## CASE REPORT

# WHEN HEALTHY EATING BECOMES UNHEALTHY - ATYPICAL EATING DISORDER IN A MALE PATIENT

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**Abstract**

***Objective:*** **This case report highlights a case of eating disorder in a male patient, traditionally a female-predominant illness, frequently missed in male patients for a variety of reasons.** ***Method:*** **We report a case of a young Chinese male with atypical symptoms of eating disorders, presenting with serious physical complications of malnutrition.** ***Result:*** **The case presented with the difficulties of diagnosing eating disorders in males and the potential complications of severe malnutrition.** ***Conclusion:*** **Clinicians need to be aware of the possibility of eating disorders in cases of medically unexplainable weight loss in males as they frequently present with atypical symptoms.** ***ASEAN Journal of Psychiatry, Vol.12(2), July – December 2011: XX XX.***

**Keywords: eating disorders, males**

## Introduction

Eating Disorders (ED) has traditionally been seen as a female-predominant illness. Recent studies suggest that males represent 10-20% of cases of Anorexia Nervosa (AN) and Bulimia Nervosa (BN) and up to 40% of binge eating disorder [1]. Males have been found to be less likely to recognize their eating disorder, are more likely to be misdiagnosed with other mental health problems, and are less likely to receive treatment and be referred to a specialist eating disorder service [2]. Undiagnosed and untreated eating disorders can result in serious long-term physical and psychological complications. We present the case of a young Chinese man with an atypical eating disorder, presenting with serious medical complications. The aim of this case report is to discuss the difficulties of diagnosing eating disorders in males and the potential complications of severe malnutrition as a result of delayed diagnosis.

## Case Report

Mr. C was a 29 year-old, single, male engineer when he was first admitted in 2004 to haematology with a 1-year history of severe weight loss of 23kg, associated with loss of appetite, early satiety, general malaise and lethargy. He had no past medical or psychiatric history.

On physical examination, weight was 42kg, height 1.70m (BMI 14.5). There was mild hepatomegaly but no other significant findings. Laboratory studies showed pancytopenia and mild transaminitis (Table 1). A comprehensive work-up was done to exclude an organic cause. Thyroid function, tumour/ autoimmune markers, septic work-up, Computerized Tomography (CT) neck/ thorax/ abdomen/ pelvis, esophagogastroduodenoscopy (OGDS), colonoscopy and nasopharyngoscopy were normal. Bone marrow aspiration showed decreased haemopoesis with serious atrophy of the marrow consistent with severe malnutrition.

A psychiatric consult was requested to exclude an ED. Mr. C recognized he was severely underweight but attributed it to an

inability to eat as a result of early satiety and loss of appetite. He denied body-image issues and felt he was thin even at his premorbid weight of 64kg (BMI 22.4). He was careful with his diet – taking more fruits/ vegetables/ steamed food – but claimed it was for health reasons rather than to lose weight. He ran 30min a day to "keep fit". A definitive diagnosis of an ED was difficult at that point as he was still undergoing medical investigations.

He was discharged after a week. The pancytopenia persisted and he continued follow-up with haematology but defaulted after a few months.

In 2010, he was readmitted after he was found unresponsive at home. He regained consciousness after IV Dextrose 200ml was given in the ambulance. His weight had plummeted to 34.5kg (BMI 11.9). Lab studies showed severe panyctopenia and transaminitis (Table1). CT brain showed cerebral atrophy disproportionate with age. Neuropsychological testing showed that his current neuropsychological test profile was below expectations in several domains such as executive functioning, attention and processing speed.

Detailed history revealed an increasing preoccupation over the last few years in wanting to be healthy. To Mr. C, being healthy meant "eating healthily and exercising regularly". He was preoccupied with "eating healthily according to the food pyramid". His diet consisted predominantly of fruits and steamed vegetables whilst omitting most proteins, fats, salt, and sugars, which he deemed "unhealthy". He was consuming approximately 1200kcal/ day. He was also exercising compulsively - running an hour a day and developing a highly ritualistic manner of doing static stretching exercises. He recognized that he was extremely underweight and wished to gain weight. However, his misconceptions and obsessions with "healthy eating and exercising" made it difficult for him to alter his patterns. Throughout this period, he was still able to work. He was not depressed and did not exhibit any other obsessive-compulsive behaviour.

Mr. C was diagnosed to have an atypical eating disorder, also known as Eating Disorder Not Otherwise Specified (EDNOS) according to DSM IV. He also had community-acquired pneumonia with septic shock. After initial medical stabilization, he was transferred to our Eating Disorder Unit for further nutritional rehabilitation. He was educated about his illness and encouraged to complete his meals. He was referred to the dietician to work out his caloric requirements to gain weight and to correct his misconceptions about supposedly healthy eating. To prevent refeeding syndrome, his caloric requirement was gradually increased to include supplements (Resource 2.0 twice a day) to aid weight gain. He completed his meals but found it difficult to comply with resting in bed. He was frequently caught exercising in his room despite repeated advice to rest. He was started on Fluoxetine 10mg OM to help with his compulsion to exercise. He was also referred to the physiotherapist to educate him about appropriate exercise.

Mr. C stayed 7 weeks and gained a total of 9.3kg to 43.8kg (BMI 15.2) upon discharge. He returned to work 2 months later and continued to make good weight gain, eventually achieving a minimum healthy weight of 54.2kg (BM 18.7) 3 months post-discharge. Eight months after discharge, he has managed to maintain his weight, his eating had largely normalized and he was exercising moderately - running 30mins twice a week. His lab results showed significant improvement in tandem with his weight gain (Table 1).

**Table 1 Chronological report of weight, blood count, electrolytes and liver enzymes**

| Date | Wt (kg) | BMI ($kg/m^2$) | HB (g/DL) | TW ($x10^9$/L) | PLT ($x10^9$/L) | NA (mmol/L) | K (mmol/L) | ALT (IU/L) | AST (IU/L) |
|---|---|---|---|---|---|---|---|---|---|
| 2004 | 42 | 14.5 | 10.7 | 1.8 | 139 | 137 | 4.3 | 151 | 118 |
| Sep 2010 | 34.5 | 11.9 | 7.8 | 0.6 | 125 | 128 | 4.8 | 440 | 293 |
| Wk 1 (Sep) | 35.7 | 12.4 | 7.7 | 2.5 | 142 | 131 | 4.2 | 186 | 124 |
| Wk2 (Sep) | 38.2 | 13.2 | 9.2 | 2.6 | 319 | 131 | 4.3 | 108 | 69 |
| Wk3 (Sep) | 37.2 | 12.9 | 8.0 | 1.9 | 425 | 131 | 4.1 | 71 | 77 |
| Wk 4 (Sep) | 41.0 | 14.2 | 6.9 | 2.4 | 294 | 131 | 4.0 | 45 | 48 |
| Wk 5 (Oct) | 41.4 | 14.3 | 8.2 | 1.8 | 227 | 132 | 4.1 | 37 | 45 |
| Wk 6 (Oct) | 43.2 | 14.9 | 9.2 | 3.2 | 322 | 135 | 4.8 | 37 | 49 |
| Wk 7 (Oct) | 43.8 | 15.2 | 8.3 | 3.5 | 354 | 135 | 4.1 | 29 | 38 |
| Wk 8 (Oct) | 44.2 | 15.3 | 9.2 | 2.8 | 277 | 137 | 5.0 | 27 | 38 |
| Wk10 (Nov) | 44.9 | 15.5 | 11.0 | 2.4 | 204 | 140 | 4.6 | 30 | 38 |
| Nov 10 | 48.9 | 16.7 | 10.8 | 2.7 | 204 | - | - | 33 | 41 |
| Dec 10 | 51.0 | 17.6 | 11.5 | 3.2 | 213 | 139 | 5.1 | 39 | 51 |
| Jan 10 | 52.9 | 18.3 | 12.1 | 3.9 | 189 | 140 | 5.2 | 38 | 49 |
| Feb 10 | 54.2 | 18.7 | 12.3 | 3.5 | 162 | 138 | 4.8 | 33 | 40 |
| May 10 | 54.2 | 18.7 | 12.1 | 3.7 | 149 | - | - | 28 | 35 |
| Jul 10 | 54.9 | 18.9 | 12.1 | 4.5 | 171 | - | - | 26 | 39 |

BMI – body mass index
HB – haemoglobin
TW – total white cell count
Plt – platelet count
Na – serum sodium
K – serum potassium
ALT – alanine transaminase
AST – aspartate transaminase

**Discussion**

Diagnosis of ED in men is frequently missed for a variety of reasons. Firstly, ED has been seen as a predominantly female illness afflicting mainly teenagers and young women. Secondly, diagnostic criteria and most assessment instruments for the diagnosis of EDs are gender biased and normed for women rather than men [3]. They are aimed at the types of weight concern, shape concern and methods of weight control common to women (thinness, dieting) rather than men (low body fat, muscularity, strength, exercise) [4]. Thirdly, men tend to present more frequently with atypical eating disorders. Atypical eating disorders (also known as EDNOS), refers to a group of EDs that meet some but not all of the diagnostic criteria for AN or BN. Examples include binge eating disorder, in which individuals indulge in binge eating but do not exhibit the compensatory behaviour required to make a diagnosis of BN. Another example are females who are still menstruating despite satisfying all the other diagnostic criteria for AN. A third of male patients present with atypical symptoms compared to about 10% of women [5]. There was a low index of suspicion at initial consult with Mr. C as it is unusual for AN to first present in man, and he also did not present with features of body-image distortion, body weight and shape concerns typical of AN.

Mr. C was diagnosed to have EDNOS as he did not satisfy the criteria for AN. Although he presented symptoms similar to AN - severe weight loss, food restriction, excessive exercising and preoccupation with food - however he did not have the core feature of body-image distortion. He recognized he was severely underweight and wanted to gain weight. The driving force behind his symptoms was a compulsion to be healthy rather than to be thin.

Orthorexia nervosa (ON) was a term coined by Steven Bratman in 1997 to define a

pathological fixation on the consumption of appropriate and healthy food. Unlike AN/ BN, patients with ON are preoccupied with consuming healthy, pure food rather than preoccupation over the quantity and physical appearance. Mr. C falls into this subgroup but because ON is currently not a recognized diagnostic entity on its own; he still falls into the diagnostic subgroup of EDNOS.

This case also highlights the serious medical complications that could arise from the malnutrition as a result of an ED – including pancytopenia, hypoglycaemia, transaminitis and cerebral atrophy. Anaemia and low peripheral blood counts are frequently seen in the setting of chronic starvation, and are likely due to multiple vitamin and mineral deficiencies as a result of inadequate oral caloric intake [6]. Numerous studies report a strong relation between malnutrition and liver damage [7]. There is often a rapid recovery of liver enzymes with weight restoration suggesting that liver damage may be secondary to acute hypoperfusion and is reversible with refeeding, as is the case with Mr. C. His liver function normalized as weight normalized (Table 1).

In conclusion, clinicians need to be aware of the possibility of eating disorders in cases of medically unexplainable weight loss in males as they frequently present with atypical symptoms. A detailed history both from patients and family exploring weight history, typical day food diary, body-image issues and amount of exercise would frequently provide clues to the diagnosis. Eating disorders have potentially serious medical complications and high mortality rates. Earlier diagnosis could prevent serious medical complications that may arise as the eating disorder becomes more chronic.

**References**

1. Muise A, Stein D & Arbess G. Eating Disorders in adolescent boys: A review of the adolescent and adult literature. Journal of Adolescent Health, 2003; 33(6), 427-435

2. Morgan JF & Arcelus J. Body image in gay and straight men: a qualitative study. European Eating Disorders Review, 2009; 16(6), 435-443

3. Wade T, Byrne S & Bryant-Waugh R. The eating disorder examination: norms and construct validity with young and middle adolescent girls. International Journal of Eating Disorders 2008; 41(6), 551-558

4. Rhys Jones W & Morgan JF. Eating disorders in men: a review of the literature. Journal of Public Mental Health 2010; 9(2), 23-31

5. Carlat D, Carlos A, Camargo Jr & Herzog D. Eating Disorders in Males: A report of 135 patients. Am J Psychiatry 1997; 154:1127-1132

6. Vande V, Mazza J & Yale S. Haematological and Metabolic Abnormalities in a patient with Anorexia Nervosa. Wisconsin Medical Journal 2004 Vol 103, 38-40

7. Giordano F, Arnone S, Santeusanio F, & Pampanelli S. Brief elevation of hepatic enzymes due to liver ischemia in anorexia nervosa. Eat Weight Disord 2010; 15(4): 294-7

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