

Cardiovascular alterations in eating disorders

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Short Communication

The eating disorders (ED) represent the emerging psychiatric disorder in decades, with a prevalence of 0.1-2.1% for anorexia and 1-3% for bulimia [1-3]. Unfortunately, the estimated prevalence must be considered failing for the growing spread of subclinical forms of these disorders, with a frequency that is up to 5 times higher than that of full syndromes [4,5].

These diseases have clear prevalence between women (95%) and appearing on average between 14 and 18 years without predilection of class or race, are now increasing cases prepuberal and it's not uncommon forms in old age [3].

Often ED is comorbidity with other psychiatric disorders such as depression, substance abuse and anxiety disorders [1]. There are also frequent physical complications, including serious heart problems and renal insufficiency [1]. Subjects with ED presenting a risk of death 12 times greater than that of healthy subjects comparable age: they represent a very important social issue for all developed countries [6-8].

The issue of eating disorders has been a problem for the psychiatric nosography since the first description of anorexia nervosa was considered as origin mental disorder [9,10]. Until the seventies, these disorders were identified from the point of view of events as diagnostic, or as a form of neurosis or psychosis. In 1977, Hilde Bruch proposed a unified framework psychopathology of eating disorders (ED) and clinical among them apparently varied as those of mental psychogen obesity, bulimia nervosa and other forms of malnutrition psychogen were collected in a category separate diagnostics [11].

The systems currently most widespread classification of psychiatric syndromes, such as the International Classification of Diseases (ICD-10) and the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), have fully recognized the autonomy of nosographic ED, compared to major psychiatric syndromes, and distinguish the following diagnostic categories: Anorexia Nervosa, Bulimia Nervosa and Eating Disorders not otherwise specified (BED). All these conditions have a substantial overlap of features and poor psycho stability: it is not uncommon that a single person making a first framework of anorexia and bulimia later, or *vice versa*. BED has been recently proposed to the attention of clinicians and researchers but not yet formally approved as a diagnostic category [12].

The current prevalence, similar to those reported in the literature for the international segment of the population most at risk, namely for women aged between 12 and 25 years, are as follows: Anorexia Nervosa 0.3-0.5%; Bulimia Nervosa 1-3%, BED 6%. This last involves a substantial proportion of the obese population, even adults, to varying degrees depending on the degree of obesity and other factors (10-30%).

The *anorexia nervosa* is characterized by excessive influence of weight and body shape on the levels of self-esteem. They are constantly present distortions of the body and pathological fear of becoming fat. The person feels fat even if it is objectively underweight, or perceived as disproportionately fat certain parts of your body (for example, the abdomen). Therefore, to lose weight, says prolonged fasting, trying to get rid of ingested food (through vomiting and / or misuse of laxatives, diuretics or enteroclysmi), or undertake physical activity too. The result is a decrease in body weight below the minimum level considered normal for the age and stature. The person test pervasive feelings of failure and inadequacy, offset only in part by positive feelings arising from the implementation of a rigid control of their hunger. Such sentiments may push the subject to a progressive social isolation, favoured also by the need to hide pathological eating habits.

The weight loss is often accompanied by physical problems stemming dall'inadequata nutrition. The most characteristic sign is the disappearance of menstruation (or delay in their appearance, in the case of girls age prepuberty). There may be also demineralization bone, skin, gastrointestinal disorders, muscle damage. In very severe cases, death can take over for disorders of cardiac function. The framework psychopathology can complicate the emergence of depressive syndromes, sometimes with suicidal ideation, and anxiety disorders [1-12].

The onset is usually taken after a diet in order to lose a few pounds. In the early stages, the person test a feeling of euphoria for successful fulfilment of the weight loss and then, with time and continued with the weight loss, comes a time when a person completely loses control over their food, so the weight loss continues without any possibility to arrest him voluntarily. The course may be limited to a single episode of illness or, more frequently, is a chronic low - to-continuous or subcontinuo. In approximately 50% of cases, followed by bulimic behavior [1-12].

The *bulimia nervosa*, as well as the anorexia, is characterized by excessive influence of weight and body shape on the levels of self-esteem, with pervasive feelings of failure and inadequacy. The eating is characterized by the presence of episodes of uncontrolled eating (Binge), during which the person ingests mass wide variety of foods and the feeling of not being able to stop eating. Binge is normally preceded by unpleasant sensations and is often described a painful sense of emptiness. It closed because the person feels very full or nauseated or because they feared discovery or because he sold out

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stocks of food. Although binge can ease the tension and the sense of emptiness that have deteriorated, the feelings of guilt which assailed the person after the binge incident are such as to generate new anxiety and tension which, in turn, can lead to a new binge [1-12]. The person tries to mitigate the effects of food ingested during binges, implementing behaviors elimination and/or compensation, such as self-vomiting, by using laxatives, long periods of fasting and /or excessive exercise. The bulimia is often associated with depression and anxiety disorders. In several cases established abuse of alcohol and/or other substances. Bulimics people often have a normal weight or slightly overweight. Moreover, the consequences of binges are not, generally, as well as in serious nervous [1-12].

Binge eating disorders

In recent years, between partial syndromes, has aroused particular interest, even for the high frequency, a syndrome called Binge Eating Disorders (BED). In this syndrome are present crises of compulsive greed for food without the serious and dangerous behaviours compensation of BN (DSM IV). The term BED is used today in medicine and psychiatry to define a symptom which transversely across the entire area of ED and the whole spectrum of body weight. It is important to stress the high frequency of obese patients suffering from BED. Indeed, the BED seems relatively rare in the general population, with a prevalence rate between the 0.7 and 4.6%, while it is reflected by common in obese patients, with values increasing prevalence, parallel to the degree of BMI, (between 30 and 70%). Contrary to the AN and BN, BED seems to be only moderately more frequent in women (ratio 3:2).

The psychopathology core in BED as in BN is the presence of binges. In BED would be more correct to speak of days rather than episodes of binge. The binge is characterized by single rather than several binge crises. Complications related to organic BED are diabetes mellitus, hypertension, cardiovascular disease, certain types of cancer, bone diseases and their incidence is increasing in parallel to the degree of obesity. It is found in patients BED greater prevalence of depression, dysthymia, DAP and preventing disorder and borderline personality [1-12].

Cardiovascular alterations in eating disorders

Alterations in cardiovascular system occur with high frequency (up to 75% of hospitalized AN patients) on the functional level (bradycardia, arterial hypotension, orthostatic hypotension secondary to hypovolemia, reduction of cardiac output) and structural (reduction of ventricular mass and cardiac volumes, reduction of cardiac cavities with consequent valve prolapse, especially the mitral valve, pericardial effusion) (13, 14). They are usually caused by malnutrition and / or rapid weight loss and alterations in the hydro-electrolytic state related to vomiting, abuse of laxatives and diuretics. Many of the alterations described are reversible with the correction of specific deficits and with the recovery of body weight [14,15,38,39].

Monitoring with the regular execution of ECG and Ecocardiogram is necessary.

The most common abnormalities highlighted by ECG in AN are represented by reduced frequency, low QRS voltages, ST tract abnormalities, QT interval prolongation, U wave appearance linked to electrolyte imbalances, in particular hypokalemia [16]. The most critical alteration to the ECG is the prolongation of the QT interval, correlated with a high incidence of sudden death following arrhythmias (ventricular tachycardia, ventricular fibrillation) [13-18]; the QT measurement must always be correct for heart rate (QTc), higher QTc values of 0.44 are high risk for arrhythmia; in these cases a nutritional

correction (conducted with adequate criteria) and the possible correction of electrolyte imbalances, where present [19] is mandatory; it is also necessary to evaluate the possible effect of associated pharmacological therapy (serotonergic antidepressant species), which in itself may be due to alteration and other serious cardiac complications such as torsades de pointes and sudden death [20].

Bradycardia (heart rate (HR) <60 bpm) is one of the most common symptoms seen in patients with AN (FC <40 bpm is a criterion for hospital admission) [21]; it is a consequence of the reduction of the basal metabolism and of the vagal hypertone; also bradycardia is an adaptive phenomenon to protein-energy malnutrition, allowing, through a reduction in cardiac output, to reduce energy expenditure. Therefore a normal or increased CF, in cases of moderate-severe AN, may be indicative of clinical risk [22]. Arterial hypotension (PAS <90 mmHg, PAD <60 mmHg) may be secondary to the increase in vagal tone and to the reduction of the circulating levels of FT3, and also due to the state of dehydration for purging behaviors.

Orthostatic hypotension is an increase in heart rate of 20 bpm or a blood pressure reduction of 20 mmHg in the transition from clinostatism to orthostatism; consequent to the altered functioning of the autonomic nervous system [23], can cause lipothymia, if marked may be indicative of need for hospitalization [24]. Congestive heart failure may occur in severely defiled and/or re-fed AN patients (due to excess volumes administered and/or hypokalemia and, above all, hypophosphoremia) [25-27]. Finally, it should be remembered that during weight recovery there is a rapid increase in basal metabolic rate and volemia with increased demands for cardiac work, which is however conditioned by the recovery of the myocardial mass which occurs much more slowly; it follows that the lower the starting BMI, the more gradual recovery must be in order to avoid exposing the heart to a risky functional overload.

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