Psychological abnormalities in patients with irritable bowel syndrome

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Irritable bowel syndrome (IBS) is a group of functional bowel disorders with different pathophysiological mechanisms but some common clinical features. It can be conceptualized within the biopsychosocial model of illness as a dysregulation of brain-gut axis and its relationships with psychosocial and environmental variables. Using advanced neuro-imaging techniques, it has been found that some brain centers (anterior cingulate cortex, limbic system, locus ceruleus) are active in mediating gut signals and that visceral hyperalgesia mediates perceptual sensitivity. Using new criteria for diagnosing psychosocial components of somatic illnesses, persistent somatization has been found as one of the main psychological factors that contributes to persistence of symptoms and poor treatment outcome in patients with IBS. Other psychological variables influencing symptom reporting have been identified in the constructs of health-care seeking, abuse, somatosensoy amplification, and alexithymia. From a psychological viewpoint, IBS may be perceived as an abnormal cognitive processing of emotional and visceral stimuli, a tendency to perceive somatic stimuli as evidence of symptoms of disease, and to seek repeated and often unnecessary medical care. [Indian J Gastroenterol 2004;23:63-69]

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IBS challenges the basic assumption of the biomedical model of Western medicine, according to which diseases are caused by identifiable abnormalities in the structure and function of organs and tissues that in turn have a causal relationship to the individual’s perception of ill health. However, this kind of correlation between disease and subjective perception of symptom does not seem to fit with IBS symptoms. In the biopsychosocial model, illness is viewed as a multifactorial entity resulting from interaction of systems at cellular, tissue, organism, interpersonal and environmental levels. Psychosocial stress may therefore exacerbate GI symptoms and modify the experience of illness and illness behavior such as health-care seeking. In turn, chronic GI disorders may have psychosocial consequences on one’s general well-being and daily functioning.

IBS can be conceived as a biopsychosocial disorder resulting from interaction of multiple systems such as the central nervous system (CNS), psychological factors, altered intestinal motility, and increased sensitivity of the intestine. In such interacting systems, events do not occur in isolation. For example, activated immunocompetent cells in the intestinal mucosa have been found to be increased in patients with IBS. Psychosocial stressors may modulate the immune response of the gut to infectious agents, and in turn, gut-directed physiological stressors may modulate the responsiveness of the CNS. Altered outputs of central stress circuits, such as the hypothalamus-pituitary-adrenal (HPA) axis and the sympathetic arousal of the autonomous nervous system (ANS), may alter the gut immune system and increase intestinal permeability, thereby increasing susceptibility to inflammatory agents in the gut lumen. The vulnerability to psychological problems or the development of gut dysfunction may be determined by the alteration of the brain-gut axis and/or stressful events.
or genetic and environmental (from major loss to abuse history to exposure to infections) factors in early life.12

**Neuro-psychological correlates of IBS**

**Brain-gut axis**

The GI tract is controlled by a complex network, including the ANS, the CNS, and the enteric nervous system (ENS) that interacts to establish a bidirectional communication between the brain and the gut, the so-called ‘brain-gut axis’.

Numerous neurotransmitters and neuropeptides are common to the ENS and the CNS. For example, the role played by serotonin (5-hydroxytryptamine)3,5,6 in the pathophysiology of IBS has become one of the focal points of attention for therapeutic intervention in IBS.2,18,19

A new unifying hypothesis is that the symptoms of IBS result not just from afferent sensory signals arising from a disordered gut, but also from dysregulation of the complex brain-gut axis. Since neurotransmitters are not site-specific, dysregulation may occur at any level in the brain-gut axis with other physiological (endocrine and immune function) and psychosocial (behavior, personality, cognition, emotional arousal) systems.20 'Up-bottom' signals from the brain to the gut assure that the digestive function is optimized by modulating motility, secretion, immune function, and blood flow.21,22 For instance, it was found that the primary motor cortex includes bilateral representation of the anal sphincter and pelvic floor muscles and transcranial magnetic stimulation can induce changes in anorectal function.24

'Bottom-up' signals from the gut to the brain also play a role in reflex regulation.25 For instance, studies using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) showed that patients with IBS had greater activation of the anterior cingulate cortex (ACC) and thalamus with painful distention than with nonpainful stimulation, unlike control subjects.26 ACC hyperreactivity to visceral stimulation could explain the heightened pain sensitivity as well as altered descending reflexes that stimulate GI motility. Subjects who by hypnotic suggestion perceived the experience as unpleasant had increased ACC activity on PET imaging compared to subjects who were given the suggestion that it was pleasant.27

**Visceral hyperalgesia**

There is a lower pain threshold perception during rectal distension, or 'visceral hyperalgesia', in IBS patients.28 Stress, anxiety, or recall of aversive memories can enhance perception of painful stimuli, whereas distraction and relaxation can decrease it.29

Overall findings suggest that a central component, hypervigilance, influenced by non-sensory factors such as fear and anticipation, may play a prominent role in visceral hyperalgesia.30 Patients with ulcerative colitis had higher discomfort threshold and lower unpleasantness self-ratings of rectosigmoid distension, and enhanced tolerance of high-intensity sigmoid distension, than IBS patients.31 IBS patients also have lower threshold to extra-intestinal painful stimuli,32 higher activity in frontal brain areas in response to auditory stimuli,33 and greater extra-intestinal symptoms.34

**Psychological correlates of IBS**

**Psychiatric comorbidity**

The prevalence of psychiatric diagnoses in IBS ranges between 40% and 70% depending on the population, settings, and diagnostic criteria.35,36 Complaints of functional bowel symptoms have been also found in 30–70% of patients with psychiatric disorders.37–39 We found that 65.5% of patients with FGID also had a psychiatric diagnosis, and 48% of psychiatric patients had a functional disorder of the gut (55% of whom had IBS).40 Since IBS patients referred to tertiary care are more psychologically disturbed than those referred to primary care,41 the close association between IBS and psychopathology is seen as related to the patient's illness behavior (health-care seeking) rather than a characteristic of the bowel illness per se.3

By using a diagnostic classification focused on psychosomatic conditions,42 IBS patients were found to show high prevalence of alexithymia (47%), persistent somatization (34%), and somatic symptoms secondary to a psychiatric condition (30%).43 Alexithymia (82.2%) and persistent somatization (72.5%) were more frequent after 6 months of treatment among non-responders.44 In addition, alexithymia and persistent somatization independently predicted improvement.

The concept of persistent somatization derives from the hypothesis that it may be clinically advantageous to define a somatizing patient as someone in whom psychological symptoms have clustered.45 Persistent somatization as an independent predictor of failure of improvement in IBS symptom is consistent with the psychological profile of individuals with high dissatisfaction with their health, multiple and long-standing physical symptoms, low pain threshold, and somatic amplification leading to increased symptom report, high disease conviction, and therefore low ability to subjectively perceive symptom reduction with treatment.46

Somatization is not a psychiatric disorder but a cognitive process related to the way in which individuals perceive their somatic sensations as signs of medical illness, and so seek medical consultation.47 The psychological heterogeneity of patients with IBS may explain puzzling treatment outcomes. No single therapy for IBS is superior to others, and Chinese herbal medi-
Psychosocial mediators

Certain individuals may be more prone to experience chronic somatization with acute bowel symptoms and medically unexplained symptoms in other body sites in different periods ("social" or "chronic somatizers").

Indirect evidence of the stability of IBS as a psychophysiological process comes from sparse findings. For example, demoralization was the first-order dimension in a latent trait analysis of a wide range of symptoms in somatizing patients. Two studies on patients with medically unexplained symptoms found that a general somatization latent factor accounted for up to 68% of the symptoms, and that IBS symptom clustering was a second-order variable distinct from other functional non-GI disorders. A recent meta-analysis found that IBS symptoms related to (but not fully dependent on) depression and anxiety. In addition, IBS symptoms may change their clinical manifestation, as shown by patients with anorexia and bulimia nervosa who later developed functional gut disorders and cholecystectomized patients with persistent functional bowel symptoms.

Health-care seeking behavior

Several epidemiological studies suggest that people with IBS symptoms who do not consult a physician are psychologically similar to the asymptomatic population. In contrast, subjects with IBS symptoms who seek health care have more anxiety, depression, health anxiety, hypochondriasis, and are less likely to see a link between stress and their symptoms. The prevalence of these disturbances is greater in tertiary-care settings.

These data suggest that psychological symptoms are not associated with IBS per se but to the decision to consult a physician. A recent study showed that depression and conviction about disease were predictors of the frequency of physician consultations.

Sexual and physical abuse

In the last decade, several studies from the United States and Europe have found a higher prevalence of self-reported sexual and physical abuse in IBS patients (up to 67% in the US and 32% in Europe). These suggest that sexual abuse contributes to the development of IBS, perhaps through down-regulation of visceral sensitivity threshold, guilt, maladaptive adjustment, and hypervigilance to illness complaints in early life. However, numerous methodological problems have been highlighted in such research. In a population-based survey, psychological morbidity was associated with both childhood abuse and IBS, but abuse was not associated with IBS after controlling for psychological morbidity. The link between abuse and IBS may be therefore mediated by neuroticism, a personality trait characterized by exaggerated responsiveness to psychological changes. In fact, childhood abuse was found to be associated with higher levels of current psychological distress, irrespective of having IBS, idiopathic constipation, or Crohn's disease, and increased risk for developing a wide range of psychopathology.

It is likely that sexual abuse is not a cause of IBS. Consistent with the somatization construct, abuse may be better conceived as associated to the tendency to communicate psychological distress through somatic symptoms. In fact, longitudinal data showed that chronic highly threatening stressors predicted IBS symptom intensity and clinical outcome, while not the reverse.

Somatosensory amplification

Visceral hypersensitivity is conceptually close to the construct of "somatosensory amplification", that is an amplifying perceptual style entailing a hypervigilance or heightened attentional focus on bodily sensations, and a propensity for responding to them with affect and cognitions that make them more disturbing and intense. Cognitive attribution of symptoms to somatic rather than psychological causes was greater in patients with chronic functional medical disorders, while the opposite was true in patients with psychiatric disorders.

Cognitive-behavioral studies showed that patients with IBS have a self-schema characterized by social desirability. They may not identify their gut sensations in psychological terms since it is more socially desirable to have physical rather than psychological problems. The somatoform amplification construct is cross-culturally reliable; in a recent Indian study outpatients with somatoform disorders showed higher somatic attribution and mean somatic amplification scores than depressed patients (who had higher psychological attribution score), who in turn had higher scores than normals.

Alexithymia

The alexithymia ("no words for feelings") construct is defined as a cluster of cognitive and affective characteristics including difficulty identifying and communicating feelings, trouble distinguishing between feelings and somatic sensations of emotional arousal, impoverished and restrictive imaginative life, and a concrete and reality-oriented thinking style. Furthermore, it is conceived as personality trait of individuals with higher vulnerability to suffer from somatic as well as psychopathological disorders of affect regulation. We found that patients with IBS had significantly higher alexithymia than patients with inflammatory bowel disease and healthy subjects, with a prevalence of 47% to 66%.

The role played by alexithymia in mediating illness experience of IBS is intriguing. One hundred and twelve patients with PGID (half of whom had IBS) were treated...
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‘as usual’ for 6 months and evaluated with reliable scales for alexithymia, anxiety, depression, and GI symptoms. Patients who did not respond to treatment were more alexithymic, more anxious, and depressed, and had more pronounced GI symptoms before and after treatment than responding patients. In another study, we compared FGID patients with comorbid psychiatric disorders who were referred to a GI facility, to psychiatric patients with comorbid FGID who were referred to a psychiatry outpatient facility. Severity and frequency of GI symptoms did not differ, while FGID patients with comorbid psychiatric disorders were more alexithymic and had less psychopathology than psychiatric patients with comorbid FGID. Patients with high alexithymia tend to experience their emotions as bodily sensations and impulses to action rather than as subjective feelings that they can readily identify and communicate to others. In addition, they may be prone to functional somatic symptoms because of a tendency to amplify, focus on, and misinterpret the somatic sensations that accompany states of emotional arousal as well as other normal bodily sensations (somatosensory amplification). Consequently, they may experience more severe somatic symptoms, respond poorly to treatment because of the difficulty in cognitively processing emotional and somatic stimuli, and seek help from a medical rather than a psychiatric setting.

Conclusion

Current symptom-based diagnostic criteria recognize that IBS is a group of functional bowel disorders sharing some prominent clinical features rather than one single disorder. The similar bowel symptom expression may be a common final pathway resulting from the limited number of perceptual (pain, discomfort) and behavioral (bowel movements) responses to gut stimulation, regardless of the underlying mechanisms. Several pathophysiological mechanisms directly related to GI functioning have been postulated, recently it has been suggested that the immune inability to efficiently down-regulate gut inflammatory response may play a role in IBS, similar to inflammatory bowel disease.

A likely unifying hypothesis (Fig) is that IBS results from a dysregulation that may occur at any level of the interactions both within the bidirectional brain-gut axis and between this and other physiological and psychosocial systems. From a psychological perspective, IBS may be conceived as a somatization process. In particular, it may be viewed as an abnormal cognitive processing of emotional and visceral stimuli, a tendency to perceive somatic stimuli as evidence of symptoms of disease, and to seek repeated and often unnecessary medical care. Psychosocial factors such as somatosensory amplification, psychopathology (anxiety, mood, and somatoform, disorders), past and/or current life stress (e.g., sexual and physical abuse, parental reinforcement of sick role in early life), social support, persistent somatization, and alexithymia interact with the brain-gut system and contribute to stepping up from gut sensations to IBS symptoms. Degree of quality-of-life impairment, abnormal illness behavior, and concurrent psychopathology may mediate the referral to primary (mild

Fig: Schematic representation of symptom development from normal gut sensations to IBS. ENS = enteric nervous system; ANS = autonomic nervous system; CNS = central nervous system; HPA = hypothalamic-pituitary-adrenal axis; ACC = anterior cingulate cortex; HRQL = health-related quality of life.

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IBS, higher prevalence) or tertiary (severe IBS, lower prevalence) care settings.

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The 2nd S R Nalk Memorial Workshop on ‘Research Methodology’ will be held in Lucknow August 28 and 29, 2004
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