Depression Inventory (BDI) is extensively utilized in both research and clinical settings to screen for depression, assess behavioral indicators, and determine the severity of depressive symptoms. Created in 1961 by psychiatrist Dr. Aaron T. Beck, he is recognized as a pioneer in cognitive therapy. The BDI is a self-rated scale where the 21-items cover both somatic and psychological, including cognitive and vegetative or affective symptoms measuring items. It demonstrates strong internal reliability. The BDI is applicable across a wide age range, from 13 to 80 years old. After updating some items and aligning them, in 1996, the BDI was reformed to BDI-II in the Diagnostic and Statistical Manual of Mental Disorders (DSM). This is used to measure the severity of the screening and to monitor the course of treatment of depression. For the BDI-II, a score of 10–18 indicates mild depression, 19–29 indicates moderate depression, and more than or equal to 30 indicates severe depression. The validity and reliability of BDI have been tested across populations worldwide. It takes approximately 10 minutes to complete. BDI has been translated into many languages. Recently, in 2020, the BDI was validated in the Bangla version to measure depression and its severity among the Bengali population [6, 10].

Etiology

Certain sociodemographic, behavioral, and psychological variables can have an impact on mental health [5]. Many factors, including biological, genetic, environmental, and psychological ones, contribute to the etiology of MDD [3, 4]. Medical conditions are the main biological etiology of depression. Biological factors include medical illness, genetic susceptibility, vascular risk, abnormalities in neurotransmitters, and endocrine changes. 20% of individuals with Perkinson's disease fulfill the criteria for serious depression. Some diseases, like heart disease, dementia, and hip fractures, are associated with depression. Serotonin, norepinephrine, dopamine, GABA, glutamate, and glycine are among the neurotransmitters found to exhibit abnormalities in major depressive disorder (MDD). Reduced serotonin levels have been noted in individuals experiencing suicidal ideation [4].

According to recent research, depression may be exacerbated by vascular lesions in particular brain regions. Depressed elderly people had reduced left hippocampus volume and orbital frontal cortex sizes, according to research [11]. These modifications could be a result of biological modifications in non-neural (glial) tissue that impact synaptic activity. The chance of developing depression is higher in first-degree relatives of depressed people [12]. Genetic factors explained 16% of the variation in the total number of depression complaints in a twin community sample [13]. Endocrine alterations are linked to depression, including an increase in corticotropin-releasing factor (CRF) secretion. Depression is also associated with decreased levels of testosterone and dehydroepiandrosterone sulfate (DHEA-S) [11, 13].

Complications

For many years, it has been observed that a lack of social support correlates with depression. The main contributing factors to depression are loneliness, unemployment, divorce, and poverty, which indicate a loss of social engagement and increased self-involvement [12, 13]. Depression has a substantial impact on the physical health and overall quality of life of those who are affected,

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depending on the disease's severity. Globally, MDD is one of the principal causes of illness. It has a detrimental effect on family and bodily functions. It decreases life quality by causing functional impairment and negatively affecting interpersonal relationships. For instance, depression dramatically lowers workplace efficiency [2].

The majority of research on depression and its relationship to other illnesses, particularly chronic medical problems, indicates that depression raises the risk of the onset of other illnesses as well as their prognosis and death. If MDD is not treated, it becomes crippling. Weight gain as a consequence, potentially leading to conditions like diabetes and heart disease, may result from major depressive disorder (MDD). Individuals with MDD may also suffer from symptoms such as pain, anxiety, panic disorder, or fear. Depression significantly raises the likelihood of chronic physical illnesses, including hypertension, diabetes, asthma, arthritis, chronic respiratory diseases, chronic pain, and cardiovascular diseases [4]. It is reported that major depressive disorder (MDD) is influenced by cognitive impairments as functional outcomes in patients [8]. The presence of MDD increases the risk of suicide, making it the fourth leading cause of death among individuals under 30. Nearly 700,000 people die by suicide each year [3, 4].

Pathophysiological Basis of Depression

Serotonin, alternatively known as 5-hydroxytryptamine (5-HT), is a neurotransmitter essential for controlling mood, emotions, and various brain functions. The serotonin hypothesis of depression suggests that imbalances in serotonin levels may contribute to the onset and persistence of depressive symptoms. Serotonin is primarily associated with mood regulation. It helps modulate emotions, promotes feelings of well-being, and contributes to an overall sense of happiness. Research has hypothesized that individuals with depression often have lower levels of serotonin in their brains. The serotonin system relies on a delicate balance between its production, release, and reuptake. Serotonin is released into the synaptic cleft, where it binds to receptors on the receiving neuron and transmits signals. The serotonin transporter (SERT) is a protein responsible for reabsorbing serotonin back into the neuron that released it. In depression, there may be an overactivity of SERT, leading to reduced serotonin levels in the synapse and impaired neurotransmission [11, 14]. Serotonin, also known as 5-hydroxytryptamine (5-HT), is a neurotransmitter that plays a vital role in regulating mood, emotions, and many physiological processes in the brain. The serotonin hypothesis of depression suggests that imbalances in serotonin levels may contribute to the onset and persistence of depressive symptoms. Serotonin is primarily associated with mood regulation. It helps modulate emotions, promotes feelings of well-being, and contributes to an overall sense of happiness. Research has shown that individuals with depression often have lower levels of serotonin in their brain. The serotonin hypothesis of depression suggests that imbalances in serotonin levels may contribute to the onset and persistence of depressive symptoms. Serotonin is primarily associated with mood regulation. It helps modulate emotions, promotes feelings of well-being, and contributes to an overall sense of happiness. The serotonin system relies on a delicate balance between its production, release, and reuptake. The serotonin transporter (SERT) is a protein responsible for reabsorbing serotonin back into the neuron that released it. In depression, there may be an overactivity of SERT, leading to reduced serotonin levels in the synapse and impaired neurotransmission [11, 14]. Though this relationship is not entirely straightforward or uniform, the serotonin system depends on maintaining a precise equilibrium involving its production, release, and reuptake processes. The serotonin transporter (SERT) is a protein responsible for reabsorbing serotonin back into the neuron that released it. In depression, there may be an overactivity of SERT, leading to reduced serotonin levels in the synapse and impaired neurotransmission [11, 12, 14].

Many crucial brain functions rely on the presence and activities of diverse neurotransmitters at the membranes of neurons in the brain. Specific neurotransmitters are implicated in the development and clinical expression of depression, often described as the "monoamine hypothesis." This theory suggests that a functional deficit of these neurotransmitters is caused by the breakdown effects of monoamine oxidases within the presynaptic neuron. Research supports this theory by showing increased activity of monoamine oxidase enzymes in individuals with depression. The ongoing action

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of these enzymes significantly reduces the availability of biogenic amines, leading to the diminished neurotransmission observed in depression [11, 14].

In the context of neural signal transmission across synapses, the concentration of neurotransmitters (NT) in the synaptic cleft is regulated by reuptake and enzymatic destruction after releases of NT. NT balance is maintained by its balanced uptake or enzymatic destruction. Reuptake of NT is dependent on some specific transport protein on the presynaptic membrane. Therefore, the deficiency or dysfunction of this transport protein may contribute to the reduced reuptake and thereby reduce the level of monoamine NT in this group of patients. Observed deficiency or dysfunction of this monoamine transport protein in depressed patients causes a reduced concentration of monoamine NT in the synaptic cleft due to over degradation by monoamine enzymes. Thus, reduced numbers or impaired function of transport proteins could contribute to the lower levels of monoamine neurotransmitters observed in depression [14].

Not only at the presynaptic terminal, receptor dysfunction on the postsynaptic membrane also contributes to neurotransmitter disorder in depression. In individuals with depression, there are reported abnormalities in the numbers and sensitivity of serotonin receptors (such as 5-HT1 and 5-HT2) in the brain. Additionally, there is hypersensitivity of presynaptic α2-adrenoceptors, which inhibit further release of norepinephrine (NE) [11, 14, 15].

In addition to the neural factor, abnormalities in the endocrine factor may also play an important role in the development of depression. A literature review suggested that abnormalities in growth hormone (GH) levels, thyroid hormone irregularities, and dysfunction of the hypothalamus-pituitary adrenal (HPA) axis are potent endocrine abnormalities. Thyroid hormone dysfunction may indirectly produce symptoms of depression through the serotoninergic and/or adrenergic systems. Thyroid hormone increases the cortical release of serotonin and NE from the adrenal gland. On the other hand, a reduce level of serotonin is responsible for reducing the secretion of TSH. [11,14]. Studies on depressed individuals have shown increased secretion of corticotropin-releasing factor (CRF) and cortisol, along with dysfunctional feedback mechanisms of glucocorticoids. There is also evidence of inadequate suppression of the HPA axis following the administration of external glucocorticoids, as well as impaired signaling of corticosteroid receptors [15]. This hyperactivity of the HPA axis is demonstrated by the inability to reduce plasma cortisol levels after administering external glucocorticoids to individuals with depression. Those affected show notably elevated levels of cortisol in both plasma and saliva compared to non-depressed individuals. While some researchers have found no indication of heightened cortisol response in depressed individuals, others have shown increased cortisol responses in this group. Elevated cortisol levels are linked to the intensity and severity of depression, and their persistence increases the risk of depressive symptom recurrence [11,15].

Cytokines, chemical inflammatory mediators secreted primarily by white blood cells in response to foreign antigens, have also been implicated in depression. Cytokines include pro-inflammatory (e.g., IL-1, IL-6, and TNF) and anti-inflammatory (e.g., IL-4, IL-8, IL-10, and IL-13) categories [11]. In depression, elevated levels of pro-inflammatory cytokines are found in both the blood and cerebrospinal fluid of affected individuals. However, individuals with depression often exhibit an imbalance favoring pro-inflammatory cytokines over anti-inflammatory ones. This imbalance may contribute to the persistence of depressive symptoms [11, 14, 15]. Considerable evidence suggests that altered cortisol and corticotrophin-releasing hormone (CRH) are involved in depression. The hypothalamic-pituitary-cortisol hypothesis proposes that an altered cortisol response to stress is linked to depression. The brain cortex and amygdala detect stress signals, prompting heightened secretion of CRH. Cortisol levels can be elevated in severe depression, with an increase in the size of the anterior pituitary and adrenal cortex, as well as elevated CRH levels and CRH expression in the limbic brain. Elevated cortisol levels can lead to a feedback system of glucocorticoid abnormalities. On the other hand, stress stimulates the release of excess glutamate, which is an exitotoxin causes neural degeneration. Excititoxic neural degeneration in chronic stress may lead to depression [11, 12, 14]. Serotonin, or 5-hydroxytryptamine (5-HT), is a neurotransmitter essential for regulating mood,

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emotions, and numerous physiological functions in the brain. The serotonin hypothesis of depression suggests that imbalances in serotonin levels may contribute to the onset and persistence of depressive symptoms. Serotonin is primarily associated with mood regulation. It helps modulate emotions, promotes feelings of well-being, and contributes to an overall sense of happiness. The serotonin system relies on a delicate balance between its production, release, and reuptake. The serotonin transporter (SERT) is a protein responsible for reabsorbing serotonin back into the neuron that released it. In depression, there may be an overactivity of SERT, leading to reduced serotonin levels in the synapse and impaired neurotransmission [11, 14].

Power Spectral Features of EEG in Depression

In the past decades, mental disorders were evaluated by observation of symptoms. But recently, analysis of EEG has shown promising results for understanding changes in brain activity associated with various mental illnesses or disorders like epilepsy, schizophrenia, dementia, and others [16, 17, 18]. The EEG is currently employed in numerous clinics for standard diagnostic applications and as a biomarker for neuropsychiatric research due to its non-invasive value and cost-effectiveness. Quantitative methods of analysis enable the characterization of serotonin, also known as 5-hydroxytryptamine (5-HT), a neurotransmitter that plays a crucial role in regulating mood, emotions, and various physiological processes in the brain. The serotonin hypothesis of depression suggests that imbalances in serotonin levels may contribute to the onset and persistence of depressive symptoms. Serotonin is primarily associated with mood regulation. It helps modulate emotions, promotes feelings of well-being, and contributes to an overall sense of happiness. Research has shown that individuals with depression often have lower levels of serotonin in their brains, although this relationship is not entirely straightforward or uniform. The serotonin system relies on a delicate balance between its production, release, and reuptake. The serotonin transporter (SERT) is a transport protein responsible for reabsorbing serotonin back into the neuron that released it. In depression, there may be an over activity of SERT, leading to reduced serotonin levels in the synapse and impaired neurotransmission [11, 14]. Signal ocillations have been used as a tool to assess physiological and pathological cognitive processes [16, 17].

Power spectral analysis involves decomposing the EEG signal into its constituent frequency components, often categorized into different frequency bands. The fundamental frequency bands of EEG signal analysis include delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), and beta (13–30 Hz) waves. These frequency bands are associated with different cognitive and emotional processes, and alterations in their power can provide valuable information about the neurophysiological aspects of MDD [19, 20].

MDD patients showed significantly lower delta power in the frontal and occipital regions compared to healthy controls. However, the central, temporal, and parietal regions showed a slightly higher delta, but it was significant only in the frontal region [21]. But several other studies reported significantly higher delta power in individuals with MDD compared to healthy controls [22]. Delta oscillations are associated with slow-wave sleep and deep states of relaxation. Elevated delta power in MDD may indicate disrupted sleep patterns and disturbances in resting-state brain activity [20, 23, 24].

MDD patients showed lower theta signal powers in all regions compared to healthy individuals [21]. Conversely, excessive theta activity in MDD has also been noted in antidepressant-resistant patients [17]. MDD is often linked to abnormalities in theta power, which are associated with cognitive processes, including attention and working memory. Some researchers suggested that individuals with MDD may exhibit elevated frontal theta power during tasks requiring sustained attention, reflecting altered cognitive processing in depression [19, 20 ,23, 24].

MDD patients showed lower alpha signal powers in all regions compared to healthy controls [21]. It was also noticed that enhanced EEG alpha activity at rest has consistently emerged as a

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characteristic among individuals with depression. Various studies have indicated higher absolute and relative alpha power, particularly in regions such as the parietal, frontal, or occipital areas. Some studies have suggested that elevated alpha power could potentially be associated with a favorable response to antidepressant treatments. Additionally, increased slow wave activity has been discussed as another EEG biomarker in MDD [17]. Alpharhythms are associated with states of relaxation and reduced mental activity. The decrease in alpha power may reflect heightened cortical activity and difficulties in maintaining a calm and relaxed mental state in individuals with depression [19, 20, 23].

MDD patients showed increased beta signal powers in depressed individuals compared to healthy controls in the central, temporal, and parietal regions [17]. Higher beta power in individuals with MDD has been reported in some studies. When depression was associated with anxiety, EEG showed high beta activity. In health, beta waves are associated with active cognitive processes, including decision-making and motor control. Increased beta power may suggest more cognitive engagement in individuals with depression. Alterations in delta, theta, alpha, and beta power reflect disruptions in neural activity and cognitive processing associated with MDD. The findings related to power spectral features in MDD are not entirely consistent across studies. Individual variability, differences in EEG recording protocols, and comorbid conditions can contribute to discrepancies in research findings [19, 20, 23, 24].

Spectral asymmetry is an index of depressive disorder that is significant in all EEG channels [16]. Previous studies suggested that left frontal hypoactivation is prominent in depressive individuals, characterized by relatively high left alpha activity. In addition, frontal alpha asymmetry expressed in increased alpha power in the left frontal areas in comparison to the right frontal areas are also evident in depressives [25, 26]. Some authors found increased frontal alpha bilaterally [26]. Many studies found relatively higher alpha activity in the right posterior, especially parieto-temporal region, in depressed subjects in comparison to healthy individual [25, 26]. And these changes suggest a behaviorally non-responsive state in MDD patients. Frontal-lobe dysfunction has been repeatedly reported in patients with MDD. Attention and executive functions are impaired in MDD patients, and this suggests fronto-subcortical dysfunction. Many previous studies have shown a moderate effect size of neurocognitive deficit in patients with MDD in domains of processing speed, attention, executive functions, learning, and memory. Recently, cognitive dysfunction, including attention, memory, and processing deficits, was suggested as a primary mediator of functional impairment in patients with MDD [8, 16, 25].

The variability and unpredictability of EEG findings among depressed patients may be linked to the diverse clinical manifestations of depression and its co-occurrence with other disorders [17, 25].

Conventional Treatment

The most commonly used treatments for depression are antidepressants, psychotherapy or a combination of drugs and psychotherapy. For some individuals, psychotherapy may present challenges or may not suffice. The primary treatment approach for major depression remains antidepressant medication [27]. Antidepressants need several weeks of consistent use to achieve therapeutic benefits. The delayed therapeutic effects of antidepressants suggest issues related to the processing of neural network information rather than solely chemical imbalances [28]. Classical anti-depressants like selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants may lack some effectiveness and tolerability in patients with different depressive disorders [29].

Though antidepressants are widely prescribed by physicians, they are costly. Much less than half of the sufferers treated obtain complete remission via remedy with a single antidepressant drug. The most common side effects of these drugs are observed due to an increase in rapid monoamine concentration. These drugs have many side effects, like increased anxiety, gastro-intestinal and sexual problems, and decreased alertness due to increased monoamine concentration.

CONCLUSION

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Major depressive disorder poses a significant and growing global burden, affecting millions of individuals and impacting various aspects of life. As we approach 2030, it is imperative to address the predicted rise in MDD's disease burden and implement effective strategies for prevention, early diagnosis, and intervention. The integration of diverse diagnostic tools, including validated scales and EEG analysis, can enhance our understanding of MDD's neural correlates. Moreover, a comprehensive approach to treatment, considering the individual's unique biological, psychological, and social factors, is crucial for improving outcomes and reducing the associated complications. Continued research into the dynamic interactions within the brain's neural circuits holds promise for developing novel therapeutic strategies and advancing our understanding of the complex neurobiology underlying major depressive disorder.

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Author Contributions

Dr. Marefa Tuz Zohora Lima: writing, reviewing, graphics, and supervision; Fatema Tuz Zohora Toma: editing and conceptualization; and Tanvir Ahmed Tamim: draft preparation. Israt Jahan and Esaba Sadia are reviewing. All the authors have accepted responsibility for the entire content of this submitted manuscript and approved submission.

Declaration of competing interest

The authors declare the following financial interests or personal relationships that may be considered potential competing interests:

Fatema Tuz Zohora Toma reports a relationship between the Bangladesh Atomic Energy Commission, Dhaka Central International Medical College, and the Ministry of Health and Family Welfare, People's Republic of Bangladesh, which includes non-financial support.

Conflict of interest statement

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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