

LETTERS

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Are late-onset eating disorders in the elderly really the more common variant? Concerns around publication bias

Lapid *et al.* (2010) provide a fascinating insight into the much over-looked problem of eating disorders (EDs) in older adults and highlight the fact that an ED is often not considered in our differential when assessing elderly patients.

However, we have some concerns over the suggestion that the majority of EDs in older adults are new-onset (69% of cases in the paper by Lapid *et al.*) rather than a recurrence or continuation from earlier in life. This seems a surprising finding as intuitively we feel that cases of EDs in old age are more likely to be in the context of pre-existing undetected symptoms or a recurrence rather than *de novo*. We briefly describe a case of early onset anorexia nervosa (AN) carried forward into old age, which we think is more representative of the true presentation of EDs in the elderly. The patient provided written informed consent to publication.

Mrs D. is a 75-year-old patient with a diagnosis of AN, restrictive type, with onset in early life. This was in the context of ongoing family relationship difficulties since her childhood years and both obsessive and perfectionist personality traits. She did well with psychological therapy and was discharged. The patient re-presented in 2007 with depressive symptoms and a BMI of 14.8, against the background of an ongoing difficult relationship with her daughter.

She would accept only energy drinks and small amounts of liquidized meals. No other physical health issues were identified. She is currently receiving input from a family therapist and a community dietician in addition to medical and psychiatric input from primary care and community psychiatry.

If the findings of Lapid *et al.* (2010) are representative of the true picture, one potential explanation could be that smaller numbers of pre-existing/ recurrent EDs in old age are seen due to the high mortality rate in AN. However, this would not account for the similarly higher proportion of apparently new-onset cases they find of Bulimia Nervosa which does not carry a similar risk of mortality (though numbers are small, $n = 5$). That is to say, one would expect a higher prevalence of pre-existing/ recurrent BN than *de novo* cases, contrary to their finding.

We suggest that, instead, the study by Lapid *et al.* (2010) is subject to both powerful publication bias and further biases within each case study. New-onset ED in an older age patient is surprising and represents a more desirable subject for publication than a known pre-existing diagnosis in an aging patient. Hence the collection of case studies will tend to over-represent apparent “new-onset” EDs.

A second problem is with the diagnosis of “new-onset” EDs in patients at the end of their life without robust data regarding their adolescence. There will be problems with recall bias and with the reliability and validity of diagnoses and clinical information from early life in case studies of older adults ranging from 1936 to 2008 when operational criteria for the diagnosis of EDs first emerged in 1980 (DSM-III; American Psychiatric Association, 1980). Diagnostic practice will vary greatly over a patient’s life time and more so at times when there are no clear criteria.

It is known that many potential cases of ED never present to services or receive treatment. Similarly, it is known that many patients move between diagnoses of AN, BN and atypical EDs (Fairburn and Harrison, 2003). Apparent new-onset AN could then be either an exacerbation of a pre-existing subclinical or unrecognized syndrome or conversion from atypical ED. Hence it is difficult to be confident in stating the absence of ED symptomatology in an older adult’s past history.

The study by Lapid *et al.* (2010) raises further questions of etiology. It has been observed that EDs run in families and that AN is highly heritable with twin concordances of MZ = 55% versus DZ = 5% (Fairburn and Harrison, 2003). It is difficult to understand why patients with a genetic predisposition to the compulsive and impulsive behaviors seen in EDs would develop a clinically significant syndrome later in life rather than in adolescence or early adulthood. If the genetic predisposition has always been there and is etiologically significant, why would a clinical ED not emerge earlier in life? It would be interesting to see family studies in patients with apparent new-onset ED in later life and whether they reflect the patterns of depression, obsessive and perfectionist personality traits and substance misuse seen in relatives of a proband with early onset ED.

Lapid *et al.* (2010) suggest that AN could be “ushered in” by loss of appetite due to other medical conditions in this population. We would caution that whilst the *symptom* of anorexia is common in older adults due to both psychiatric

and general medical conditions, this needs to be carefully distinguished from AN, *the disorder*. They suggest greater control over concerned relatives at a time when failing health threatens autonomy could be an explanation for new onset ED. However we need to be careful when delineating significant clinical syndromes from, for example, hunger strike expressed as concern over body weight. The latter would constitute a very different clinical entity to refusal to eat due to a fear of weight gain. Given the very different psychological and environmental stressors in later life and our observation regarding heritability, it may prove useful to regard new-onset EDs in older adults as a further subcategory of ED with potentially unique predisposing and precipitating factors.

Certainly there is room for further study on the actual prevalence of continued, recurrent and new-onset EDs in the older adult population. In addition, assessment of the characteristics of ED in this population in terms of family studies

and response to treatment could prove very illuminating.

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Is the proposed DSM-V Suicide Assessment Dimension suitable for seniors?

High suicide rates are observed in elderly men, and the identification of high risk individuals is a challenging task. The Suicide Assessment Dimension (see Box 1) is intended to aid the evaluation process. We are concerned that the proposed assessment may miss some seniors in need of intensive management.

Consider the case of Mr. D, an 80-year-old widower and retired policeman who lives with his son and daughter-in-law. He receives treatment for diabetes, hypertension, and coronary heart disease. There is a past history of alcohol abuse, but Mr. D has abstained since his first stroke five years ago. The hemiparesis improved after a short period of rehabilitation, and Mr. D was once again able to enjoy his favorite pastimes of hunting and fishing. Last year, Mr. D lost his wife of 53 years. He was never one to talk about feelings, but found consolation during fishing trips with his long-time buddies. Six months after his wife's death, Mr. D suffered a second stroke. This one left him wheelchair-bound. He did not do well in rehabilitation this time, and often found excuses not to attend. Mr. D's son has arranged today's consultation. His father, who was always a "doer", now spends his days in front of the TV. On several occasions, the family has made arrangements for wheelchair transport so

that Mr. D can join in on outings, but he opts to stay at home. They ask if his loss of interest might be a sign of dementia, and if medication will alleviate the condition. Upon examination, Mr. D is lucid. His performance on a battery of cognitive tests is within the normal range. He does not appear depressed, and is able to laugh, but his tone is somewhat sarcastic. There is no psychomotor slowing, and no psychotic thought content. Upon direct questioning, Mr. D denies feelings of depression and suicidal ideation, but complains that life has pretty much lost its meaning. Mr. D abhors the fact that he is dependent on his daughter-in-law for assistance with dressing and personal hygiene.

Mr. D does not fulfil criteria for major depressive episode, but he displays loss of interest and apathy, yielding one point for "worsening of depressive symptoms" (Item 5). His thought content signals hopelessness (1 point, item 7). The total score (2 points) would not give cause for "increased concern".

Risk factors for suicide in this age group differ somewhat from those of young and middle-aged persons. While Item 2 (long-standing tendency to lose temper or become aggressive with little provocation) is highly relevant when it comes to younger suicides, personality features that infer risk for suicide may alter or attenuate with age. A recent large psychological autopsy showed an inverse relationship between impulsive aggression and age (McGirr *et al.*, 2004). However, increasing levels of harm avoidance were observed with increasing age