



Diagnostic uncertainty: Avoidant/restrictive food intake disorder and co-occurring psychosis in a severely malnourished adolescent male

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ABSTRACT

Diagnosing comorbid psychosis and avoidant/restrictive food intake disorder (ARFID) may be challenging among severely malnourished patients. Malnutrition is an identified medical cause of psychosis and in patients with ARFID, symptoms mirroring psychosis could be secondary to weight loss versus a “true” thought disorder. Conversely, dietary restriction has been demonstrated in patients with psychosis, which conceivably, could result in the development of ARFID. In this case report, we present a medically hospitalized adolescent male with severe malnutrition, diagnosed with ARFID after 12-months of food avoidance due to sensory sensitivities and fears of gastrointestinal discomfort. A significant decline in his emotional, interpersonal, and cognitive functioning was also reported and attributed to severe malnutrition. With nutritional rehabilitation, the patient's eating behaviors and mental state unexpectedly declined, and he was ultimately diagnosed with comorbid psychosis.

Introduction

Research demonstrates a significant association between symptoms of psychosis and disordered eating (Brodrick et al., 2020; Ganson et al., 2022; Mensi et al., 2020; Miotto et al., 2010; Solmi et al., 2018). However, prior studies have not described co-occurring psychosis and avoidant/restrictive food intake disorder (ARFID). Introduced in DSM-5 (American Psychiatric Association, 2013), ARFID is characterized by food avoidance secondary to sensory aversions (e.g., food taste, texture), fears that eating will result in aversive consequences (e.g., vomiting, choking, gastrointestinal pain), food neophobia, and/or low appetitive drive in the absence of weight and/or appearance related concerns (Brigham et al., 2018; Norris et al., 2014).

Food avoidance has also been described in the context of psychosis (Poletti et al., 2022). For example, paranoia about food contamination and/or olfactory hallucinations that make food unpalatable can trigger dietary restriction, conceivably resulting in the development of ARFID. For some, ARFID symptoms could also reflect prodromal signs of psychosis (Kelly et al., 2004), insofar as a functional decline can preempt illness onset (Seidman et al., 2010). For example, distinct sensory features have been described prior to the onset of psychosis, including greater sensory sensitivities, sensory avoidance, and reduced sensory

seeking (Parham et al., 2019) that could trigger food avoidance and a subsequent ARFID diagnosis.

Recognizing co-occurring psychosis and ARFID has unique challenges. In ARFID, severe dietary restriction can result in malnutrition and serious medical sequelae (Feillet et al., 2019; Nitsch et al., 2021). Malnutrition is an identified medical cause of psychosis with cognitive, emotional, and neurobiological changes that mirror those evidenced in clinical psychosis. For example, individuals with malnutrition and/or clinical psychosis can demonstrate withdrawal and avoidance, mood changes, and emotion dysregulation (Merritt et al., 2020), delayed processing speed, impaired reasoning and decision making (Fonville et al., 2014; Van den Eynde et al., 2012), atypical sensory processing (Saure et al., 2022), global visuospatial deficits and biased detail processing (Mountjoy et al., 2014; Solmi et al., 2018), and progressive reduction of cortical gray matter (Kerem and Katzman, 2003; Olabi et al., 2011; Van den Eynde et al., 2012). Thus, in severely malnourished individuals with ARFID, symptoms approximating psychosis may be solely conceptualized as malnutrition sequelae and in cases of “true” co-occurring psychosis, could negatively impact treatment planning and prognosis.

Herein, we present an adolescent male who was medically hospitalized for severe bradycardia in the context of ARFID. Emotional, social,

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and cognitive decline coinciding with weight loss was attributed to severe malnutrition. Upon admission, the patient reported treatment motivation and slowly increased his nutritional intake. However, with weight gain and the resolution of bradycardia, he became progressively agitated and withdrawn, with a notable regression in his eating behaviors, prompting discharge to an inpatient psychiatric facility upon stabilization. Ultimately, the patient reported imminent safety concerns and disclosed symptoms of comorbid psychosis.

Case presentation

A 16-year-old white cisgender male presented to the emergency department of a pediatric medical center in the United States for severe abdominal pain and constipation in the setting of a 22.3 kg weight loss, or 30% of his total body weight. Assessment indicated severe and critical bradycardia to 27 beats per minute, prompting admission to the pediatric intensive care unit. Elevated transaminase and prolonged coagulation were also noted. After five days, he was transferred to the hospital's adolescent medicine eating disorder service, where he received specialized, multidisciplinary care.

Upon hospitalization, the patient's diet was limited to liquids and soft foods, including cream of rice, oatmeal, bananas, apples, and a specific type of protein bar, which he heated in the microwave to soften. He had begun restricting his diet 12 months prior, when he and his immediate family (including his biological parents and fraternal twin brother) collectively started a vegetarian diet. Although his family members resumed eating meat within a few weeks, the patient described meat as "repulsive" and maintained the vegetarian diet. (Notably, there were no prior concerns with eating meat.) The next few months, he developed numerous sensory aversions, including chewing sounds and specific smells, flavors, textures, and food combinations, resulting in his avoidance of many foods and situations that involved eating. He articulated a genuine fear of eating many foods and was emphatic that his limited diet was "safer." He began experiencing gastrointestinal discomfort which further reinforced food avoidance. He became increasingly isolated, refusing to be in the company of others when they ate and refusing to eat when others were present. He reported that peers began making unkind comments about his eating behaviors, prompting distress and avoidance of social situations. (In confidence, his mother described the patient as socially hypersensitive; she believed that his peers had expressed concern about his health.)

The patient had no prior psychiatric, medical, or developmental problems. Previously described as kind and shy, the patient had become increasingly sad and irritable over the past year. Sleep difficulties were also noted. He was an above average student with no history of learning or attention problems yet in recent months, had struggled to concentrate, pay attention, and make decisions. Socially, the patient was described as an introvert who had a few friends but preferred spending time alone or with his twin brother. His family history was notably significant for obsessive compulsive disorder, depression, and schizophrenia, all in second-degree relatives. Upon admission, he presented as cachectic yet well-groomed, and appeared his stated age. He was guarded with providers, with minimal speech, limited eye contact, and blunted affect. When asked direct questions, he demonstrated a long response time which although not formally assessed, appeared consistent with cognitive processing delays. His responses were limited to "yes" or "no" and he provided minimal details to open-ended questions.

Following admission, underlying medical causes of the patient's gastrointestinal concerns and cardiac complications were ruled out. A primary diagnosis of ARFID was given, insofar as food refusal was secondary to sensory aversions and fear of anticipated gastrointestinal discomfort, in the absence of weight and/or shape concerns. Comorbid psychiatric diagnoses were tentatively ruled out in the absence of pre-morbid mental health concerns, coupled with the onset of functional impairment (e.g., emotional changes, social isolation, concentration difficulties) after he began losing weight. As such, his recent functional

decline was conceptualized as a consequence of severe malnutrition. Further, sensory aversions were tentatively classified as misophonia, a condition characterized by strong emotional reactions (e.g., irritation, disgust, anger) and autonomic arousal to specific auditory, visual, and/or sensory triggers resulting in a hyperfocus on and/or avoidance of cue-related stimuli (Swedo et al., 2022), most often developing in childhood or early adolescence (Rouw and Erfanian, 2018). The patient's experience of worsening sensory sensitivity with the progression of weight loss was also attributed to malnourishment, since atypical sensory processing and heightened sensory sensitivity/vigilance are associated with greater malnourishment (Saure et al., 2022; Zucker et al., 2013). Finally, the patient's gastrointestinal symptoms were considered secondary to dietary changes and weight loss, consistent with research highlighting this association (Norris et al., 2014).

Of note, there was family history of schizophrenia, and the medical team considered the possibility of a burgeoning psychotic illness. However, the patient's severely malnourished state precluded a valid assessment of "true" versus medically induced symptoms of psychosis. Thus, medical stabilization and nutritional rehabilitation were prioritized before psychosis could be ruled out.

The primary goal of hospitalization was the resolution of the patient's acute medical complications. Given risks for refeeding syndrome, a standard refeeding protocol was implemented upon admission, including a slowly advanced, calorie-controlled diet with intensive medical monitoring and per needed supplementation. Initial meetings with the psychologist focused on psychoeducation about ARFID, with an emphasis on nutritional rehabilitation via *volume before variety* (e.g., meeting caloric needs by consuming an adequate volume of preferred foods before increasing the variety of foods consumed; Brigham et al., 2018). Providers encouraged the patient to consume nutrition by mouth, but he was very anxious and requested that nasogastric (NG) feeds be used to supplement his oral intake. Although NG feeds can foster ongoing food avoidance in patients with ARFID and prolong hospitalization (Feillet et al., 2019; Strandjord et al., 2015), his providers lacked confidence that he could meet caloric needs orally. Given his critical medical condition, an NG tube was placed and considered a short-term, life-saving intervention.

Secondary goals of the hospitalization included: 1) weaning off NG feeds and consuming all nutritional requirements orally; 2) learning and implementing skills to manage gastrointestinal discomfort; 3) beginning exposure-based interventions to increase the variety of foods consumed; and 4) eating in the presence of others. Discharge disposition would be contingent on the patient's progress toward secondary goals, with options for outpatient psychotherapy or inpatient psychiatric treatment.

Upon NG placement, the patient consumed a small proportion of his daily calories by mouth, restricting his intake to "safe" foods like oatmeal, ice cream, and bananas. He disliked the NG tube and was motivated for its removal. With encouragement, he agreeably increased his intake of liquid supplements and "safe" foods over the first ten days of hospitalization. NG feeds were concurrently weaned and ultimately discontinued. The patient's affect brightened significantly, and he was more engaged with providers. Gastrointestinal concerns were denied during this time and the patient declined offers to learn pain management strategies for any future discomfort.

Following NG removal, the psychologist introduced exposure-based interventions, including the development of a fear hierarchy and initiating low-level exposures to prime the patient and family for future outpatient interventions. The patient lacked insight into ARFID-related consequences; he was emphatic that avoiding foods did not cause impairment and that having a very limited diet would not have future consequences. He was unable and/or unwilling to identify any potential benefits of exposures and declined participation. A reward system was implemented and per needed hydroxyzine (10 mg) was prescribed, yet the patient's resistance to exposures intensified. Whereas moderate resistance to exposure-based interventions among individuals with anxiety is common (Selles et al., 2013), the patient's objections were

markedly higher than those typically displayed in our clinical experience.

Despite these setbacks, the patient consistently met daily caloric expectations by eating and drinking – a vast accomplishment considering his behaviors upon admission. However, his persistent avoidant behaviors were extreme and severely impairing. His inability and/or unwillingness to participate in exposures while in a controlled and supportive environment (i.e., the hospital) did not bode well for successful outpatient treatment. His mother was encouraged to set clear expectations for the patient to complete exposures and to integrate feared foods into his diet. If these expectations were not met, discharge to an inpatient psychiatric program would be recommended, where the patient could receive intensive treatment. The patient expressed frustration about the plan but agreed to participate because he wanted to go home. He ultimately agreed to do an exposure of holding crunchy cereal in his hands. His mother was present during the exposure and received coaching to assist with these interventions at home. The patient appeared highly anxious during the exposure and was fidgety while holding the cereal. He reported a significant decline in anxiety over ten minutes and immediately asked the provider to leave his room when the exposure was completed.

The patient declined participation in additional exposures and was surprisingly agreeable to a psychiatric hospitalization upon medical stabilization. He acknowledged the need for additional help but was unwilling to engage in additional evidence-based treatments for anxiety during his medical stay. He became increasingly irritable and withdrawn over the next two weeks, refusing to talk with providers and declining visits with staff and family. His eating behaviors also regressed; he stopped eating “safe” foods, instead drinking liquid supplements to fulfill caloric requirements. He implored that providers place another NG tube, reporting that eating was “too hard,” yet unable to articulate why. His mental and behavioral decline were unexpected considering a 7.6 kg weight gain and adequate resolution of bradycardia. His providers began to question the presence of co-occurring psychosis, particularly given the patient’s family history of schizophrenia.

After 36-days of medical hospitalization, the patient was transferred to an inpatient adolescent psychiatric unit with specialized eating disorder treatment. He agreed to integrate new foods into his diet, reporting motivation to go home. Over nine days, he decreased his intake of liquid supplements, consumed a greater volume of “safe” foods, and added a few more foods to his repertoire. He gained an additional 3.3 kg and was discharged to outpatient cognitive behavioral therapy (CBT) with family participation.

One month after discharging home, the patient reported suicidal and homicidal ideation, visual and command auditory hallucinations, and paranoia at a weekly therapy appointment. He was readmitted to the inpatient psychiatric unit and was formally diagnosed with comorbid psychosis. These symptoms were not previously disclosed because the patient was afraid to talk about them and believed that they were his “own” thoughts.

Discussion

Accurate diagnosis is imperative for illness conceptualization, treatment planning, and optimal outcomes in psychiatric treatment. This novel case highlights the potential for symptom overlap in psychosis and ARFID and demonstrates the potential to overlook co-occurring psychosis in severely malnourished patients with ARFID. As such, it is recommended that diagnostic assessment be a dynamic process, conducted over the course of weight restoration. Malnutrition can “mask” comorbid psychopathology (Brodrick et al., 2020) and nutritional rehabilitation is required to rule out co-occurring psychosis in ARFID.

In this case, the patient’s functional decline was initially attributed to malnutrition. However, with weight gain it became increasingly evident

that these impairments were neither solely nor primarily due to significant weight loss. Upon diagnosis of comorbid psychosis, the patient’s treatment plan was shifted to prioritize evidence-based safety planning, medication management, and CBT for psychosis and ARFID.

In the described case, identification of comorbid psychosis was imperative for accurate symptom conceptualization and treatment planning. However, earlier detection of psychosis would not have altered his medical admission. For severely malnourished adolescents, evidence-based treatment necessitates that weight restoration and medical stabilization take precedence over psychotherapy for eating disorders and comorbidities (Lock and LeGrange, 2015). As such, if the patient had been diagnosed with comorbid psychosis upon hospitalization, initial interventions would have still prioritized nutritional rehabilitation and medical stabilization prior to psychotherapy. However, diagnostic conceptualization and treatment recommendations would have differed.

Conclusion

This case report highlights the potential for comorbid psychosis and ARFID. In cases of clear symptom overlap, accurate conceptualization and diagnosis may be hindered by severe malnutrition which is a medical cause of psychosis. Ongoing psychiatric assessment throughout the weight restoration process is critical for diagnostic accuracy and optimal treatment planning. Future research should explore the nuanced interplay between symptoms of psychosis and ARFID.

Consent to publish

Written consent was obtained for this manuscript from the patient and primary caregiver.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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