Challenging the Existence of IBS-D

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Irritable bowel syndrome (IBS) constitutes approximately 50% of a gastroenterologist’s practice and amongst the most common gastrointestinal disorders [1]. Diarrhea predominant IBS is thirty percent (30%) of that number and involves twelve percent (12%) of people across five continents with significant social, emotional and economic impact [2]. Hence, addressing this entity and understanding the pathogenesis and therapeutic options is of utmost importance.

Chronic Diarrhea is defined as greater than three bowel movements per day for at least three months. This symptom may vary from simple urgency and frequent bowel movements to incontinence resulting in tendency to locate the closest bathroom to avoid embarrassment, “bathroom mapping”. This imposes significant emotional stress on the suffering subject and frequently results in social embarrassment. What technically separates functional diarrhea from IBS-D is the satisfaction of special criteria created to define this exact entity. The latest of which is Rome III criteria in which discomfort and/or pain is used as a mainstay of the criteria together with altered bowel habits [3] (Table 1).

Table 1: Rome III diagnostic criteria for irritable bowel syndrome

Although Rome III criteria was created to help the practicing physician differentiate IBS-D from other causes of chronic diarrhea, it ended up being an academic reference for researchers rather than a practical guideline for practicing clinicians. Most physicians, in reality, lump chronic diarrhea of unknown etiology under the umbrella IBS-D rather than systematically investigating the causes for such diarrhea and applying the appropriate therapeutic measures.

As such, detailed history starting with food intolerance such as lactase or any carbohydrate should be explored. Lactose intolerance may be both qualitative and quantitative and does not have to be an all or none phenomenon. Gluten intolerance with or without genetic markers (HLA Q2, HLA Q8) or antibodies (tissue Transglutaminase antibody [TTG] IgA and Endomysial antibody should also be considered. It has been well documented that gluten intolerance rather than sensitivity may play a significant role in the etiology of chronic diarrhea. Medications such as NSAID, Metformin or any other prescribed or OTC medication are frequently overlooked and may again be the cause for diarrhea. Surgical history such as ileal resection, pancreatic surgery, cholecystectomy and gastric surgery such as vagotomy or fundal plication may also contribute to the underlying etiology [4].

In a study of 303 patients fulfilling the criteria for IBS-D the etiology of underlying diarrhea was identified in thirty-one percent (31%) of the studied population by history without further investigation or work-up [5]. This emphasizes the importance of time honored detailed history taking in dealing with such symptoms.

The initial work-up should include the routine blood count and chem profile as well as Celiac disease antibodies (TTG and Endomysial antibodies). The genetic testing for Celiac should be reserved for patients with strong gluten intolerance and negative antibodies as well as those with a strong family history of Celiac disease and negative antibodies. Stool analysis, particularly C diff, O/P, C/S and Giardia antigen should be routinely performed with particular reference to increasing C diff recurrent infections with or without prior antibiotic use. Fecal Elastase-1 should be measured to evaluate the increasingly recognized entity of exocrine pancreatic insufficiency.

If a diagnosis is not attained at this level, further work-up with particular reference to colonoscopy and biopsy is warranted rather than empiric therapeutic trials. Biopsy of the normal looking intestine is the only way to identify microscopic colitis as a cause for chronic diarrhea as therapeutic management of this entity is very specific and rewarding. Inflammatory bowel disease should also be ruled out with visualization of the mucosa. Needless to say, red flag symptoms such as rectal bleeding, nocturnal symptoms, anemia and weight loss, should be investigated promptly and therapeutic blind trials should not be attempted when these signs and symptoms are present as they may be indicative of serious underlying pathology and should alert the investigating physician.

If all of the above were negative, at this point, the general practitioner and gastroenterologist are inclined to tag the patient with IBS-D diagnosis and a series of therapeutic trials are implemented. Most physicians will address the entity with the patient and start different dietary restrictions, antispasmodics and anti-diarrheal agents such as loperamide or diphenoxylate with variable success. Of the two agents, loperamide is the preferred agent as it is less likely to interfere with bladder function or exacerbate glaucoma and tachycardia. The use of anti-depressants (tricyclic agents) has also been studied and has shown partial response [6,7]. The mechanism is thought to be neuromodulatory and analgesic effect. Generally these agents have
more relief for pain than diarrhea; hence, the results have been questionable at best. Alosteron, a 5-HT3 antagonist, is the only therapeutic agent approved for IBS-D therapy [8], but its use has been limited to gastroenterologists for patients under an on-going risk management plan. Recently antibiotics were also entertained as bacterial and inflammatory component for IBS-D was hypothesized [9]. The response to such therapeutic measures is temporary and might just be addressing bacterial overgrowth rather than IBS-D.

Bile induced diarrhea has increasingly been identified as a cause for chronic diarrhea frequently confused with IBS-D [10]. In a study of patients with IBS-D indicated Bile induced diarrhea constitutes sixty-eight percent (68%) of the studied patients [11]. These included post-cholecystectomy patients (23%), Habba Syndrome (41%) [12] and empiric therapy with bile acid binding agents (4%). Because bile induced diarrhea has a very favourable response to bile acid binding agents (98%), gall bladder dysfunction should be evaluated with DISIDA scan with CCK to evaluate the ejection fraction and identify Habba Syndrome.

So for most physicians, after a trial of the above mentioned agents and dietary restrictions, partial relief may be achieved, but for the most part, the patient continues to suffer from this debilitating ailment with a diagnosis of IBS-D. This entity has realistically been used as a “wastebasket diagnosis” and a “catchall” term when no explanation can be found particularly when only preliminary testing is done with negative results. In fact, some publications have implied that further work up beyond the initial evaluation is a waste of time and can be counter-productive considering the cost of the investigations [13].

In continuing this saga and in a desperate move, clinicians have been searching for fundamental understanding of IBS-D where antibiotics and anti-inflammatory agents are entertained with questionable result at best [14]. Perhaps failure to treat IBS-D is exactly due to the lack of in depth investigation and work-up particularly with the mounting pressures of medical costs and policy restrictions. In fact, some literature encourages minimal work up and claim similar outcomes [15]. Researchers have been so mesmerized by the term IBS-D that even lactose intolerance and Celiac disease is now attached to this entity, once abdominal pain is associated with these entities or any other entity for that matter.

The detachment from the term IBS-D is a challenging concept, to say the least, but certainly one that requires serious consideration. Status quo is simply unacceptable. Millions of sufferers are awaiting relief from this devastating illness and have been disappointed with management of this ailment because of their physician’s reliance on an entity that was only created to justify the failure to treat chronic diarrhea. Perhaps the time has come to acknowledge that failure to treat IBS-D is because there is no such entity as IBS-D and work up should be pursued until a definitive etiology is found. Only then millions of sufferers will have a real hope of living a normal life.

References