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Research Article

RETHINKING BULIMIA NERVOSA : FROM SYMPTOM TO SOLUTION

Tanmayee S. Pakhale
Sandip Institute of Pharmaceutical Sciences

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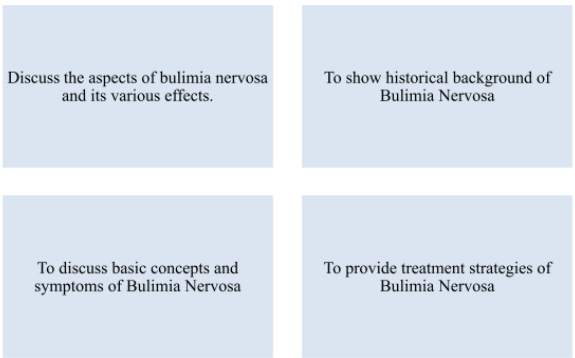
ABSTRACT

The study's main goal is to address any nutritional concerns and change in behavior. Therapy looks at how your thoughts, feelings, and behaviors relate to one another. Bulimia nervosa is an eating disorder that typically begins in adolescence and peaks at the age of 18. It is thought to be a result of anorexia nervosa and is characterized by purging and binge eating. Additionally, to examine the DSM V-based diagnosis of bulimia nervosa. This study employed a qualitative methodology using a library research approach. Thus, bulimia nervosa patients require extensive medical and psychological care, including individual and family therapy as well as hospitalization if required. This page also provides an overview and treatment of bulimia nervosa.[1]

* **Corresponding Author- Tanmayee S. Pakhale,** pakhaletanmayee@gmail.com Sandip Institute of Pharmaceutical Sciences

OBJECTIVE OF STUDY

The principle objective of this study is to:



INTRODUCTION

Eating disorders are psychiatric illnesses that have serious physical and psychosocial repercussions. Bulimia nervosa is characterized by irregular episodes of uncontrollably excessive eating (binge eating), coupled with drastic measures to counteract the feared effects of the overeating, and an intense fear of gaining weight or refusing to maintain body weight at or above average body weight, at least according to age and height. Selective serotonin reuptake medications, such as citalopram, fluoxetine, and sertraline, have been shown to reduce the symptoms of bulimia nervosa.[2] additionally, three categories of eating disorders are recognized by the Diagnostic and Statistical Manual of Mental Disorders – V (DSM-V): binge-eating disorder (BED), anorexia nervosa (AN), and BN (BN).[1]

EPIDEMIOLOGY

According to community-based studies, the prevalence of bulimia nervosa ranges from 0.1% to 0.4% for men and from 0.3% to 9.4% for women. Women make up about 90% of those with a bulimia nervosa diagnosis. Though the incidence has subsequently plateaued or even decreased, with 6.6 new diagnoses per 100,000 in

2000, the number of people presenting with bulimia nervosa in industrialized nations grew during the decade after its diagnosis in the late 1970s.[3]

ETIOLOGY/ RISK FACTORS

Although the causes of bulimia nervosa are multifaceted, the promotion of dieting and social pressure to be slender appear to raise risk.[3] There are several factors at play. The bingeing behavior linked to this illness may be exacerbated by impairments in interoceptive function, especially of the insula. According to a 2016 study, patients with bulimia nervosa and anorexia nervosa exhibit broad abnormalities with diffuse changes in white matter structural and useful connectivity, especially within taste-reward and appetite-regulating networks. A potential change in the intrinsic functional structure of the brain has been suggested by other research.[4]

SIGNS AND SYMPTOMS

Bulimia nervosa is a diverse syndrome that is characterized by rapid and uncontrollable eating. Unbalanced electrolytes can cause cardiac arrest, irregular heartbeats, and even death.Weight fluctuations are prevalent in BN patients, and hypoglycemia can occur after vomiting, weight loss, or irregular menstruation in girls, while fainting, dizziness, and sleep disturbance are observed in women.They have mental health conditions include anxiety disorders, bipolar disorder, and significant depression.[5]

extensive literature review, no analytical methods are reported for the tablet dosage form. The challenges for the quality control and bioavailability of RTV in generics produced in India inspired the authors to develop simple methods for quantification of RTV in bulk and stability study in UV – Visible Spectrophotometer. The developed methods were validated according to International conference of harmonization (ICH) guidelines [11].

Sign	*Underlying pathophysiology*
Anorexia nervosa	
Amenorrhea	Hypothalamic dysfunction, low fat stores, malnutrition
Arrhythmia	Electrolyte disorders, heart failure, prolonged corrected QT interval
Bradycardia	Heart muscle wasting, associated with arrhythmias and sudden death (common in anorexia nervosa)
Brittle hair and nails	Malnutrition
Edema	Heart muscle wasting, associated with arrhythmias and sudden death (common in anorexia nervosa)
Hyperkeratosis	Malnutrition, vitamin and mineral deficiencies
Hypotension	Malnutrition, dehydration
Hypothermia	Thermoregulatory dysfunction, hypoglycemia, reduced fat tissue
Lanugo (fine, white hairs on the body)	Response to fat loss and hypothermia
Marked weight loss	Self starvation, low caloric intake
Osteoporosis at a young age	Malnutrition
Bulimia nervosa	
Dental enamel erosions and gum disease	Recurrent vomiting washes mouth with acid and stomach enzymes; mineral deficiencies
Edema	Laxative abuse, hypoproteinuria, electrolyte imbalances
Parotid gland enlargement	Gastric acid and enzymes from vomiting cause parotid inflammation
Scars or calluses on fingers or hands (Russell sign [knuckle calluses])	Self-induced vomiting
Weight fluctuations; not underweight	Alternating between bingeing and purging

(Information from references 11 through 12.)

DIAGNOSTIC CRITERIA

Episodes of binge eating:

- Patients are eating portions more significant than what most people would consume in a similar period (usually less than 2 hours) and under comparable conditions.
- During eating episodes, the patient loses control and is unable to curb the servings he consumes.

Binging episodes are followed by inappropriate compensatory behavior to prevent weight gain:

- Self-induced vomiting
- Laxatives abuse
- Diuretic use
- Extreme physical activity
- Fasting

The episodes should occur at least once a week for three months to establish a diagnosis.[4]

DIAGNOSIS BASED ON DSM-IV

Bulimia was initially listed as a diagnostic in the Diagnostic and Statistical Manual of Mental Disorders

(DSM) third edition, which was released in 1980. However, at that time, bulimia was only described as the existence of binge eating behaviors. DSM-IV-TR diagnoses were made during the follow-up period and symptoms were evaluated at intake using a modified version of the Longitudinal Interval Follow-Up Evaluation. Every six months, if at all possible, it was conducted in person by qualified interviewers. Weekly psychiatric status rating scores were obtained with this instrument.

Psychiatric status ratings range from 0 to 6 for anorexia nervosa and bulimia nervosa, Where, 0=no history of the disorder;

1=a past disorder with no current symptoms;

2=residual symptoms (e.g., minor eating disorder cognitions without current behavioral symptoms);

3=partial symptoms (i.e., does not meet full criteria; e.g., for bulimia nervosa, experiences binge eating and/or compensatory behaviors 1–3 times a month with significant cognitive symptoms);

4=marked symptoms (just misses full criteria: e.g., for bulimia nervosa, experiences binge eating and compensatory behaviors 4–7 times a month);

5 and 6=full criteria, depending on symptom severity or degree of impairment; e.g., for bulimia nervosa

5 would indicate binge eating/compensatory behaviors two or more times a week, and

6 would indicate daily binge eating/compensatory behaviors.[6]

DIAGNOSIS BASED ON DSM-V

The frequency of inappropriate weight-compensatory behaviors is the basis for the DSM-V severity criterion for bulimia nervosa (BN). DSM-V severity levels were used to classify 199 community volunteers with BN, and demographic and clinical characteristics were compared. Participants were classified as mild in 77 cases (39 percent), moderate in 68 cases (34 percent), severe in 32 cases (16 percent), and extreme in 22 cases (11%).[7]

DRUG TREATMENT ON BULIMIA NERVOSA

In six- to sixteen-week double-blind controlled trials, some antidepressant medications (tricyclics: imipramine, desipramine, amitriptyline; IMAO: phenelzine, isocarboxazide; trazodone; fluoxetine) seem to work better than a placebo. Similar trials have shown that fluvoxamine and meserine, two additional antidepressants, do not work. The small fraction of patients who are completely abstinent at the end of treatment seems to have poor prognostic value for long-term outcomes, and improvement reports are frequently insufficient.[8]

COGNITIVE BEHAVIOURAL THERAPY

For bulimia nervosa, a particular type of cognitive behavioral therapy (CBT) called CBT-BN involves three over lapping phases in 19 sessions spread over 20 weeks. Although its exact mechanisms of action are unknown, cognitive-behavioral therapy (CBT) is an effective treatment for bulimia nervosa. In order to determine potential mediators of CBT improvement for BN and its time course of action, the authors of this study examined the findings of a randomized control experiment that contrasted CBT with Interpersonal Psychotherapy (IPT). Both binge eating and vomiting improved after therapy when dietary limitation was reduced as early as Week 4.[9]

MEDICAL COMPLICATIONS

1. Oral complications
2. Gastrointestinal complications
3. Electrolyte complications
4. Endocrine complications
5. Other toxic effects.[10]

DISCUSSION AND CONCLUSION

Our review article begins with an overview of bulimia nervosa, including its various causes, epidemiology, and alternate treatments. Our findings demonstrate that medications do work. It comes to the conclusion that bulimia nervosa, or just bulimia, is an eating disorder that is typified by binge eating, or eating a lot of food in a short amount of time, or going out of control, followed by compensatory behaviors like vomiting, over exercising, or fasting to avoid gaining weight.in addition to assessing patients' emotional and physical well-being, counseling-based therapy will produce more accurate data regarding bulimia nervosa and its management. Although the exact origin of bulimia is unknown, there is growing evidence that hereditary factors play a significant influence. The manner and frequency of purging are linked to the medical difficulties of bulimia nervosa, while hunger (restricting) and weight loss are the causes of anorexia nervosa.[1]

Reference:

1. Fatimah Aufatunnisa[1], Endah Ronawulan[2]., 2024. Diagnosis of Bulimia Nervosa Based on Dsm V. International Journal of Social Science and Human Research, pp.8051-8055.

2. Srivastav, Y. and Alam, S., Schematic Brief Discussion on Bulimia Nervosa: A Psychiatric Disorder and Its Medical Management.

3. Hay, P.J. and Claudino, A.M., 2010. Bulimia nervosa. *BMJ clinical evidence*, *2010*, p.1009.

4. Jain A, Yilanli M. Bulimia Nervosa. [Updated 2023 Jul 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing;

5. Mohajan, D. and Mohajan, H.K., 2023. Bulimia Nervosa: A Psychiatric Problem of Disorder. *Innovation in Science and Technology*, *2*(3), pp.26-32.
6. Eddy, K.T., Dorer, D.J., Franko, D.L., Tahilani, K., Thompson-Brenner, H. and Herzog, D.B., 2008. Diagnostic crossover in anorexia nervosa and bulimia nervosa: implications for DSM-V. *American Journal of Psychiatry*, *165*(2), pp.245-250.
7. Grilo, C.M., Ivezaj, V. and White, M.A., 2015. Evaluation of the DSM-5 severity indicator for bulimia nervosa. *Behaviour research and therapy*, *67*, pp.41-44.
8. Corcos, M., Flament, M., Atger, F. and Jeammet, P.H., 1996. Pharmacologic treatment of bulimia. *L'encephale*, *22*(2), pp.133-142.
9. Wilson, G.T., Fairburn, C.C., Agras, W.S., Walsh, B.T. and Kraemer, H., 2002. Cognitive-behavioral therapy for bulimia nervosa: time course and mechanisms of change. *Journal of consulting and clinical psychology*, *70*(2), p.267.
10. Mehler, P.S., 2003. Bulimia nervosa. *New England Journal of Medicine*, *349*(9), pp.875-881.

11. Yager J, Devlin MJ, Halmi KA, et al.; American Psychiatric Association Work Group on Eating Disorders. Practice guideline for the treatment of patients with eating disorders. 3rd ed. Washington, DC: American Psychiatric Association; 2006.
12. Mitchell JE, Crow S. Medical complications of anorexia nervosa and bulimia nervosa. *Curr Opin Psychiatry*. 2006;19(4):438-443.