Understanding childhood depression

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Major depressive disorder in children is a severe and a chronically disabling disorder. This population appears to be a special group in terms of consequences of poor psychosocial and academic outcome and increased risk of substance abuse, and suicide. Studies have revealed several major findings in genetic, familial, psychological, and biological aspects of such depression, some of which have explored into the issue of its relationship with adult depression. Considerable advances have been made now in the area of childhood depression providing a better understanding of its nature. We review literature available on historical aspect, epidemiology, clinical characteristics, and aetiology of childhood depression.

Key words Aetiology - assessment - childhood depression - epidemiology - genetics - historical perspective - management

The existence of major depressive disorder in children was controversial before the late 1970s as it was felt that pre-adolescent children were incapable of experiencing depression because of immature personality structures. Review of literature has revealed reports of clear description of depressive symptoms in children in the past but these were not diagnosed as cases of depression due to lack of diagnostic criteria, and were even denied on theoretical grounds. The thinking changed after the fourth Congress of the Union of the Pedopsychiatrists was held in Stockholm in 1970. The theme of this Congress was “Depressive states in childhood and adolescence.” This conference put steam into the research that clearly showed children were capable of experiencing episodes of depression that met standard criteria. Research over the past 30 yr have established clearly that children are capable of experiencing depressive states but the manifestations are not always like adult depression. Studies done in the area of neurobiology and psychosocial factors have revealed interesting leads to the fact that adult depression has its basis in experiences in childhood. Understanding childhood onset depression helps not only to detect cases but also to reduce morbidity associated with it and to intervene early to prevent its continuation into adulthood.

Historical perspective

According to the psychoanalytic theory and stages of psychosexual development it was shown...
that the development of depressive disorder is precluded until the development of super-ego in young children. However, others argued that depression did occur in children but it assumed clinically different forms such as ‘anaclitic depression’, masked depression’ and ‘depressive equivalents’.

**Concept of anaclitic depression:** The first study to describe a form of childhood depression with symptoms akin to adult depression was by Spitz and Wolf in 1946, who observed that infants between 6 and 11 months when separated from their primary caregiver, developed symptoms over weeks resembling adult depression viz., sad, apprehensive facial expression, with progression through crying and screaming, to apathy, reduced babbling, and reduced physical activity, to complete withdrawal, dejection and detachment from environment. This was also accompanied by greater susceptibility to inter-current illness and a decline in the development quotient, with the condition often continuing for more than five months. It was referred to as ‘anaclitic depression’.

**Concept of masked depression:** The traditional psychoanalytic view held that children were developmentally immature to have developed a harsh super-ego to drive their aggressiveness towards their own ego. Depression was present but masked by other behaviours called ‘depressive equivalents’ which included conduct problems (hyperactivity, delinquency, aggressiveness, irritability), somatic complaints (headache, stomach ache, and enuresis), school problems (school phobia, poor school performance).

Frommer divided childhood depression into three groups, enuretic depressives as moody, immature, hostile and aggressive; phobic depressives as irritable, weepy and tense, who sometimes denied any feelings of depression; and pure depressives or mood disorders as weepy, irritable, with temper outburst, sleep disturbances, some with anti-social behaviour.

According to Strober et al. symptoms of “masked depression” are in some instances, early prodromal manifestations of illness in predisposed individuals, whose specific nature is influenced by the child’s age and personality structure. There are subgroups of children and adolescents genetically predisposed to depression who manifest an incomplete form of illness via psychosomatic disturbance, drug abuse, conduct problems, etc., symptoms that overshadow the underlying affective basis of their psychopathology.

**Typical depression:** Cytryn and Mcknew reported the presence of typical depressive syndrome consisting of dysphoria, hopelessness, social withdrawal, psychomotor retardation, sleep and eating disturbances, and other depressive symptoms characteristics of adult depression. They classified “typical” depression as (i) acute depression without previous psychiatric disorders and significant familial psychopathology; and (ii) chronic depression with poor adjustment before the onset of the disorder and a tendency to develop from a maladjusted environment.

Weinberg et al. were the first to use diagnostic criteria, to diagnose depression in children between 6-12 yr, loosely derived from Feighner’s criteria for major depressive disorder for adults. The depressed patients were treated with tricyclic drugs. In childhood depression they observed (i) an absence of severe psychomotor retardation; (ii) a change in behaviour, aggressive children showed inappropriate aggression, passive children withdrew; (iii) hyperactivity in a depressed child may correspond to manic behaviour in adults; (iv) depressed children did show a high incidence of school phobia, enuresis, and temper tantrums. When such developmental behaviour lessened in adolescence, depression becomes easily recognizable; and (v) with antidepressant medications significant improvement was noted.

Puig-Antich et al. used research diagnostic criteria and structured interview schedule
(K-SADS-P) in their study of children aged 6-18 yr and observed that depressed children displayed a disorder similar to adult major depressive disorder.

In 1980s consensus evolved regarding the similarities between child and adult major depressive disorder, which is reflected in classificatory systems (DSM-III, DSM-IV, ICD-9, 10) where no distinction had been made between pre-pubertal and adult depression. However, certain age-related considerations in diagnostic criteria were included (Table). Several other clinical syndromes of depression like adjustment disorder, mixed anxiety and depression are seen in children. Validating data on these disorders do not exist in children. At present adult diagnostic criteria are applied to diagnose these disorders in children. Reasons for this could be that it was first necessary to establish the existence of depression in childhood and only then research into various other depressive syndromes could be carried out.

**Assessment**

There are unique developmental challenges and considerations in diagnosing and treating childhood depression. For example, children often have difficulty in expressing or recalling information related to their disorder due to which it is often required that corroborating information be obtained from parents, school teachers, and other adults in the child’s life. The other factor that complicates diagnosis and treatment is the high rate of comorbidity that is characteristic of childhood depression. Three methods are considered for diagnosis of childhood depression and various comorbidities.

**A clinical/developmental interview:** The clinical/developmental interview in the form of open ended questions is needed to assess the background and onset of the mood episode by providing a developmental story of the disorder which seeks to describe the current presentation in the light of child’s social, physical, cognitive, linguistic, emotional development and its interaction with life events. This also includes a mental state examination which assesses apart from the mood state, the child’s cognitive development.

**A structured interview:** Structured interviews are frequently done for research purposes by trained researchers, *e.g.*, Child and Adolescent Psychiatric Assessment. There are a number of problems like low reliability in younger age groups, discrepancy between different sources of information and it has

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<th><strong>Table.</strong> Comparison of child related modifications of major depressive episode in DSM-III, DSM-III-R, DSM-IV, and ICD-10</th>
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<tr>
<td><strong>DSM-III</strong></td>
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<td>Dysphoric mood; sad, irritable, hopeless; for children &lt;6 yr old consider persistently sad facies</td>
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<tr>
<td>Anhedonia; for child &lt;6 yr old, signs of apathy</td>
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<td>Poor or increased appetite or weight; for children &lt;6 yr old consider failure to make expected weight gain</td>
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DSM, Diagnostic and Statistical Manual-III,III-R (revised), IV
ICD, International Classification of Diseases, Tenth revision
been suggested that interview with both the parents and the child is required till 14 yr of age\textsuperscript{13}. An alternative method of assessing psychopathology in children aged 10 yr or less includes a structured pictorial questionnaire\textsuperscript{14} based on DSM and puppet interviews\textsuperscript{15}.

\textbf{Self- and observer-rating scales:} Self-rating scales are probably best used as screening devices or as severity measures once the diagnosis is made\textsuperscript{16}. The sensitivity of depression rating scales may be high, but the specificity is low. There are fewer observer-rating scales, the most widely used are the Children’s Depression Rating Scale\textsuperscript{17}; and the Hamilton Rating Scale for depression\textsuperscript{18}.

\section*{Epidemiology}

\textbf{Prevalence:} Varying prevalence rates have been reported for depression. Such variations may result from differences in samples studied, in sample sizes, and diagnostic criteria applied, and the type of interviewing techniques used. Kashani and Sherman\textsuperscript{19}, in a study of preschoolers drawn from general population, reported a prevalence of 0.3 per cent. A point prevalence estimate of major depressive disorder is less than 1 per cent in pre-pubertal children (Costello \textit{et al})\textsuperscript{20}. Choudhury \textit{et al}\textsuperscript{21} studied 813 cases registered with child and adolescent psychiatry services in a tertiary care centre in Bangalore, between January and December 1992 and followed them up till October 1993. There were 48 cases (5.9\%) of manic depressive psychosis (MDP) of which eight patients had unipolar depression and 3 had MDP (circular) depression till the end of the study. Malhotra \textit{et al}\textsuperscript{22} in a study of children between 7-14 yr age attending child and adolescent psychiatry clinic at Chandigarh over a period of 6 yr (1991-1996) observed that of the 1600 patients, 33 had a diagnosis of affective disorder (2\%), of which 23 had a diagnosis of unipolar depression and 1 bipolar depression. In comparison to the previous study by the same researcher by retrospective screening of records for the period 1984-1988, it was observed that clinic prevalence rate was 1.2 per cent\textsuperscript{23}. In another study of 963 school children aged 4-11 yr in Chandigarh using multi-stage and multi-informant assessment procedures prevalence rate of psychiatric disorders was found to be 6.33 per cent, of which there were 12 cases of emotional disorder and 5 cases of stress related and somatoform disorder\textsuperscript{24}. In a recent epidemiological study of child and adolescent disorders undertaken in Bangalore by Srinath \textit{et al}\textsuperscript{25} which included 2064 children aged between 0-16 yr, the prevalence rate was noted to be 0.5 per cent (2 cases out of 1578 cases between 4-16 yr). Both the studies highlight that community prevalence is low compared to prevalence in samples presenting to child clinics. In a review of literature by Birmaher\textsuperscript{26}, the prevalence rates of depression in children have been reported to be in the range of 0.4 to 2.5 per cent. There are only a few epidemiological studies on dysthymic disorder which have reported a point prevalence rate from 0.6 per cent to 1.7 per cent\textsuperscript{26}. Studies conducted in specialized paediatric populations have revealed 28 per cent of the patients in child psychiatric clinics\textsuperscript{27} and, 59 per cent of child psychiatric inpatients\textsuperscript{28} as depressed. There are no community studies on adjustment disorder in children.

\textbf{Gender differences:} In children, major depressive disorder occurs at the same rate in girls and boys where as in adolescence the ratio is 2:1 as in adults\textsuperscript{29-31}. A prospective, 10-yr longitudinal study of preadolescent into young adults revealed critical time for gender difference to emerge was between 15 to 18 yr\textsuperscript{33}.

\textbf{Socio-economic status:} According to Costello\textsuperscript{33} and Bird \textit{et al}\textsuperscript{34} low socio-economic status was not associated significantly with depression but Gilman \textit{et al}\textsuperscript{35} reported that participants from lower socio-economic backgrounds had nearly a two-fold increase in life time risk for major depression compared to those from the highest socio-economic background independent of childhood socio-demographic factors, family history of mental illness, and adult socio-economic status. Children in disadvantaged situations may acquire less control over their environment (i.e.,
learned helplessness) and may develop difficulties in forming intimate relationships (attachments); both of these factors may increase children’s vulnerability to depression throughout the life course. In addition, individuals from disadvantaged backgrounds may be more likely to experience stressful life events and be less capable of coping with such events when they occur. Other potential mediators of this association include family disruption, strained social relationships, and poor physical health, each of which is related to depression.

**Co-morbidity:** Clinical and epidemiological studies in depression have shown that 40-70 per cent of depressed children and adolescent have co-morbid psychiatric disorder and at least 20-50 per cent have two or more co-morbid diagnosis.

The most frequent co-morbid diagnoses are dysthymic disorder and anxiety disorder (both between 30-80%), substance abuse (20-30%), and disruptive disorder (10-20%). Except for substance abuse, major depressive disorder is more likely to occur after the onset of other psychiatric disorders. Co-morbid conditions appear to influence the risk for recurrent depression, duration of the episode, suicide attempt or behaviours, functional outcome, response to treatment. Depressed patients with co-morbid disruptive disorder tend to have worse short term outcome, fewer melancholic symptoms, fewer recurrences, a lower familial aggregation of mood disorders, a higher incidence of adult criminality, more suicide attempts and a higher response to placebo.

**Clinical characteristics**

**Onset:** Retrospective studies reveal prevalence of depression extremely low up to 9 yr of age and rising sharply from 9 to 19 yr, especially in females. Age of onset of initial depression is shown to be inversely related to the degree of familial loading.

The age of onset of mood disorders decreased over the years and the incidence rates among youth has increased which is most evident in mild-moderate depression, a phenomenon called ‘cohort effect’. The mechanism of cohort effect is not clear but it is proposed that genetic anticipation in successive generations is responsible.

**Natural course:** Clinical and epidemiological studies reveal that mean length of an episode is approximately 7 to 9 months which is similar to adult depression. Approximately 90 per cent of major depressive disorder have remitted by 1.5 to 2 yr after onset with 6 to 10 per cent becoming protracted. Recurrence of major depression has been estimated to be 40 per cent by 2 yr and 70 per cent by 5 yr in children. These results are identical with the 70 per cent recurrence rates in adult unipolar depressives followed over 5 yr period. Risk of developing bipolar disorder has been found to be 20 to 40 per cent in adolescents within 5 yr of onset of major depression. Clinical characteristics associated with an increased risk of developing bipolar disorder include early onset depression, psychomotor retardation, psychotic features, family history of bipolar disorder, pharmacologically induced hypomania. Early onset dysthymic disorder has a prolonged course with high risk of subsequent major depression (70%), bipolar disorder (13%), and substance abuse (15%).

**Phenomenology:** The characteristics of childhood depression vary across the developmental stages, but these are similar to adult depression. The clinical picture of depression contains features that are associated with mood changes. Older the children, the more similar are the symptoms to those of depressed adult. Unlike adults, however depressed children are less likely to make serious suicidal attempts, but are more likely to demonstrate symptoms of depressed appearance, anxiety (*i.e.*, separation anxiety), irritability, frustration, tantrums, apathy and disinterest, lack of co-operation, withdrawal from family and friends, physical complaints (headache, stomach ache), and auditory “hallucinations”. According to David Shaffer in 90 per cent of the cases, the episode can be dated back to a precipitating event. The symptoms in more than two-thirds of the
cases include tearfulness, brooding about a past experience, being irritable, feeling pessimistic, having difficulty concentrating, feeling worthless, and fatigue. Weight and appetite changes affect in three-fourths, with anorexia and weight loss being considerably more common than overeating and weight gain. Insomnia affects four-fifths, mostly difficultly in falling asleep, early morning awakening is less common. Certain symptoms change with age such as diurnal variation, expression of depressive cognitions increase with age whereas other symptoms either decrease or show no change in its presentation (Fig.).

**Sequelae:** During the episode of depression, children frequently experience impairment in school performance and relationship with others. Factors like co-morbid psychiatric disorders, poor family functioning, low socio-economic status and exposure to stressful life events impact the psychosocial functioning of the patient. Increased risk of suicidal behaviour, abuse of substances are also noted. Prospective studies have reported that after recovery, patients continue to show sub-clinical symptoms of depression, negative attributions, impairment in interpersonal relationships and global functioning, increased substance abuse, increased teenage pregnancy.

**Aetiology:** Aetiology of any psychiatric disorder is multi-factorial, resulting from interactions between various factors like biological, psychological and social factors. In depression no particular factor has
been observed to play a role of primacy rather all these factors interact differently in different individuals leading to manifestations of depression. Understanding aetiology not only helps to elucidate pathogenic mechanism but also has treatment implications. Present knowledge though limited gives us some insight into the fact that aetiological factors implicated are similar to those of adult depression.

**Genetic influence:** Studies on adult depressives have revealed a genetic component in aetiology of depression. Family studies have suggested a high risk of psychiatric disorders in relatives of probands as well as in children of depressive parents.63

**Family aggregation studies:** Family studies of children of depressed parents revealed that such children were three times more likely to have lifetime episode of major depressive disorder than the children of normal parents. The lifetime risk for major depression in children of depressed patients has been estimated to range 15 to 45 per cent.64,65 The risk is more in cases where both parents had mood disorder associated with early onset and recurrences.66 Age adjusted lifetime prevalence rates of depression in the first degree relatives of depressed children and adolescent have been estimated to range from 20 to 46 per cent. The risk seems to be greatest in early-onset (<20 yr).45,49 Studies on twins have found that concordance for affective disorders in monozygotic twins was 76 per cent compared with 19 per cent in dizygotic twins.3 When monozygotic twins were reared apart, the concordance rate dropped to 67 per cent. This discrepancy has been interpreted as supporting a passive gene environment correlate, that is, that a depressed parent, as a consequence of his or her genetic make up, establishes a environment that is depressogenic causing his or her children to present with such manifestation.58

**Molecular genetic studies:** Serotonin system is known to be involved in the pathophysiology of affective disorders.67 The serotonin transporter has received particular attention because of its involvement in the re-uptake of serotonin at brain synapses, and is the target of selective serotonin reuptake inhibitors. The promoter activity of the 5-HTT gene is regulated by a deletion–insertion polymorphism in the proximal 5' promoter region, designated the 5HTT gene-linked polymorphic region (5-HTTLPR). The short (S) allele in the 5-HTTLPR is associated with lower transcriptional efficiency of the promoter compared with the long (L) allele. Only one-third of the population is homozygous for a long allele of the gene. The remainder has one long and one short allele (approximately 50%) or two short alleles (17%). Nobile M et al studied the above association in children and observed an excess of the SS-genotype and of the S-allele among depressed children. The family-based results suggested that the S-allele was preferentially transmitted to depressed children.

**Psychological influence**

**Family environment:** Studies on depressed adults, offsprings of depressed parents, and depressed youths have shown that their family interactions were characterized by more conflict, more rejection, less expression of affect, more problems with communication, more abuse, less support.69 Parental affective illness account for increased rates of psychopathology in children of these parents by way of genetic predisposition, maladaptive parent-child interactions, and marital conflicts and chronicity and severity of parental illness.71 Specifically, maternal depression has been observed to be significantly associated with depression in adolescents.

**Stressful life events:** Studies of both clinical and community samples of depressed children and adolescents have shown a modest but significant relationship between stressful life events (bereavement, family disruption) and depression.72 Maltreated children (includes physical and sexual abuse) are at a significant risk for the development of a number of problems, including insecure attachment, poor emotional and behavioural self-regulatory skills, lowered cognitive functioning,
poorer adaptation to school, and language delays. Depression, viewed as a manifestation of disturbances in self-regulation, is shown to be associated with various types of maltreatment.

Temperament: Prospective investigations have concluded that dimensions of temperament may have a predictive specificity for later psychopathology. It has been observed that infants who demonstrate a low threshold to become distressed and aroused when confronted with unfamiliar stimuli (i.e., those with behavioural inhibition) are more likely than others to become fearful and subdued during early childhood. Merikangas et al. observed that across the age-span anxiety and depression were associated with low scores on adaptability, approach/withdrawal linking behavioural inhibitions with internalizing disorders. Elovainio et al. in a prospective epidemiological study observed that an increased risk to development of depression was related to Cloninger’s temperament dimensions of shyness with strangers, sentimentality, and persistence. Persistence has been linked to enthusiasm, industriousness, and viable coping skills. However, highly persistent individuals are likely to push themselves far beyond what is necessary, an attribute contributing to the risk of depression.

Biological influence

Neuroanatomy: Steingard et al. in a retrospective chart review study of depressed (dysthymia, major depression) hospitalized children (3-17 yr) over a 5 yr period who had undergone MRI (Magnetic resonance imaging) compared with psychiatrically non-depressed patients, observed a significantly larger ratio of lateral ventricular volume: total cerebral volume and a smaller ratio of frontal lobe volume: total cerebral volume. The findings correlated with those in adult.

Nolan et al. studied 22 psychotropic naïve patients with depression aged 9-17 yr; of whom 9 had a diagnosis of only depression, others had comorbid disorders, 12 patients had at least one first degree relative with major depression, and found that patients with non-familial major depression had significantly larger left-sided pre-frontal cortical volume compared with patients with familial depression in contrast to decreased pre-frontal gray matter volumes in adult patients with familial major depression. This is perhaps explained by left sided prefrontal degeneration with progressive illness in terms of recurrence, increased severity, and duration of illness.

The anterior cingulate cortex has been implicated in the pathogenesis of major depression. Structurally, both postmortem and in vivo MRI studies in adult patients with major depression, including patients with childhood onset depression, have shown reduction in volume of the subgenual region of the anterior cingulated cortex.

White matter hyperintensities (WMH) defined as hyperintense signals on T₂-weighted MRI, classified as peri-ventricular hyperintensities (PVH) and deep white matter intensities (DWMH) had been shown to be associated with both unipolar and bipolar disorder. Ehrlich et al. studied 153 inpatients aged 6-21 yr with history of suicide attempt; of whom 48 subjects had unipolar depression but WMH failed to be a marker to differentiate between ideators and non-suicidal subjects. Amygdala and its related structures have been implicated in the pathophysiological characteristics of adult depression. Thomas et al. studied two groups of children aged 8-16 yr, one group with a diagnosis of anxiety disorder (generalised anxiety disorder and/panic) and the other group with major depression. Both the groups underwent fMRI while viewing successive photographs of fearful and neutral facial expressions. Patients with depression showed a blunted amygdala response (decreased blood oxygen level dependent signal) to these facies whereas an exaggerated response was seen in patients with anxiety disorder. There have also been reports of abnormal amygdala and hippocampal volumes in adult major depression. MacMillan et al. in their study of amygdala and hippocampal volumes observed, increases in
amygdala volumes that did not reach significance levels and no change in hippocampal volume however, MacMaster and Kusumakar observed reduced hippocampal volumes in adolescent depression. Rosso et al studied 20 children and adolescents and observed a significant reduction in both right and left amygdalae without any change in size of hippocampus.

Bonte et al studied 21 children and adolescents with major depression, aged 11-18 yr with single photon emission computed tomography-using 99m Tc-hexamethyl propylene-amine-oxime (SPECT HMPAO), divided into two groups, one group showed significant deficits in occipital lobes (Broadmann’s areas 18,19), whereas the other group showed significant bilateral frontal and prefrontal deficits.

Proton magnetic resonance spectroscopy (H-MRS) allows for direct, in vivo, and noninvasive measurement of brain chemicals like choline, N-acetyl aspartate(NAA) and creatine/phosphocreatine ratio. Farchione et al studied 11 psychotropic-naive depressed children and adolescent (11-16 yr), and observed a significant increase in concentration of choline in left dorsolateral prefrontal cortex similar to another study by Steingard et al which showed an increased left orbito-frontal choline concentrations in adolescents with major depression. Both studies signify the importance of prefrontal cortex in neurobiology of depression. Using H-MRS, Pfeiderer et al reported reduced anterior cingulate Glx (glutamine and glutamate) in severely depressed adults which was in contrast to increased levels in schizophrenia or frontal Glx levels in bipolar patients. Mirza et al studied 13 psychotropic-naive depressed children and adolescent with five having sole diagnosis of major depression. Each subject underwent H-MRS with short echo single voxel double spin-echo-point-resolved spectroscopy (PRESS) pulse sequence. A significant decrease in Glx was observed in anterior cingulated cortex but failed to show the same in the occipital cortex.

Serotonergic studies: Dysregulation of serotonin system in adults have been attributed as a vulnerability factor but data in children are not robust enough to conclude the same; studies using serotonergic probes have reported findings opposite to that in adults. Serotonergic precursors elicit augmented response in children in contrast to blunted response in adults. It is postulated that dysregulation might be the underlying phenomenon but the nature varies across life cycle.

Electroencephalography: Certain characteristic findings have been repeatedly observed in depressed adults, like prolonged sleep latency, sleep continuity disturbances, decreased time to first rapid eye movement (REM) period, increased REM density, and decreased delta stage (stage 3 and 4). These findings are more consistent in depressed adolescents than in children.

Hypothalamic-pituitary axis

Dexamethasone suppression test (DST): Elevated glucocorticoid activity is a hallmark of mammalian stress response. When blood levels of glucocorticoids are increased in normal individuals, less adrenocorticotropin hormone (ACTH) is released from anterior pituitary and less steroids is produced by adrenal glands. The integrity of this feed-back mechanism is tested by the dexamethasone suppression test. In a review of literature by Birmaher, following observations were made. (i) an estimated 50 to 70 per cent of the depressed children were considered to be DST non-suppressor, more or less comparable with rates reported in adult samples; and (ii) higher rates of non-suppression are noted in patients with endogenous and psychotic symptoms, prior history of major depressive disorder and family loading of depression.

Growth hormone (GH) studies: Altered GH response to pharmacological challenge is a psychobiological correlate of depression. A variety of agents has been used to challenge the GH response, e.g., clonidine, insulin. These agents act through hypothalamic
postsynaptic $\alpha_2$ receptors, indirectly stimulating the release of GH in pituitary, by causing release of growth hormone-releasing hormone. Children like adults with major depression show a blunted response compared to normal subjects\textsuperscript{95,96}.

Management

Certain factors are to be considered in deciding the management of childhood depression, e.g., the age of the patient, developmental stage, contextual issues (family conflict, academic problems), number of prior episodes, chronicity, subtype of depression (psychotic, melancholic, atypical), and co-morbidity. A multimodal treatment approach is considered consisting of psychotherapy and pharmacotherapy. Treatment should continue for a period of 6-12 months following remission due to high rates of relapse and recurrences. Psychotherapies in the form of play therapy, behavioural therapy, supportive therapy, social skills training, group therapy, family therapy and cognitive behavioral therapy (CBT), interpersonal therapy (IPT) (in older children) are useful in short-term management of mild to moderate depression\textsuperscript{97}. Child’s cognitive and emotional development guides the decision regarding the choice of intervention, e.g., play therapy and parental training would likely be the most appropriate for use in depressed preschool-aged children, while cognitive behaviour therapy would be more appropriate in older children and adolescents. Research in children older than 10 yr of age has shown the cognitive behaviour therapy to be an effective intervention with effects lasting even after termination of therapy\textsuperscript{98}. Pharmacological therapy in depressed children has been a matter of controversy due to limited research, the high placebo response, safety issues, side effects, and ethical issues. This placebo response rate is about 50 per cent in studies using tricyclic antidepressants\textsuperscript{99}. Pharmacological management with SSRI’s (selective serotonin re-uptake inhibitors) is the treatment of choice based on trials indicating efficacy in severe depression, however, there is evidence in literature that questions the efficacy and safety of SSRI in treatment of childhood depression due to high placebo response and high rate of suicidality particularly in paroxetine trial\textsuperscript{100}. Fluoxetine has been approved by both Food and Drug Administration (FDA), US and Committee on Safety of Medicines (CSM) (UK) for treatment of paediatric depression, whereas paroxetine has not been approved. Tricyclic antidepressants are not the first-line drugs but patients with co-morbid MDD and attention deficit hyperkinetic disorder (ADHD) might benefit from it\textsuperscript{101}.

Conclusion

Childhood depression is a recurrent, familial, disabling, disorder with significant co-morbidity particularly with anxiety and disruptive disorders, with increased risk of suicide, poor psychosocial outcome, and risk of substance abuse. Phenomenology of childhood depression needs to be understood in its developmental and psychological background which makes it difficult to diagnose and at times being overlooked leading to significant morbidity. This makes assessment of childhood depression a field that needs expertise due to its often myriad presentations, though progress has been made through structured interviews, self-rating questionnaires. Childhood major depression is only a part of constellation of depressive syndromes observed in clinical practice. Till research is done in these presentations, these categories other than major depression remain clinical realities needing attention and treatment in their own right. Clearer diagnostic criteria that have evolved through years of research in major depression have helped towards identifying correlates of onset, course, and recurrence. Progress has been made to understand the neurobiology of childhood onset depression much of which is in line with the neurobiological findings of adult depression. Therefore, childhood depression could well be in continuity with adult depression. Psychosocial and pharmacological treatments are indicated in both acute and long-term management of depression in children. However, the controversy over use of antidepressants would perhaps continue to rage till their efficacy and safety could be proved beyond
doubt by methodologically sound studies. Till then, psychosocial management, informed decision regarding drug therapy in selected cases would be the best possible way of treating childhood depression.

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