شبكة المعلومات الجامعية

التوثيق الإلكتروني والميكروفيلم

بسم الله الرحمن الرحيم

HANAA ALY

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جامعة عين شمس

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قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

HANAA ALY

INTRODUCTION

Irritable bowel syndrome (IBS) is a chronic functional gastrointestinal disorder characterized by abdominal pain or discomfort, in association with altered bowel habit. The natural history of the condition is a relapsing and remitting one ***(Ford et al., 2008),*** with most sufferers experiencing episodes of exacerbation of symptoms and other periods where symptoms are less troublesome, or even quiescent.

The prevalence of IBS in the general population varies between 5 and 20% in cross-sectional surveys ***(Hillila et al., 2004),*** and may be influenced by the demographics of the population under study. For example, in our study it is 7%, IBS is commoner in females ***(Sperber et al., 2005)*** and younger individuals ***(Hungin et al., 2005).***

Prevalence is also higher in those with coexisting functional gastrointestinal diseases, particularly dyspepsia and gastro-oesophageal reflux disease ***(Ford et al., 2010),*** and other functional disorders, such as fibromyalgia and chronic fatigue ***(Riedl et al., 2008).***

It seems unlikely that there is a single unifying explanation for these symptoms. It is more plausible that a combination of factors contributes to the abdominal pain and disturbance in bowel habit. Proposed etiological mechanisms that may be

involved in the disorder include altered gastrointestinal motility, visceral hypersensitivity, abnormal pain processing in the central nervous system, dysregulated intestinal immunity ***(Chadwick et al., 2002),*** low-grade inflammation and altered gastrointestinal permeability following enteric infection, imbalances in intestinal flora and altered psychological state ***(Kassinen et al., 2007).***

Irritable bowel syndrome also aggregates in families but whether this is due to genetic factors, shared upbringing, or both is unclear. Diagnosing IBS can be challenging for the physician, due to the potential for overlap between the symptoms that sufferers report and those of organic gastrointestinal conditions such as coeliac disease, small intestinal bacterial overgrowth, bile acid diarrhea, exocrine pancreatic insufficiency, inflammatory bowel disease and even colorectal cancer. Attempts to identify a biomarker for the condition have, to date, been unsuccessful ***(Lembo et al., 2007)***.

Medical treatment for IBS is considered to be unsatisfactory, with patients representing a significant financial burden. Despite this, there is evidence that fiber, antispasmodic drugs, antidepressants and probiotics are all more effective than placebo in the short-term therapy of IBS, although no single medical treatment has been demonstrated to alter the long-term natural history of the disorder ***(Moayyedi et al., 2010).***

The definition and classification of IBS are both of paramount importance to the management of sufferers. Accurate definitions

allow physicians to diagnose IBS with confidence, but physicians should be discouraged from over-investigating young patients who are otherwise well and clearly meet these criteria, and in whom the diagnostic yield of such investigations is likely to be low, hence reducing the costs and also avoid unnecessary surgery in patients with IBS ***(Erdrich et al., 2020).***

Classification of IBS according to symptoms allows the tailoring of therapy according to the predominant symptom reported by the patient, as well as the assessment of which of the existing, as well as novel, treatments are effective in particular subgroups of patients. As a result, in the latest Rome definition it is possible to classify IBS into diarrhea-predominant (IBS-D), constipation-predominant (IBS-C), or those who fluctuate between the two, so-called mixed IBS (IBS-M) ***(Berstad et al., 2020).***

This is a useful approach for several reasons. Firstly, it allows the targeting of therapies by the physician towards the most troublesome by the patient. Secondly, it aids the development of new pharmaceutical agents to treat these symptom reported subgroups discretely. Thirdly, it allows the investigation of patients according to these subcategories in order to explore possible underlying pathophysiological mechanisms, towards which future therapies may be directed ***(Marciani et al., 2010).***

Mental balance, the ability to cope with stress, as well as physical activity and fitness, remain key elements in maintaining

physical and mental health. Based on research in various fields of medicine, bearing in mind the overall pro-health effect, it should be assumed that they also bring added benefits to the treatment of patients with IBS ***(Longstreth et al., 2004).***

Taking into account the symptoms reported by patients (up to 80% of respondents say the occurrence of symptoms is dependent on their current diet) and the available test results, it can be assumed that diet is important in the occurrence of symptoms of irritable bowel syndrome ***(Schnabel et al., 2018).*** Many trials have shown that efficacy of probiotics is strain-dependent. Therefore, in this analysis, although probiotics in general were also considered ***(Kabir et al., 2011).***

Functional gastrointestinal tract disorders have been considered for several years as a manifestation of disorders of interactions of the brain-gut-microbiota axis. Abnormalities leading to the occurrence of abdominal symptoms include disturbances of nerve conduction which result in hypersensitivity to stimuli and a hyper-reactive neuronal response. In patients with IBS, emotional disorders often occur (mood disorders, depression, anger, somatisation) ***(Dinan et al., 2006).***

AIM OF THE WORK

The aim is to study the associated factors with irritable bowel syndrome among medical Ain Shams university students.

IRRITABLE BOWEL SYNDROME

DEFINITION:

Irritable bowel syndrome (IBS) is a chronic and debilitating functional gastrointestinal disorder that affects 9%-23% of the population across the world ***(World Gastroenterology Organization, 2009)***.

Over the past 20 years, the definition of IBS has evolved by expert opinion and based on studies that have identified symptoms that discriminate those labeled as IBS from organic disease, as well as factor analyses that have identified clear symptom clusters. Classically, IBS presents with abdominal pain or discomfort that is relieved by defecation or is associated at its onset with a change in stool frequency (either an increase or decrease) or a change in the appearance of the stool (to either loose or hard). The absence of alarming symptoms such as gastrointestinal bleeding, weight loss, fever, anemia or an abdominal mass support such a symptom complex as IBS rather than as structural disease. A number of other comorbid conditions may occur more often than expected by chance in those with IBS, including gastro-esophageal reflux, genito-urinary symptoms, fibromyalgia, headache, backache and psychological symptoms. Hence, IBS can present to a number of different subspecialists and is often initially misdiagnosed ***(Ford et al.,2018).***

IBS can be subdivided into those who tend to have predominant diarrhea or predominant constipation ***(Ballou et al., 2019).*** There is also a group of IBS patients who have mixed constipation and diarrhea. To complicate matters, those with one predominant bowel pattern can alternate with the other. Highly variable bowel symptoms support a diagnosis of IBS, but the coexistence of abdominal pain and disturbed defecation remains a paramount for diagnosis. According to the world health organization (WHO) the Diagnostic and Statistical Manual of Mental Disorders (fourth edition)(DMS-IV) code classification for IBS and its subcategories, IBS can be classified as either diarrhea-predominant (IBS-D), constipation-predominant (IBS-C), or with alternating stool pattern (IBS-A) or pain-predominant. In some individuals, IBS may have an acute onset and develop after an infectious illness characterized by two or more of the following: fever, vomiting, diarrhea, or positive stool culture. This post-infective syndrome has consequently been termed "post-infectious IBS" (IBS-PI) ***(Linsalataet al., 2018).***

EPIDEMIOLOGY:

The prevalence of IBS in the general population varies between 5 and 20% in cross-sectional surveys, and may be influenced by the demographics of the population under study.Prevalence estimates for IBS vary greatly internationally, both within and between countries ***(Wang et al., 2018).***

PATHOPHYSIOLOGY:

The biopsychosocial model of IBS integrates a number of psychosocial, motility, sensory abnormalities and abnormalities in central nervous system processing of visceral pain as the causes of abdominal pain and altered bowel habits. Motor dysfunction contributes to some symptoms of IBS, such as abdominal pain, defecatory urgency, and postprandial bowel movements. Rapid small bowel and colonic transit times have been reported in patients with diarrhea-predominant IBS. Patients with constipation-predominant IBS may have a component of disordered defecation, resulting, at least in part, from abnormal function of the pelvic floor and anal sphincter muscles. Another factor in motor dysfunction is the abnormal passage and handling of gas. Colonic and rectal hypersensitivity "visceral hyperalgesia" are also important factors in the causation of symptoms. Enteric propulsion and sensation are, in part, mediated by acetylcholine and serotonin ***(Defrees and Bailey, 2017).***

There is increasing evidence that organic disease of the gastrointestinal tract can be identified in subsets of patients who fulfill the Rome criteria for IBS. Evidence for subtle inflammatory bowel disease, serotonin dysregulation, bacterial overgrowth and central dysregulation continue to accumulate. The underlying causes of IBS remain to be adequately identified, but IBS-PI is a clear-cut entity. Furthermore, a genetic contribution to IBS also seems likely ***(Ng et al., 2018).***

CAUSES:

1. Infection:

Theories concerning IBS associate the inflammation of enteric mucosa or neural plexuses with symptoms. It is hypothesized that inflammatory cytokines may activate peripheral sensitization or hypermotility. There is increasing evidence regarding the role of immune activation in the etiology of IBS, which has mainly been shown in studies investigating mechanisms of IBS-PI ***(Facciola et al., 2017).***

One group of researchers was able to predict the development of IBS in patients with infectious enteritis in the presence of stressful life events and hypochondriasis. Approximately 1 in ten patients with IBS believe their IBS began with an infectious illness. Prospective studies have shown that 3%-36% of enteric infections lead to persistent new IBS symptoms; the precise incidence depends on the infecting organism. Whereas viral gastroenteritis seems to have only short-term effects, bacterial enteritis and protozoan and helminth infections are followed by prolonged IBS-PI. Researchers in Ontario recently demonstrated that post infection inflammation (Trichomonas spiralis) alters visceral sensitivity. In this particular study, National Institute of Health (NIH) Swiss mice were infected with T spiralis. Six days after infection the mice experienced jejunal enteritis, which returned to normal after 28 days. Using a latex balloon placed in the distal colon, investigators found hyperalgesic sensory

response following distension that persisted despite the lack of acute inflammation ***(Ghoshal and Gwee., 2017).***

Risk factors for developing IBS-PI include, in order of importance, prolonged duration of initial illness, toxicity of infecting bacterial strain, smoking, mucosal markers of inflammation, female gender, depression, hypochondriasis, and adverse life events in the preceding 3 months. Age older than 60 years might protect against IBS-PI, whereas treatment with antibiotics has been associated with increased risk. The mechanisms that cause IBS-PI are unknown but could include residual inflammation or persistent changes in mucosal immunocytes, enterochromaffin and mast cells, enteric nerves, and the gastrointestinal microbiota ***(Parida et al., 2019).***

Exposure to intestinal infection induces persistent low-grade systemic and mucosal inflammation, which is characterized by an altered population of circulating cells, mucosal infiltration of immune cells and increased production of various cytokines in IBS patients. Recent studies have also indicated an increased innate immune response in these patients by evaluating expression and activation of Toll-like receptors ***(Lee et al., 2017).***

These findings suggest that immune activation may play a crucial role in the pathogenesis of IBS. In addition, psychological stress has been reported to be one of the factors that induce

immune activation. However, it remains unknown whether immune activation in IBS patients is largely dependent on infectious gastroenteritis and/or psychological stress. Additional studies are necessary to understand the precise mechanism of immune activation and its relationship to the development of IBS ***(Long et al., 2018).***

- ***High sensitivity C-reactive protein(hs-CRP):***

Although IBS is not considered an inflammatory disease, recent studies suggest a possible role for alterations in the intestinal immune function and low grade inflammation in its pathogenesis. The first evidence for an inflammatory component in IBS was reported in 1960 showing that IBS patients have a higher number of mast cells in their intestinal wall compared to healthy subjects. More recent studies have described additional histopathologic abnormalities in biopsies from the intestinal mucosa of patients with IBS, including increased numbers of activated immunocompetent cells, such as: intraepithelial lymphocytes, lamina propria CD3+ cells, CD25+ cells, neutrophils and mast cells compared with controls***(Altun et al., 2019)***

Additional support for a potential role for low-grade inflammation in IBS came from epidemiological observational studies showing that in 6-17% of IBS patients the onset of symptoms may relate to an acute episode of gastrointestinal infection; usually referred to as IBS-PI. Of particular interest are

the findings of ongoing alterations in enteroendocrine cells, higher number of T-cell lymphocytes and increased expression of interleukin 1β in mucosal biopsies of patients with PI-IBS. Other studies that investigated systemic immune function in patients with IBS have demonstrated that an underlying inflammatory response can also be identified in peripheral blood. Examples are genetic studies demonstrating reduced levels of IL-10 expression, findings of increased ratio of pro- to anti-inflammatory cytokines (i.e., IL-10 to IL-12), increased release of pro-inflammatory cytokines (e.g., IL-1β_, IL-6, and TNF-α) from peripheral blood mononuclear cells (PBMCs) and increased numbers of activated T cells in the peripheral blood in patients with IBS compared to controls ***(Nasser et al., 2019).***

In a recent studies that investigated the possibility of detectible systemic inflammatory response in IBS by comparing the levels of hs-CRP, a non-specific marker of inflammation, in patients with IBS and healthy controls (HC). It was found that a significantly higher levels of hs-CRP in patients with IBS compared to HC. In addition, it was demonstrated, that hs-CRP levels correlate with symptoms severity of IBS suggesting that IBS, and specifically IBS-D, may be associated with systemic inflammatory responses ***(Hod et al., 2016).***

- ***Food Poisoning:***

The most common bacteria that cause food poisoning like Shigella, Campylobacter, C. difficile, Salmonella, and E. coli.