EATING DISORDERS AND PSYCHOSIS AS INTERTWINED DIMENSIONS OF DISEMBODIMENT: A NARRATIVE REVIEW

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Abstract

Although psychosis is not one of the most commonly recognized psychiatric comorbidities of Eating Disorders (ED), there is an increasing empirical evidence of associations between these psychopathological conditions. Indeed, ED as well as early manifestations of schizophrenic psychosis emerge during developmental years and might present some affinities in the presentation at onset. For example, adolescents with anorexia nervosa (AN) may report psychotic-like eating-related symptoms such as delusional-like body image distortions and/or “anorexic voice”. Conversely, early expressions of schizophrenia spectrum vulnerability might involve altered bodily experiences, delusional ideation on food and eating as well as pseudo-anorectic behaviors. From a phenomenological perspective, this partial symptomatic overlap may lie on common features of disturbed corporeality in terms of disembodiment, although these alterations of embodiment are presumably rooted in distinct pathogenic pathways (e.g., primary childhood ontogenetic pathway in schizophrenia vs. a secondary adolescent ontogenetic pathway in AN). A clinical-phenomenological attitude could be important not only to better discern potential overlaps and affinities between the two conditions, but also to better conceptualize and treat those background alterations of the embodied self. In particular, a phenomenological exploration of the experiential side of eating-related behaviors could be a decisive step to support early differential diagnosis and treatment appropriateness.

Key words: disembodiment, schizophrenia, eating disorders, anorexia, psychosis, self-disorders

Introduction

Although attenuated psychotic symptoms and related clinical high-risk constructs are conceptualized as transdiagnostic predictive features of persistent clinical severity (McGorry et al., 2018), data on their prevalence across diagnostic categories outside the schizophrenic spectrum are relatively scarce. A recent study (Mensi et al., 2020) partly compensated for this lacuna in the area of Eating Disorders (ED): authors investigated the presence of subthreshold psychotic symptoms in 94 adolescents with clinically ascertained ED, finding an astonishing prevalence of 84% (i.e., more than 4 out of 5 adolescents with ED manifest attenuated psychotic symptoms). This finding deserves further empirical confirmation but is surprising since it preliminarily suggests that pre-psychotic features in ED could be considered the rule rather than the exception, and it is unexpected because, allegedly, psychosis is not one of the most commonly recognized psychiatric comorbidities of ED (Miotto et al., 2010; Keski-Rahkonen and Mustelin, 2016).

Although ED and psychosis, at least in terms of full-blown categorical diagnostic phenotypes, have been only modestly associated at epidemiological level, there is increasing empirical evidence of possible connections in multiple directions: longitudinally, ED preceding schizophrenia (Kelly et al., 2004; Malaspina et al., 2019) or psychotic experiences preceding ED (Solmi et al., 2018); cross-sectionally, psychotic features in ED (Brödric et al., 2020; Rojo-Moreno et al., 2011) and eating-related symptoms in psychotic disorders such as schizophrenia (Fergusson and Namier, 1988; Kouidrat et al., 2014; Lyketsos et al., 1985; Yum, 1988).
In this perspective, Seeman (2014) delineated seven hypotheses on the relationship between ED and psychosis: I) ED and psychosis as entirely separate disorders that can, by chance, occur in the same person; II) one condition as primary and causing secondarily the other one: for example, ED-related starvation causing transient psychotic symptoms, or inversely, psychotic symptoms as delusional fear of contamination or of poisoning causing secondarily ED; III) control of food intake as an attempt to ward off acute psychosis; IV) body image distortion of ED representing a delusional-like psychotic phenomenon; V) one disorder representing the prodromal early sign for the onset of the other one; VI) antipsychotics used to treat psychosis inducing ED; VII) psychotic symptoms as a marker of severity for ED. These hypotheses are not mutually exclusive and distinct phenotypic relationships may occur longitudinally and cross-sectionally between ED and psychotic symptoms, standing on shared genetic vulnerability (Duncan et al., 2017), as confirmed by their familial co-aggregation (Zhang et al., 2021). Moreover, a possible pathophysiological link may be represented by dopamine dysfunction, i.e., an established feature of schizophrenia spectrum disorders (Howes et al., 2012) and a possible feature of ED (Barry & Klawans, 1976). Interestingly, psychotic symptoms are associated with hyper-dopaminergic states, and hyper-dopaminergic states are described also in early stages of AN (Frank et al., 2018), although, in the absence of longitudinal prospective studies, it is difficult to determine whether putative abnormalities in dopamine precede the disorder representing a risk factor or arise as a consequence of starvation (Beeler & Burghardt, 2022).

Overall, all these findings suggest that the intertwine between ED and psychosis is clinically salient and rather important to discern beyond the pure diagnostic co-occurrence. In this respect, a phenomenological perspective might be illuminative. Indeed, there are some phenomenological affinities between ED and psychotic conditions, such as schizophrenia, which may confer a vulnerability for the development of both disorders. This paper reviews psychotic features in ED and eating-related symptoms in psychosis with the specific and focused aim of discussing the phenomenological intertwine between these mental disorders.

Methods

Given the specific clinically-tailored scope, a focused review on salient aspects is more suited than a systematic review. We searched articles in Medline, EMBASE and Cochrane Library databases with the keywords “eating disorders”, OR “anorexia nervosa” AND “psychosis” OR “schizophrenia”. The search was conducted on December 15, 2021 and repeated on March 28, 2022.

After the screening of titles and abstracts, those studies that appeared to represent “a best fit” with the research questions (1. Psychotic-like features in Eating Disorders; 2. Eating disorders-related features in psychosis) were fully inspected and relevance was discussed by at least two authors. In summarizing the results of this procedure, a narrative approach has been followed to offer to a wide readership the main findings of the review. Because of the eminent focus on overlapping and distinct features between these symptomatic areas, findings are organized in two sections. Therefore, in the discussion section, clinical, developmental and phenomenological aspects relevant for differential diagnosis are emphasized.

Results

Psychotic-like features in Eating Disorders

Attenuated psychotic experiences may precede the full development of ED (Solmi et al., 2018) as well as psychotic features may be concomitant with ED (Brodrick et al., 2020). In this perspective, in the clinical pictures of ED and particularly in case of Anorexia Nervosa (AN), there are at least two phenomena that present phenomenological affinities with psychotic symptoms.

The first prominent feature is represented by distortions of body image (Glashouwer et al., 2019) including shape concerns and drive for thinness (Hartmann et al., 2013), usually associated with idiosyncratic convictions on food effects on body (O’Connell et al., 2018). Both these features may range dimensionally from overvalued ideas to delusion-like beliefs, for their characteristics of being felt with poor insight, dominating mental contents and being poorly amenable to change in light of conflicting evidence (Behar et al., 2018; Phillipou et al., 2017); indeed, studies with the Brown Assessment of Belief Scale (BABS: Eisen et al., 1998) found that a minority ranging from 20% to 28.5 of patients with ED and in particular with AN, presented beliefs on body image with delusional characteristics (Konstantakopoulos et al., 2012; Steinglass et al., 2007), i.e., associated with poor insight and not easily amenable to change, globally indicating the severity of eating disorder attitude (De Young et al., 2022). In a perspective of differential diagnosis, overvalued ideas and delusional beliefs are subjectively felt as more distressing and preoccupying (i.e., egodystonic) in AN than in the schizophrenia spectrum (Mountjoy et al., 2014).

A second phenomenon worth considering is the presence of auditory hallucinations focused on eating and body concerns, subjectively felt in terms of a kind of “anorexic voice” (Rojo-Moreno et al., 2011), described as powerful, negative and omnipotent (Aya et al., 2019), that at least in some cases may be associated to childhood trauma and mediated by dissociative processes (Pugh et al., 2018). The phenomenon of the “anorexic voice” is associated with more negative eating attitudes, more severe compensatory behaviors, longer duration of illness and greater likelihood of having the binge-purge subtype of AN (Pugh and Waller, 2017). A phenomenological study on AN clearly identified a process of progressive externalization of such voice, often coinciding with a switchover from controlled dieting to ED (Williams and Reid, 2012). In this perspective and probably not casually, eating disorder symptoms in AN are strongly related to obsessions (Levinson et al., 2019, that phenomenologically may represent the subjective precursor of the development of auditory hallucinations along a progressive process of externalization (Poletti and Raballo, 2019; Raballo, 2017; Rasmussen et al., 2020).

Overall, ED present some clinical features with eating contents, that along a dimensional gradient of severity may reach a psychotic threshold, usually reflecting the severity of the whole clinical picture, probably in a bidirectional relationship with starvation and BMI decrease.
Eating disorders-related features in psychosis

ED or anomalies in eating behaviors may precede schizophrenia (Kelly et al., 2004; Malaspina et al., 2019) as well as eating-related symptoms may occur in psychotic disorders such as schizophrenia (Bou Khalil et al., 2011; Ferguson and Namier, 1988; Kouidrat et al., 2014; Lyktos et al., 1985; Yum, 2005; Yum et al., 2009). A recent review (Sankaranarayanan et al., 2021) on ED in schizophrenia reported heterogeneous prevalence rates, ranging from 4.4% to 45% for binge eating, from 16.1% to 64%, for food craving, from 27% to 60.6% for food addiction, and from 4% to 30% for night eating, with positive association between antipsychotics and binge eating and between disordered eating and psychotic symptoms, suggesting that eating symptoms may fluctuate together with psychotic symptoms, as well as that long-term antipsychotic exposure may impact on eating behaviours.

Discussion

Intertwine between Eating Disorders

Starting from the astonishing findings of 4 out of 5 adolescent girls with ED presenting prodromal psychotic features (Mensi et al., 2020), we reviewed the border zone of symptomatic overlap between ED, in particular AN, and psychosis, in particular schizophrenic psychosis. In addition to the possible categorial comorbidity allowed by DSM in different longitudinal reciprocal positions (e.g., in association or one preceding the other one), the clinical and phenomenological examination of AN and schizophrenia identified some affinities and differences that could help clinicians in the differential diagnosis of complex cases.

At the clinical level, adolescents with AN can present psychotic-like symptoms i.e., eating-related symptoms that reach a psychotic threshold (such as delusional-like body image distortion and auditory hallucinations), but the content of such symptoms is usually circumscribed to body and food issues, in a rigid and monothematic fashion. Therefore, the most severe fraction of patients with AN present a lack of insight on their symptoms that is similar to the loss of insight that is associated with subjective adhesion to the psychotic dimension in schizophrenia (in the transition from prodromal at-risk symptoms to first episode psychosis). Conversely, in schizophrenia spectrum disorders, psychotic eating-related symptoms are generally not isolated fragments but rather parts of a more extensive psychopathological network including other psychotic and non-psychotic (e.g., negative and disorganized) symptoms, within a more general, pervasive alteration of the subjectivity (Raballo et al., 2021). Moreover, in chronic schizophrenic patients rather than in patients in prodromal or early clinical stages (first episode of psychosis), ED or anomalies in eating behaviors may be caused or triggered by long-term antipsychotic exposure (de Beaurepaire, 2021; Sankaranarayanan et al., 2021).

In the subsequent paragraphs possible overlaps in etiopathogenetic mechanisms at the basis of ED and schizophrenia are discussed.

Etiopathogenetic mechanisms: possible overlaps

In the schizophrenic spectrum, the emergence of full-blown psychotic symptoms does not generally emerge suddenly but rather reflects a prolonged developmental trajectory facilitated by progressive anomalies of subjective experience (i.e., Self-disorders). Self-disorders are trait-like, non-psychotic anomalies of subjectivity that have been recursively and meta-analytically corroborated as schizophrenia-spectrum disorders vulnerability phenotypes (Raballo et al., 2021). They encompass varieties of depersonalization, derealization and similar distortions of the subjective experience, characterized by a diminished sense of existing as an embodied, generally immersed in the world and author of his own actions. A key phenomenological feature underlying Self-disorders is disembodiment, i.e., the lack of an immediate and implicit attunement between the Self and the body. This pathological detachment from the bodily side of the Self has been described in various prototypical forms, e.g., deanimated body (that is a body deprived of the possibility of living personal experience as its own) or disembodied spirit (that is a sort of abstract entity which contemplates its own existence from outside, in a third-perspective rather than in a first-perspective) (Stanghellini, 2009). Several body-centered manifestations (such as cenesthopathies) and bodily-directed symptoms (such as anorectic-like behaviors or recurrent self-harm as expressions of a search for control or contact with the disembodied bodily dimension) might be emergent manifestations of a common alteration of embodiment. Disembodiment in schizophrenia is relatively early along the etiopathogenetic trajectory leading to the clinical stage and might be the primary subjective correlate of an atypical neurodevelopment (Poletti et al., 2019; Poletti and Raballo, 2020a), endophenotypically expressed through subtle childhood motor difficulties and neurological soft signs, as detected in offspring of schizophrenic parents (Hameed and Lewis, 2016; Poletti and Raballo, 2020b).

Disembodiment has been proposed as a key etiogenetic mechanism also in ED, especially in AN (Castellini et al., 2022; Lester, 1997; McBride & Kwec, 2018); although intriguing, probably due to intrinsic difficulties in its operationalization for an experimental investigation, disembodiment has been less extensively studied in AN in comparison with a related but not overlapping construct as body image, whose distortion is a key clinical AN feature. From a phenomenological perspective, disembodiment in AN could be mainly considered secondary to the pubertal shift from a childhood spontaneous bodility to the pubertal body-objectivation (Fuchs, 2022), in which the anorexic body is progressively seen from outside through an optical-and coenesthetic distortion (Stanghellini, 2019), resulting in imbalance between subjectivity and physicality of own’s body (Legrand, 2010). For example, a psychometric study based on a network analysis (Cascino et al., 2019) found that in a sample of AN patients, two of the main dimensions of embodiment (1. feeling extraneous from one’s own body and 2. feeling oneself through objective measures) were the nodes with the highest strength together with receptive awareness, with the latter being a node included in several pathways connecting embodiment dimensions with most of the AN psychopathological dimensions.

In sum, disembodiment in schizophrenia and in AN may follow a distinct pathogenetic trajectory, situated in distinct developmental stages. In schizophrenia disembodiment may result from the primary ontogenetic failure of the Minimal Self in the relation between the subjective experience and its bodily correlates;
this impairment will progressively emerge at the conscious level of Narrative Self around adolescence, in combination with early prodromal symptoms. Otherwise, in ED such as AN, the disembodiment is probably more related to the developmental stage of pubertal transition from childhood to adolescence and to the associated remapping of the embodiment relationship with the changing body (from the first person perspective) and the changing body image (form the third person perspective). Indeed, a recent pilot study has evidenced an increased prevalence of SD in young girls with AN (as compared with age-matched healthy controls) and highlighted the mediating role of body image distortions with respect to the relationship between Self-disorders and eating symptoms (Moccia et al., 2021). Interestingly, a similar finding is reported by a study that investigated such relationship not from the side of ED but from the side of clinical high-risk for psychosis, reporting a direct association between Self-disorders and disordered eating (Rasmussen et al., 2020).

In this perspective, only a direct comparison of SD in ED vs. schizophrenia spectrum disorders could allow a quantitative and qualitative phenomenological distinction of affinities and differences between dimensions of disembodiment in these disorders.

Conclusions

At the phenomenological level, both ED and schizophrenia are based on a core dimension of disembodiment, that is an alteration at the level of the implicit attunement with the body. This might be a result of possible distinct pathogenetic mechanisms: the process of disembodiment is probably primary in schizophrenia, being ontogenetically structured in childhood and representing the basis of prodromal positive and negative symptoms through the intermediate level of Self-disorders. This is likely to be different in AN, where the process of disembodiment could be secondary, possibly conditioned by body image and coesthetic distortions and resulting in a troubled transformation and adaptation of the previous embodied relation with the child body into the adolescent body. However, while the hypothesis on disembodiment in schizophrenia is supported by a robust amount of empirical evidence (Poletti et al., 2019), the hypothesis on disembodiment in AN (and more generally in ED) is more speculative and deserves further empirical investigation, given that childhood premorbid functioning in adolescent AN has been up to now far less investigated and characterized, especially in relation to motor development, with preliminary findings in high-risk children (e.g. offspring of mothers with ED: Kothari et al., 2014; Martini et al., 2020).

A clinical examination not limited to the more behavioral ascertainment of eating-related symptoms but including a neurodevelopmental anamnesis and a phenomenological exploration of the subjectivity in which eating symptoms are experienced, could provide key clinical elements to orient the differential diagnosis between primary ED with symptoms that may reach the psychotic threshold vs. primary psychotic disorder with a complex clinical picture including eating-related symptoms.

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