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Panic Disorder Subtype Gastrointestinal Response: Phenomenon and Treatment Recommendations

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While research has shown strong associations between Panic Disorder and symptoms of gastrointestinal distress, there is a dearth of evidence on limited symptom Panic Disorder with the primary symptom of gastrointestinal distress. Most published studies have been single case studies or small case series, and proper classification remains unclear. Although a formal diagnosis does not exist for this presentation, this paper proposes the diagnosis of Panic Disorder (PD) Subtype Gastrointestinal (GI) Response. This particular cluster of symptoms currently creates a diagnostic conundrum, which can directly affect treatment options. This paper explores the relationship between Panic Disorder and gastrointestinal distress, and discusses the challenge of differential diagnosis among disorders with considerable symptomological overlap (e.g., Obsessive Compulsive Disorder, Agoraphobia, and Irritable Bowel Syndrome). Theories such as the brain-gut loop and cognitive theory are discussed to explain how the interaction of cognitions and physiological reactivity maintain and exacerbate the proposed disorder. Finally, specific treatment recommendations and behavioral assessment methods are provided.

Panic Disorder (PD) has been conceptualized as a multiple symptom disorder by the American Psychiatric Association (DSM-IV-TR, 2000). However, limited symptom PD (i.e., one symptom cluster predominates) confounds the categorization and treatment of PD. Gastrointestinal (GI) distress and PD overlap considerably, yet there is little research on limited symptom PD with the primary symptom of GI distress. The current article explores the relationship between the two conditions, proposes the diagnosis of PD subtype Gastrointestinal Response as a more accurate classification of this specific symptom presentation, and offers appropriate treatment recommendations.

The criteria for PD includes recurrent, unexpected panic attacks accompanied with four or more of the 13 physiological symptoms (e.g., pounding heart, sweating, trembling, nausea or abdominal distress) and cognitive symptoms (e.g., fear of dying, and fear of losing control) which peak within 10 minutes (DSM-IV-TR, 2000). An unexpected panic attack is defined as one that an individual does not immediately associate with a situational trigger (i.e., it is perceived as occurring "out of the blue"). Additionally, at least one of the following must be experienced for one month following an attack: 1) persistent concern about having additional attacks; 2) worry about the implications of the attack or its consequences; and/or 3) a significant change in behavior related to the attacks. In some cases, patients report experiencing only one or two of these symptoms during a panic attack. Symptom specific presentations of panic (e.g., cardiovascular and respiratory systems, and GI distress) that do not meet full criteria for PD have been referred to as a limited symptom panic attack (Lelliott & Bass, 1990; Rosqvist, 2005).

PD accounts for approximately 10% of individuals referred for mental health consultation. PD is often accompanied by Agoraphobia, characterized by anxiety about being in situations perceived as inescapable; thus 'unsafe' places are often avoided (DSM-IV-TR, 2000). Approximately one-third to one-half of individuals diagnosed with PD in community samples also suffer from Agoraphobia (DSM-IV-TR, 2000).

Panic Disorder Subtype Gastrointestinal Response

Case studies and other research document PD in terms of limited symptom attacks and characterize GI distress as a limited symptom PD (Eldridge, Walker, & Holborn, 1993; Hatch, 1997; Lelliott & Bass, 1990). Individuals from select case studies report debilitating symptoms such as abdominal distress, heart palpitations, hot flashes, sweating, and shaking (Eldridge et al., 1993; Hatch, 1997). Ruminating on possible fecal incontinence (e.g., "if a toilet is not nearby, I will have an accident") or spending excessive time contemplating the location of toilets in public is also reportedly common (Hatch, 1997). Behavioral changes range from spending an increased amount of time in bathrooms and modifying diets by

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eliminating certain foods, to eliminating all food intake prior to leaving home or avoiding leaving home altogether (Eldridge et al., 1993; Hatch, 1997).

For example, Eldridge et al. (1993) illustrated the suffering endured by one patient. The patient avoided all public situations where she might feel embarrassed by the length of time she spent using the toilet. She was unable to work, ride public transportation, or wait in a line. She also reported distress while waiting for a phone call or a house visitor for fear of experiencing sudden diarrhea. The patient refrained from eating all together on days when she would need to leave her house. Her catastrophic misinterpretations of her GI sensations reinforced her need to avoid or escape certain situations. Furthermore, her worries were confirmed by evidence that, when she did use the toilet, she experienced diarrhea. In sum, physiological sensations trigger cognitive distortions (e.g., catastrophic thinking) and avoidance behaviors, resulting in impaired daily functioning due to persistent avoidance of situations where access to a toilet is limited (Eldridge et al., 1993; Hatch, 1997).

However, research on this phenomenon is minimal, and likewise, data on prevalence, gender and age differences, and diagnostic criteria remains largely unknown. In addition, the literature indicates an underreporting of PDs involving GI functioning, which Hatch (1997) ascribes to societal influences that deem speaking of bodily functions, even to medical and health professionals, as improper. As a result of social norms, individuals suffering from irregular bowel functions and fear of incontinence may feel uncomfortable discussing personal bowel functions. Due to the lack of a formalized classification and dearth of research, this presentation remains difficult to diagnose and treat.

To better understand the mechanisms governing this particular cluster of symptoms and to make a case for a new diagnosis, we compare existing diagnoses to the proposed diagnosis of as PD Subtype GI Response. Specifically, we discuss psychiatric and medical diagnoses that report manifestations of GI symptoms that may be related to Panic Symptoms: bowel obsessions (a variant of Obsessive-Compulsive Disorder), Agoraphobia without a history of PD, and Irritable Bowel Syndrome (IBS). We then consider brain-gut interaction theory, cognitive theory, and extant treatment strategies used for IBS, OCD, and PD to inform treatment recommendations for the proposed diagnosis of PD subtype GI response.

Bowel Obsessions

Bowel Obsession Syndrome (BOS) has a similar presentation to PD subtype GI response and has been researched and proposed as a variant of Obsessive-Compulsive Disorder (OCD; Hatch, 1997). BOS is characterized by excessive worry about fecal incontinence and compulsive behaviors of evacuation checking. Secondary symptoms may include fears of social judgment and inaccessibility of bathrooms when not at home. Individuals who experience these cognitions also engage in behaviors directed at controlling their bodily functions, such as spending considerable amounts of time in the bathroom as well as limiting food intake (Hatch, 1997).

Debate exists over the diagnosis of BOS. Some research proposes that bowel obsessions would be better conceptualized as an anxiety disorder, such as PD, due to the inconsistency of OCD patients' response to pharmacological treatment (Hatch, 1997). The following study highlights the lack of consensus regarding the diagnosis of BOS. Hatch (1997) argued that if OCD and BOS shared a symptom profile, individuals suffering from bowel obsessions would score highly on obsessive thinking on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS). However, Hatch (1997) showed in two case studies that both patients with bowel obsessions at pre-treatment baseline scored lower on the (Y-BOCS) than classic OCD patients. This suggests that if conceptualized as an OCD variant, bowel obsessions would present within the mild range of severity. Due to the debilitating effects of bowel obsessions, it seems disproportionate to diagnose this syndrome as of mild severity.

Treatment implemented by Hatch (1997) included cognitive-behavioral interventions, such as cognitive restructuring and in-vivo exposures, which resulted in significant symptom reduction of bowel obsessions. Given the positive response to cognitive-behavioral treatment strategies among BOS patients, and the overlapping symptomotology of BOS and the proposed PD subtype GI response, we suggest that individuals suffering from PD subtype GI response would benefit from a similar treatment protocol as those with BOS.

Agoraphobia Without a History of Panic Disorder

The DSM-IV-TR (2000) describes Agoraphobia without a History of PD as the presence of Agoraphobia related to fear of developing panic-like symptoms (e.g., dizziness or diarrhea) and that criterion have never been met for PD. The fear of fecal incontinence while in public and the fear that access to a bathroom may be limited, overlap with the aforementioned symptoms of BOS and PD, and may contribute to certain agoraphobic conditions. While Agoraphobia without a History of PD has been questioned as a legitimate diagnosis separate from PD, two models have been proposed. The first model describes the development of Agoraphobia as a sequelae of PD and the second model suggests that Agoraphobia is a conditioned avoidance response from the pairing of situations (e.g., driving, traffic, shopping) to noxious experiences of panic or fear of vomiting, panicking, or bowel incontinence (DSM-IV-TR, 2000; Goisman et al., 1995). Goisman et al. (1995) found that individuals who met criteria for Agoraphobia without a History of PD had experienced limited symptom attacks that narrowly missed the criteria for PD with Agoraphobia, suggesting that both can be seen

on the same continuum. Goisman et al. (1995) also found that a high number of individuals with Agoraphobia reported catastrophic cognitions associated with their disorder, the most common of which were *1)* fear of doing something embarrassing (*n* = 11, 25%), *2)* fear of fainting (*n* = 8, 18%), *3)* fear of losing control (*n* = 7, 16%), and *4)* fear of becoming ill (*n* = 7, 16%; Goismann et al., 1995).

Irritable Bowel Syndrome

Drossman, Li, Andruzzi, Temple, and Talley (1993) reported that functional GI disorders effects 69% of the United States population with 40% complaining of bowel distress. Of these sufferers, 8% - 17% experience debilitating effects of the disorder. Moreover, research indicates that many individuals who seek treatment for IBS have a psychiatric history (Lydiard & Falsetti, 1999).

Taylor (2000) speculated that PD presents as one psychiatric disorder often comorbid with IBS because IBS symptoms conceivably include undesirable body sensations that can easily lead to misappraisals. Because those with IBS are often hypervigilant of bowel symptoms, any bowel sensation (even those that are benign) could be interpreted as an indication of possible loss of bowel control. This catastrophic thinking of possible 'loss of control' could then trigger a panic attack.

To determine the link between PD and GI symptoms, Lydiard et al. (1994) conducted a study among a community-based sample consisting of 13,537 participants. Four groups were created from this random probability sample: (a) individuals diagnosed with PD at any time in their lives, (b) participants diagnosed with any other anxiety disorder, (c) individuals diagnosed with any other major psychiatric disorder, and (d) those who did not meet qualifications for any DSM-III classified disorder. The results of this study showed that participants with PD reported experiencing GI symptoms at a higher frequency than the other groups. Symptoms of diarrhea were reported by 12.9% of individuals with PD as compared to 2.9% of those without a psychiatric diagnosis.

Additionally, Lydiard and colleagues (1994) found evidence to suggest that, contrary to previous research on treatment-seeking populations, PD and IBS symptoms do in fact co-exist. This diagnostic overlap generalizes the co-occurrence beyond treatment-seeking populations and speaks to the challenge of differential diagnosis.

Brain-gut Interaction and Cognitive Theory

The enteric nervous system (ENS), which regulates behaviors, such as bowel performance, has been compared to the brain in its organizational and functional structure. Similar to the brain, which processes external stimuli transmitted to the central nervous system (CNS) through the dorsal root and cranial nerve ganglion cells to control behaviors, the ENS responds to stimuli signaled through intrinsic primary afferent neurons (IPANS). Although both systems can work independently of one another, they generally work in tandem. The CNS and ENS both rely on receiving information from the bowel to make informed decisions (Gershon, 2005). Lydiard and Falsetti (1999) postulate that by understanding the relationship between the CNS and ENS, explanations for GI distress may be established. They propose a brain-gut loop model to explain the positive feedback cycle that occurs in IBS patients. This model suggests that when aversive stimuli are detected by the gut, the locus ceruleus (LC), a CNS noradrenergic nucleus that mediates fear and arousal states is activated. Activation of the LC leads to activation of the CNS, sending messages to CNS fear and arousal-mediating components, such as the amygdala and medial hypothalamus (Coplan & Lydiard, 1998). Given the interaction between the CNS and ENS, individuals with increased arousal (or hypervigilance) could experience GI distress due to the increased CNS sympathetic outflow and responsive input to the LC creating a positive feedback loop (Lydiard & Falsetti, 1999). This brain-gut loop explains how hypervigilance and increased sensitivity to aversive stimuli is reinforced by neurochemical feedback sent to the brain by way of the CNS and ENS, thus validating irrational or negative cognitions associated with sensations experienced during high arousal states.

Cognitive theory, as described by Chambless et al. (2000), conceptualizes panic using a similar feedback loop. When a person grossly misinterprets somatic sensations, anxiety levels are increased. This, in turn, heightens sensitivity to subsequent bodily sensations creating an experience of panic. The negative cognitions and feared bodily sensations interplay with one another to create this positive feedback loop of heightened anxiety followed by heightened sensitivity. Understanding the brain-gut interaction and cognitive processes is crucial to informing appropriate treatment interventions focusing on GI-related cognitive distortions and obsessive thinking.

Many individuals who experience GI distress seek relief through pharmacological interventions. For example, Sandler (1990) reported that over two million prescriptions for IBS are written per year in the United States. Masand and colleagues (2002) studied treatment effects of paroxetine, a commonly prescribed selective serotonin reuptake inhibitor (SSRI), in two groups of IBS patients: (a) 10 patients with coexisting anxiety disorders (Specific Phobia, PD, and Social Phobia as indicated by the Structured Clinical Interview [SCID]), and (b) 10 patients with no anxiety disorder diagnosis. Both groups of patients received 12 weeks of paroxetine at a mean dose of 31 mg/day. Results showed that seven patients with anxiety disorders reported a 70% or greater improvement in abdominal pain, versus 20% of the non-anxiety disorder group. Diarrhea frequency and severity decreased in 71% of anxiety patients versus 43% of the non-anxiety patients. These results may be due to the effects of psychotropic medication on the interplay between the CNS and ENS,

particularly for those with co-existing anxiety disorders (Masand et al., 2002).

Treatment Recommendations

While cognitive-behavioral treatment of PD has been studied extensively, specific examination of GI symptoms is less widespread (Barlow, Gorman, Shear & Woods, 2000; Landon & Barlow, 2004). A relevant study by Lelliot and Bass (1990) compared the subjective and physiological responses to imaginal exposure and voluntary hyperventilation of two groups of patients with PD. One group was classified as experiencing cardiovascular and respiratory (CR) symptoms of panic (e.g., shortness of breath, smothering sensations, and chest pain), while the second group primarily reported GI symptoms (e.g., abdominal discomfort, diarrhea, and fear of fecal incontinence). The findings indicated that the GI group experienced significantly less distress than the CR group. The authors concluded that because the hyperventilation exposure induced sensations similar to those experienced during a panic attack for the CR patients, they were more likely to produce distress in the CR group. This finding suggests that PD is heterogeneous in presentation and effective treatment is dependent upon on accurate targeting of the specific symptoms experienced.

Thus, designing an effective treatment for PD subtype GI response would require use of interoceptive exposures that induce symptoms of GI distress. Chambless et al. (2000) endorsed this methodology asserting that Cognitive-Behavior Therapy (CBT) approaches for inducing panic-like experiences should be targeted to induce the symptoms experienced by each patient. As Andrews et al. (2003) suggest, in order to improve treatment outcome, exposures should closely resemble the actual feared situations, as well as aim to modify maladaptive cognitions.

Exposure therapy prompts change by challenging the inappropriate anxiety response that has been conditioned to produce or elicit fear. A reduction in anxiety occurs by re-experiencing the feared stimuli (e.g., limited access to bathroom, travel, and traffic) and associated catastrophic cognitions (e.g., " I can't hold it," "people will see," and "I will make a fool of myself") without actual threat (e.g., bowel incontinence; Rosqvist, 2005). In order for new non-threatening conditioning to occur, an individual must first habituate to the perceived threatening stimulus. Habituation occurs when a person does not flee or avoid anxiety-provoking situations, but remains present. Remaining present despite a perceived threat provides the body with a new experience (i.e., new physiological evidence) that actual threat or danger is not occurring and anxiety responses begin to diminish (Foa & McNally, 1996).

Based on the literature reviewed herein, we recommend a specialized CBT treatment strategy for PD subtype GI response that includes graded exposures to GI-related somatic sensations in addition to psycho-education about anxiety, somatic skills training (e.g., utilizing diaphragmatic breathing), and cognitive restructuring (e.g., distorted versus objective thinking). Additionally, we suggest integrating bowel control training (e.g., distinguishing normal abdominal sensations from bowel distention), as well as utilizing in-vivo exposures to challenge agoraphobic avoidance and escape behaviors (e.g., eating in public, going places where restroom access is limited).

Despite the diagnostic challenges the proposed symptom presentations create, recommended intervention strategies would reflect those most commonly used to treat PD, OCD, and IBS. Treatment for IBS, for example, mimics similar components used to treat other anxiety disorders (e.g., muscle relaxation, diaphragmatic breathing, activity scheduling) and would, likewise, be presumed effective for PD subtype GI response. In addition, interoceptive and in-vivo exposure, both established treatments for PD and OCD (Barlow, 2008), would also likely be effective with GI-related symptoms.

Assessment Strategies

To effectively administer and monitor the proposed treatment, psychometrically sound assessment tools are required. Cognitive, physiological, and motor information can be gained through self-report, in addition to diagnostic clinical interviews. We recommend the Anxiety Sensitivity Profile (ASP) for cognitive aspects of the treatment and the Body Vigilance Scale (BVS) for the physiological symptoms (Schmidt, Lerew, & Trarkowski, 1997; Taylor & Cox, 1998). In addition to these formal self-report measures, behaviors can be monitored through self-report measures such as a daily log to record one's eating schedule or agoraphobic exposure experiments.

The ASP measures cognitions of the perceived danger associated with the anxiety symptoms. The questionnaire consists of 60 questions that list specific bodily sensations and asks the respondent to answer how likely it is that each sensation would lead to something bad happening (e.g., "going crazy" or dying). It is divided into six subscales that assess the amount of fear associated with a particular group of experiences: cardiovascular, respiratory, gastrointestinal, observable, neurological, and cognitive dyscontrol. The ASP takes approximately 10 minutes to complete. While normative data is not yet available, the reliability found within a group of university students was good with an internal consistency for GI symptoms of 0.88 (Taylor & Cox, 1998).

The BVS consists of a four item self-report inventory. Each item uses an 11 point Likert scale. The first three questions measure the degree of focus given to internal physiological stimuli. The fourth item lists 15 bodily sensations, as listed in the DSM-IV for panic attacks, and assesses how much attention is given to each sensation. The BVS takes 3 to 5 minutes to complete. The BVS has shown good internal consistency (Cronbach's alpha = .82;

Schmidt et al., 1997). Validity of this measure has been studied and found to be supported; patients with PD have been found to have higher scores on the BVS pre-treatment than patients of social phobia or nonclinical trials, and scores on the BVS tend to decrease following cognitive-behavioral treatment (Schmidt et al., 1997).

Utilization of the aforementioned narrow-band measures allows for proper assessment of physiological and cognitive symptoms indicative of PD subtype GI response. Such detailed assessment is necessary for accurate conceptualization and treatment planning, while also providing an opportunity for objective symptom tracking throughout the treatment process.

Conclusion

PD with GI response represents a rare but significant form of the disorder, where in the person does not experience the full range of conventional symptoms associated with a panic attack. Instead, the single symptom of GI reactivity predominates. While many panic attack sufferers typically experience automatic thoughts (e.g., "I'm having a heart attack!" and "Is this a stroke?") that are often dispelled by an emergency room visit, the GI distress symptom can present with an actual reality of fecal incontinence. For those who experience PD subtype GI response, this misappraisal fundamentally changes from a perceived threat to something that has additional social implications.

When considering a PD subtype GI response diagnosis, it is necessary to consider and rule out that the particular GI focus is not better explained by another psychological disorder, such as Agoraphobia or IBS. Therefore, careful diagnostic interviewing is critical. To this end, relying on such diagnostic tools as The Anxiety Disorders Interview Schedule – Fourth Revision (ADIS-IV, 2007), medical records, previous treatment records, and collateral information may aide in accurately diagnosing this type of PD vis-à-vis other conditions that GI problems could represent.

Due to its unique presentation, PD subtype GI response needs a tailored treatment strategy. While potentially capitalizing on medications (e.g., Paroxetine) to reduce sheer gut reactivity, the multi-pronged treatment recommendations include many conventional panic control treatment strategies (e.g., psycho-education, cognitive restructuring), while including an emphasis on bowel control training and in-vivo exposure to feared situations that are related to anticipated bowel problems (e.g., eating in public, taking longer trips while snacking). This strategic approach takes advantage of learning theory and conditioning paradigms to ensure that treatment gains generalize to various natural environments.

In conclusion, it remains largely true that the brain-gut relationship is not yet well understood, especially in regards to PD subtype GI response. More research is needed to better grasp how to specifically tailor treatment to accommodate the pragmatic addition of this particular symptom to any panic presentation. Additionally, it remains to be determined whether PD subtype GI response constitutes PD per se, or if this unique presentation warrants its own diagnostic category. Future research is needed to further understand the nature and consequences of this distressing problem.

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