# Eating Disorders in Adolescents

*Manjusha Mardikar*

## Introduction

Adolescence marks the transition from childhood to Adulthood. It is a time of dramatic and rapid physical and psychological change. During this period many lifelong health relevant behaviors are established, including dietary habits, exercise patterns, tobacco and alcohol use and sleep patterns. Adolescents are particularly prone to risk taking behaviors. They have a strong desire to establish an identity that is increasingly independent of the family. (1)

## Eating pattern in Adolescence

The unique physical, psychological and social transitions of adolescence provide a context for the development and perpetuation of eating patterns. Many adolescents have a voracious appetite in response to the increased energy and caloric requirements generated by the growth spurt. Control over dietary intake is perhaps one of the first mechanism that adolescents adopt to establish autonomy and achieve independence from family. (1)

## Eating disorders in adolescents

Eating disorders are syndromes characterized by severe disturbances in eating behaviours and by distress or excessive concern about body shape or weight. (2)

## Historical Perspective

In 1689, Richard Morton published a magnum opus Phthisiologia, seu Exercitationes de phthisi, which is considered the first medical account of anorexia nervosa-a condition that he referred to as "a nervous consumption" caused by "sadness and anxious cares". In his book, which was translated into English in 1694, as Phthisiologia, or a Treatise of Consumption. Morton described anorexia nervosa in two patients. One was a young woman called "Mr. Duke's daughter", who was afflicted at age 18 and suffered for 2 years before seeking Morton's help. The other was a boy, described as "The son of Reverend Minster Steele, my very good friend." This young man became ill at the age 16 and also suffered for about 2 years before seeking Morton's assistance (28).

*Address for correspondence*

**Associate Professor,**
Department of Medicine,
NKPSIIMS,Digdoh, Nagpur.

## Prevalance

Eating disorders affect adolescents with increasing frequency. They rank as the third most common chronic illness in adolescent females (3), with an incidence of upto 5% (4.5), a rate that has increased dramatically over the past three decades. The prevalence of diagnosed eating disorder is increasing, though many adolescents with this disorders remain undiagnosed and untreated. Only 0.25 to 4 % of females fit the diagnostic criteria of the DSM IV for eating disorders (6)

## Gender Prevalence

Eating disorders are more commonly seen in females. Estimates of female to male ratio range from 6:1 to 10:1. However the number of males afflicted with these disorders is significantly increasing (2). This was shown by a 10 year study led by Y. May Chao of Wesleyan University in Middletown, CT, done from 1995 to 2005 (7).

## Ethnic Considerations

While white female adolescents are the most

likely to practice weight control, white males are least likely to practice it. In contrast Hispanic adolescents are the most likely, due to higher prevalence of overweight amongst them. Black female adolescents have moiré flexible concepts of beauty, which make them less vulnerable to social pressure for weight reduction. However, this puts them at increased risk for becoming overweight (7).

**Cultural Considerations**

Eating disorders are more common in industrialized societies where there is an abundance of food and being thin, especially for women, is considered attractive. They are most common in United States, Canada, Europe, Australia, New Zealand and South Africa. However, rates are increasing in Asia., especially in Japan and China, where women are exposed to cultural change and modernization (7)

Prevalence in India is lower than that of western countries but appears to be increasing. The only study over 6 year period led by P Mammen, S. Russell at al, from the Child and Adolescent Psychiatry Unit, CMC, Vellore in 2007 reported the prevalence of eating disorders to be 1.25% in Indian population.

**Classification**

The major subgroups of the eating disorders recognized are : Anorexia Nervosa, Bulimia Nervosa and Eating Disorders Not Otherwise Specified (ED NOS). Anorexia Nervosa is a restrictive form in which food intake is severely restricted. Bulimia nervosa is a bulimic form in which binge eating episodes are followed by attempts to minimize the effects of overeating via vomiting, catharsis, exercise or fasting.

Uncounted teenagers preparing to be models, entertainers, dancers, gymnasts, jockeys and other athletes who manipulate their weight, also suffer from longterm effects of chronic malnutrition, whether they do or do not meet the criteria for anorexia nervosa or bulimia nervosa. They may be categorized as having an 'Eating disorder not otherwise specified (ED NOS) (8).

**Diagnostic Criteria**

Diagnostic criteria for eating disorders such as described in DSM-IV may not be entirely applicable to adolescents. In clinical practice, the diagnosis of an eating disorder should be considered in an adolescent patient who engages in potentially unhealthy weight control practices and /or demonstrates obsessive thinking about food, weight, shape or exercise and not only in one, who meets established diagnostic criteria.(a).

Criteria of the Diagnostic and statistical Manual of Mental Disorders, fourth edition, text revision (DSM IV-TR1) (2) American Psychiatry association 2000.

**Anorexia Nervosa**

a) Refusal to maintain body weight at or above a minimally normal weight for age and height : weight loss leading to maintenance of body weight < 85% of that expected or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected.

b) Intense fear of gaining weight or becoming fat, even though underweight.

c) Disturbance in the way one's body weight or shape are experienced, undue influence of body weight or shape on self evaluation, or denial of the seriousness of the current low body weight.

d) A menorrhea (at least three consecutive cycles) in postmenarchal girls and women.

**Restriciting Type:** During the episode of Anorexia nervosa, the person has not regularly engaged in binge eating or purging behaviour.

**Purging Type:** During the episode of Anorexia nervosa, the person has regularly engaged in binge eating or purging behavior

**Bulimia Nervosa**

1) Recurrent episodes of binge eating characterized by both:

a) Eating, in a discrete period of time (e.g. within any 2-hour period), an amount of food at is definitely larger than most people would eat during a similar period of time and under similar circumstances.

b) A sense of lack of control over eating during the episode defined by a feeding that one cannot stop eating or control what or how much one is eating.

2) Recurrent inappropriate compensatory behaviour to prevent weight gain such as self induced vomiting, misuses of laxatives, diuretics, enemas, or other medications, fasting or excessive exercise.

3) The binge eating and inappropriate compensatory behaviour both occur, on average, at least twice a week for 3 months.

4) Self evaluation is unduly influenced by body shape and weight.

5) The disturbance does not occur exclusively during episodes if anorexia nervosa.

**Purging Type :** During the current episode person has regularly engaged in self induced vomiting or misuse of laxatives, diuretics or enemas.

**Non Purging Type :** During the current episode person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise.

**Eating disorders not otherwise specified**

Eating disorders not otherwise specified includes disorders of eating that do not meet the criteria or any specific eating disorder.

a) For female patients all of the criteria for anorexia nervosa are met except that the patient has regularly menses.

b) All of the criteria for bulimia nervosa are met except that the binge eating and inappropriate compensatory mechanisms occur less than twice a week or for less than 3 months.

c) All of the criteria for anorexia nervosa are met except that, despite significant weight loss, the patient's current weight is in normal range.

d) The patient has normal body weight and regularly uses inappropriate compensatory behaviour after eating small amounts of food.

e) Repeatedly chewing and spitting out, but not swallowing, large amount of food.

**Etiology**

Eating disorders are said to have multifactorial etiology. It is complex and appears to include predisposing genetic factors, serotonin dysregulation, as well as psychological factors that include a history of physical and mental trauma and childhood sexual abuse. (6)

**Pathophysiology**

Biologic and Psychosocial factors are implicated in the pathophysiology of eating disorders. Environmental and Social factors in adolescents who are biologically vulnerable and have a psychological predisposition are probably responsible for the eating disorders in them. Recent familial aggregation studies are renewing interest in the contribution of genetic predisposition (9).

**Biological Vulnerability**

The prominent physiological disturbances in anorexia nervosa led to speculation that the abnormal behaviours are caused by a primary biological abnormality. Disruptions of the pituitary, hypothalamus and various neurotransmitters have been postulated to be casual factors in the development of anorexia nervosa. The neurotransmitter serotonin is known to affect appetite control, sexual and

social behaviour, stress responses and mood. It modulates feeding by producing the sensation of satiety or fullness. Serotonin antagonists increase food consumption and promote weight gain. Decease in brain serotonin function are associated with depression, impulsivity and aggressive behaviour. The major serotonin metabolite, 5 hydroxyindoleacetic acid is low in adolescents who are underweight with anorexia nervosa, but then rises to above normal levels in those who have made long standing recoveries. (10,11). One study showed that patients with more severe binge eating have lower cerebrospinal fluid s-hydroxyindoleacetic acid than do controls (12).

Leptin, a hormone secreted by fat cells seems less likely to play an important role in anorexia nervosa. Adolescents with anorexia nervosa or low weight have low serum leptin, which increases with weight gain.

**Psychological predisposition**

There is an increasing association between major depression and anorexia nervosa, with depression a risk factor for development of incident eating disorders in adolescence (13). Body image distortion is a core characteristic of eating disorder in adolescents. There is unrealistic perception of body size and there are unrealistic expectations of oneself. The rapidly of physical change occurring during development contributes to the difficulty of the task of acceptance. Adapting a mental image of one's body image, which is a basic feature of development, is not accomplished in these disorders (8).

**Sociocultural and Environmental Factors**

These factors as they relate to ideal body shape are thought to play an important role in the development of eating disorders in adolescents. They are more common in industrialized nations where food is plentiful and where thinness for women id correlated with attractiveness. (9)

**Genetic Predisposition**

Familial transmission of risk has emerged as an increasing focus of research attention.

**Clinical Features**

They are varied and complex. Semisatrvation experienced in these disorders in the setting of rapid physical growth and development accounts for the vulnerability of any adolescent to longterm consequences. They are subject to greatest harm from food deprivation at periods when they are synthesizing tissue. They need nutrients to build tissues and food energy to fuel the process (8). With insufficient energy available, tissue maintenance and synthesis cannot occur,. The most commonly affected system abnormalities are as follows-

**Electrolyte abnormalities-**

Because of fluid loss and fluid restriction the most serious and documented abnormality is hypokalemia, which can result in cardiac symptoms, specifically arrhythmias and ECG abnormalities. When chronic it can lead to constipation, skeletal muscle myopathy and nephropathy (14) Low magnesium is also common which can lead to muscular weakness, diminished concentration, muscle cramps, paraesthesias, arrhythmias, and recent memory loss (15). Low phosphate levels correlate with osteoporosis and high incidence of scoliosis (16).

**Cardio Vascular effects-**

These occur frequently due to electrolyte abnormalities. Bradycardia, hypotension, orthostatic hypotension and symptoms of dizziness and fainting occur as a result of starvation and dehydration (17). Ventricular tachycardias are a well accepted cause of death in eating disorders.

**Gastrointestinal effects –**

These can be life threatening, swollen Salivary glands, Eosophagitis. Eosophageal spasm and tearing and potential fatal ruptures can occur from constant vomiting. Gastric dilatation and Acute pancreatitis can

occur due to binge eating (17).

**Endocrine imbalances-cortisol-**

Sustained elevation of cortisol along with proportionately decreased levels of DHEA and androstenedionc can result in depression (18).

Thyroid Function-Normal T4, normal TSH and low T3 are the most common laboratory findings. These findings correlate with symptoms of fatigue, hypothermia, constipation, bradycardia and hypercholesterolemia (19).

**Gonadal Hormones –**

Amenorrhea is a classical feature of anorexia nervosa. Bulimics are also at risk of HPG dysfunction and may experience irregular menses (17)

Over 50% of all patients with anorexia nervosa have evidence of significant osteoporosis (20).

**Blood Sugar Metabolism and Diabetes Mellitus-**

Fasting and postprandial glucose levels are low in eating disorders. The incidence of eating disorders in young females with Type-1 Diabetes Mellitus is significantly higher than in nondiabetic young females. Diabetics with eating disorder have a three fold increased risk for diabetic retinopathy (21).

**Nutrient Deficiencies- Zinc**

A significant amount of research has been attempted to evaluate the role of zinc in eating disorders. Several signs of zinc deficiency, including weight loss, appetite loss, dermatosis, amenorrhea and depression are commonly seen in anorexia nervosa (22).

Zinc deficiency also reduces leptin concentrations, resulting in underweight (23)

**Protein Energy Malnutrition-**

Also exists in patients with eating disorders. Other vitamin deficiencies namely Thiamine and Riboflavin is also documented in many studies.

**Psychosocial disturbances-**

Eating disorders in adolescents interfere with adjustment to pubertal development (24) disorders of anxiety, depression, dissociation and behaviour are very common (25).

**Mortality**

Anorexia nervosa has highest mortality of any psychiatric diagnosis estimated at 10% occurring within 10 years of diagnosis (26). Mortality of Bulimia nervosa is expectedly less, approximately 1% occurring within 10 years of diagnosis (27)

**Treatment**

Since the incidence of eating disorders is rising among adolescents, the necessity for comprehensive treatment is vital. The goals of the treatment are to help the adolescent achieve and maintain both physical and psychological health. Because of the complex biopsychosocial aspects of eating disorders in adolescents, the assessments and ongoing management of these conditions appear to be optimal with an interdisciplinary team consisting of professionals from medical, nursing, nutritional and mental health disciplines (25).

Both inpatient and outpatient treatments need to be available to adolescents with eating disorders.

**Indications for in patient care-**

- Significant malnutrition
- Physiological or physical evidence of medical compromise.
- Arrested grown and development.
- Failure out patient treatment
- Acute food refusal
- Uncontrollable binging, vomiting or purging
- Family dysfunction that prevents effective treatment

- Acute medical or psychiatric emergencies (30)

Optimal duration of hospitalization has not been established. These criteria are in agreement with the recent revision of the American Psychiatric Association practice guidelines. In hospital gradual increase of caloric intake and close monitoring of weight, vital signs, fluid shifts and serum electrolytes is done. Parenteral feeding is very rarely necessary. There is no evidence to support the longterm role of nasogasrtic tube feeding (31).

The expertise of the treatment team who work specifically with adolescents and their families is as important as the setting in which they work. Smooth transition from inpatient to outpatient care can be facilitated by an interdisciplinary team that provides continuity of care in a comprehensive, co-ordinated, developmentally oriented manner. Treatment should be of sufficient frequency, intensity and duration to provide effective intervention to prevent relapse, recurrence, crossover (from anorexia to bulimia and vice versa) and comorbidity. (31)

Mental health evaluation and treatment is crucial for all adolescents with eating disorders. Treatment should be family based. Cognitive behavioural therapy has not been evaluated in adolescents with anorexia nervosa, but it is most effective in bulimic patients. This therapy focuses on changing the specific eating attitudes and behaviours that maintain the eating disorders (32).

Antidepressants have been shown to reduce big eating or purging (33). Interpersonal psychotherapy (34) and dialectal behaviour therapy have also been demonstrated some beneficial effect in older adolescents with bulimia nervosa. The optimum treatment of the osteopenia associated with anorexia nervosa remains unresolved. Current recommendations include weight restoration with the initiation or resumption of menses, calcium (1300-1500 mg/day) and Vit D(400 IU/day) supplementation and carefully monitored weight-bearing exercise (35,36). Patient should be followed on longterm basis to prevent recurrence and relapse.

**References :**


1. Timothy Walsh;Eating Disorders Harrison's Principles of Internal medicine 2005;Mc Graw Hill Publication; New York, USA 16th Edition, Vol I, Chapter 65; 430 -433.
2. Kathleen N Franco: Eating Disorders : Cleveland Clinic, centre for continuing education. Publications: disease management Project-Jan, 1,2009.
3. Whitaker AH. An epidemiological study of anorectic and bulimic symptoms in adolescent girls: implications for pediatricians. Pediatr Ann 1992;21:752-9
4. Stein DM. The prevalence of bulimia: A review of the empirical research.J Nutr Edue 1991;23:205-13
5. Drewmowski A, Hopkins SA, Kessler RL. The Prevalence of bulimia nervosa in the US college student population. Am J public Health 1988;78:1322-5
6. Eating disorders: a review of the literature with emphasis in medical complications and clinical nutrition-eating disorders, alternative medicine. Review, June 2002 by Lyn Patrich.
7. Y. May Chao. Emity M. C talk: " ethnic differences in weight control practices among US adolescents from 1995-2006. internal Journal of eating disorders.
8. Jane Mitchbell Rees PhD, RD, CD departments of Health Sciences and Paediatrics Maternal Child Health and Adolescent medicaine, university of Washington, sealthe, WA 98/95-early disorders during adolescences : Nutritional problems and interventions.

9. Ellen S Rome, Seth Ammerman, David S Rosen CL al. Children and Adolescents with Eating disorders: the state of the Art PAEDIATRICS Vol. III No. I January 2003 pp e98-e108

10. Jimmerson DC. Lesem MD etal : eating disorders and depression: is there a serotonin connection ? Biol psychiatry. 1990:28:443-454 (cross Rcf) (Web & Science) (medicine).

11. Kaye WH, Weltzin T. Hsu 4K6, ct,al, An open trial of fluoxetive in patients with anorexia nervosa. J. Clinical Psychiatary. 1991;52:464-471 (Web of science) (Medicine).

12. Jimmersen DC. Lesem MD, Kaye WH, Brewerten TD. Low serotonin and Dopamin metabolic concentrations in cerebral spinal fluid from bulimic patients with frequent binge episodes. Arch gen psychiatry, 1992, 49:132-138.

13. Patton GC, Selzer R, Coffet C et al. Onset of eating disorders : Population based cohort over 3 years. BMJ.1999; 318:765-768.

14. Mitchell JE, Pyle RI, et all-Electrolyte and other physiological abnormalities in patients with bulimia. Psychol med 1983; 13:273-278.

15. Hall RC, Hoffman RS, Beresford TP, et all, Hypomagnesemia in inpatients with eating disorders. Psychosomatics 1988:29: 264-272.

16. Crosby Lo, Kaplan FS et al, The effect of an on bone morphometry in young women. Clin orthop 1985 : Dec: 271-279.

17. Pmeroy C, Mithell J. Medical complications of an and Bn, In: Fairburn CG, eds. Eating disorders and obesing 2nd ed. New york, Ny-Gullford Press:2002:278-285.

18. Winterer J. Gwirtsman HE. George DT et al. Adrenocorticotropin stimulated adrenal androgen secretion in an : impaired secretion at low weight with normalization after longterm wt. recovery. J Clin endocranial. Metab 1989:68:517-522.

19. Bannai G, Kuzu N, Koide Y, et, al. Assessment of the relationship between serum thyroid hormone levels and peripheral metabolism in patients with an Endocrinal Jpn 1988:35:455-462.

20. Grirspoon S, Herzog D, Kilbansti A. Mechanism and treatment options for bone loss in an psychopharmacol Bull 1997:33: 399-404.

21. Rydall AC. Rodin GM, Olmsted MP et al. Disordered eating behaviour and microvascular complications in young women with IDDM-NEJM 1997;336:1849-1854

22. Bankan R. The role of zinc in an: etiology and treatment. Med Hypothesis 1979:5:731-736.

23. Mantzoros Cs. Prasad AS, Beck Flo-et al. Zinc may regulate serum Leptin Concentrations in humans. J AM Coll Nutr 1998; 17:270-275.

24. Killen JDS, Hayward C, Litt I, et al. Is puberty a risk factor for eating disorders ? Am Dis Child 1992; 146:323-5.

25. Comrci GD, Eating disorders in adolescents. Pediatr Rev 1988;10;1-11.

26. Sullivan P. course and outcome of an & Bn. In: Fairburn GC, Bownell KD eds. Eating disorders and obesity 2nd edition, New York NW; Guilford Pres:2002:226-232.

27. Sullivan PF. Mortality in AN Am J Psychiatry 1995;152;1073-74.

28. Joseph A Silverman. Anorexia

Nervosa: Historical perspective on treatment. Chapter I, Section I, Page 3, Handbook of Treatment for eating disorders 2nd edition. Edited by David Garner Paul Garfinkel 1997.

29. Arnold E. Anderson, Wayna Bowers and Kay Evans Chapter17, Section IV, Page 327-354 Handbook of treatment for eating disorders 2nd edition. Edited by David Garner. N Y Guilford Press 1997.
30. Kreipe R E, Uphoff M. Treatment and outcome of adolescents with anorexia nervosa. Adolesc Med State Art Rev 1992; 3:519-540.
31. Neville H Golden, Debra Kartzman. Richard Kreipe at all. Eating disorders in adolescents: position paper of the society of adolescent medicine. Health Vol 33, Issue 6, Pages 496-503 December 2003.
32. Fairburn C. A Cognitive behavioural approach to the treatment of bulimia. Psychol Med 1981;11;707-711.
33. Fluoxetive in the treatment of bulimia nervosa. A multicentre, placebo-controlled, double-blind trial. Fluoxetine Bulimia Nervosa Collaborative study group. Arch. Gen. Psychiatry 1992;46:139-147.
34. Fairburn CG. Interpersonal psychotherapy for bulimia nervosa. In: Garner DM. editor. Handbook of Treatment for eating disorders. New York Guilford press:1997. P-278-294.
35. Standing committee on the scientific evaluation of dietary reference Intakes for Calcium PMVDaf. Washington DC: Natimal Academy Press:1997.
36. Golden NH Osteopenia and Osteoporosis in anorexia nervosa. Asdolesce Med 2003; 14: 97-108. Medline.