Disorders of gut–brain interaction, eating disorders and gastroparesis: a call for coordinated care and guidelines on nutrition support

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Untangling the complex interplay between eating disorders, gut–brain disorders and motility disorders is challenging. Nationally and internationally, there has been a concerning increase in patients receiving artificial nutrition that may be unnecessary.[1] This places patients at risk of iatrogenic harm and results in considerable economic burden to health services. There is a clear need for high quality research to guide care, but in its absence, now more than ever, we require a national treatment consensus to support clinicians working in this field. This article aims to highlight the current status, knowledge, and service limitations in treating this difficult cohort of patients, and make recommendations on future strategies within Australia to move forward for better patient outcomes.

Concerning trends

Diagnostic labels applied to patients with postprandial pain, nausea, regurgitation and satiety limiting oral intake, vary considerably and are influenced by the specialty first encountered.[2] When a formal eating disorder diagnosis is made, gastrointestinal symptoms are accepted as secondary and may go untreated, leaving patients feeling invalidated and stigmatised. In contrast, patients presenting to gastroenterologists are more likely to receive a diagnosis of a disorder of gut–brain interaction (DGBI), such as irritable bowel syndrome (IBS), functional dyspepsia, chronic nausea vomiting syndrome or gastroparesis, and the eating disorder is overlooked. Once a diagnosis of motility disorder is applied, patients may identify strongly with this, and find it difficult to accept a diagnosis of a co-existing disorder of gut–brain interaction.

In the internet era, diagnoses are also influenced by unregulated health information and social media. In an attempt to understand their condition, patients may extensively research and invest in diagnoses that are incorrect. In an attempt to help their patients, clinicians may place too great an emphasis on overarching diagnoses that are not yet well understood (eg, mast cell activation syndrome, superior mesenteric artery syndrome, median arcuate ligament syndrome, hypermobility syndromes, postural orthostatic tachycardia and autonomic neuropathy). Some such diagnoses carry greater risk than others, for instance, there are no long term studies describing the clinical outcomes of surgical intervention for superior mesenteric artery syndrome or median arcuate ligament syndrome.

Critically, there is significant overlap between functional dyspepsia and chronic nausea vomiting syndrome, eating disorders and gastroparesis, regardless of the diagnostic label. There are currently no gastrointestinal investigations that can accurately separate these diagnoses. Indeed, it is possible that these conditions represent a spectrum that cannot be separated. For example, a large American prospective cohort study defined patients with the same symptom phenotype as having either gastroparesis or functional dyspepsia based on an abnormal gastric emptying study. The study found a 37–42% crossover between groups based on changes in gastric emptying over time, and determined the two groups were clinically indistinguishable.[2] Adult-onset avoidant/restrictive food intake disorder (ARFID) is increasingly recognised as a unifying diagnosis in this cohort. Up to 77% of patients with gastroparesis met criteria for ARFID,[3] 96% of patients with an eating disorder reported functional gut symptoms,[4] and at least 20% of patients with IBS or functional dyspepsia met criteria for an eating disorder.[5,6] Recognition of a continuum of gut–brain, motility and eating disorders may prove to be more accurate.

Clinicians are increasingly faced with challenging decisions on artificial feeding, including pressure from desperate patients and families to provide invasive feeding for symptom relief, despite a lack of evidence of long term benefit.[7,8] In a diagnosed eating disorder, including ARFID, oral intake is mandated and symptoms managed conservatively with the expectation that symptoms will improve with nutritional rehabilitation. In contrast, despite the overlap in phenotypes, for patients diagnosed with either gastroparesis or DGBI with malnutrition, artificial feeding is often introduced sooner. If symptoms persist, tube feeding may be escalated to parenteral nutrition, which carries even greater risk of morbidity and mortality.[1] Given the risks of iatrogenic harm, best practice requires careful consideration by a multidisciplinary team before commencing artificial nutrition,[9] notably difficult to access in regional and private facilities. Current guidelines lack recommendations appropriate for patients with overlapping eating disorder and IBS or functional dyspepsia diagnoses,[10] and no Australian guidelines exist to guide best practice.

Pathophysiology: current knowledge and advances

In patients with both IBS or functional dyspepsia and eating disorder symptoms, whether the disordered eating is causal or a secondary attempt to avoid symptoms can be unclear. Often both are present in a bidirectional manner. Although altered interoception is a driver of symptoms in eating disorders[11] and

DGBI, motility changes also contribute to symptom burden. Studies show that up to 50% of patients with anorexia nervosa have delayed gastric emptying[4] and two-thirds have delayed colonic transit.[12] Anorexia nervosa is associated with gastric dilation in up to 82% of patients.[13] Reassuringly, there are data that suggest that motility changes improve following refeeding, although full resolution of gastrointestinal symptoms is more variable.[14,15]

There is also evidence for significant dysbiosis of the gastrointestinal microbiota in patients with an eating disorder, which may correlate with the severity of gastrointestinal symptoms. The luminal microbiome is directly affected by dietary composition, including content restriction and starvation of any cause. In eating disorders, nutritional, weight and hormonal recovery are associated with improvement, but not full restoration, of dysbiosis at one year after admission.[16]

We are progressing in our understanding of the impact of psychological stress on the mucosal immune system and enteric nervous system.[17] This may prove to be an aetiological link common to eating disorders, DGBI and motility disorders. The connection between psychological comorbidity and both DGBI and eating disorders is well established, as is the link between chronic stress and immune dysregulation. For example, corticotrophin-releasing hormone triggers mast cell degranulation,[18] immune mediators then contribute to visceral hypersensitivity through nociceptor sensitisation and mucosal barrier dysfunction. More research is needed on this topic in relation to eating disorders. Although advancing, we are in our infancy in understanding these complex interactions and how they may translate to treatments.

The need for cross-disciplinary research, training and treatment

The management of patients with complex psychological and physiological conditions requires intensive multidisciplinary care. Unfortunately, the historic divide between medicine and psychiatry underpins a lack of cross-disciplinary research, training and treatment services. The deficit starts at a training level. DGBIs and eating disorders are undertaught across mental health, medical, surgical and allied health professions,[19,20] often with minimal or no clinical exposure, and few formal training positions in Australia. This leads to a self-perpetuating lack of confidence and interest in senior clinicians treating these disorders outside of specialist services. This is compounded by hospital funding models that are designed predominantly for acute care. Eating disorders and DGBI are better treated in the community, where physicians and surgeons are not publicly funded, therefore limiting affordable multidisciplinary care where it is needed most. As a vulnerable patient group, research is limited, but the consequence is that we are without high quality data to advance our understanding, improve treatment models, and reduce harm. There is a clear need for nationwide dedicated multidisciplinary services, ideally embedded within community eating disorder programs, incorporating consultation–liaison psychiatry, eating disorder clinicians (psychiatry, psychology, dietetics), gastroenterology and surgical specialties.

In the clinical setting, distinguishing the predominant pathology and treatment priority is key to determining the treatment plan. Eating disorder treatment programs historically are strictly regimented, not incorporating investigations or treatments for DGBI symptoms, for the reasons stated above. Conversely within medical models, if a primary or comorbid eating disorder diagnosis is missed, there is an increased risk of illness-perpetuating treatments. The therapeutic approaches for eating disorder and DGBI may also directly contradict each other, such as elimination diets in DGBI versus oral graded exposure therapy in restrictive eating disorders.[5] Within DGBI services, mental health clinicians are hugely under-resourced, despite the known burden of psychiatric comorbidity and need for psychiatrist-guided psychotropic medications in complex cases.[21] In the absence of guidelines that incorporate eating disorder and DGBI care, treatment approaches, even within individual units, are inconsistent, causing confusion and distress for patients and increasing risk of harm.

Future perspectives: a call for better patient care

This article highlights the continuing gaps in cross-disciplinary training, research and publicly funded treatment services despite an increasing cohort of patients with overlapping eating, gut–brain, and motility disorders. Sufferers compose a vulnerable population exposed to significant iatrogenic harm at high economic cost. At the coal face, it is clear that no one clinician can do it all. To ensure patient safety, we call for multidisciplinary team review to become the standard of care before commencing artificial nutritional support. Invasive investigations should only be undertaken if they will result in a clear positive change in management. Nutritional support should use the least invasive feeding route possible, with management encompassing the biopsychosocial model of care.

At a health service level, a review of public hospital and community health funding is required. Improved exposure and training in gut–brain and eating disorders would directly and positively affect patient outcomes. Careful multidisciplinary research is needed to address the gap in cross-sectional data. Finally, we urgently call for the formation of a national working group to provide a consensus document to guide consistent treatment of these individuals, relevant to the Australian population.

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