INTRODUCTION
Irritable Bowel Syndrome (IBS) is a common multi-factorial disorder affecting approximately 12% of the populations worldwide resulting in significant global morbidity. Approximately one in ten patients with Irritable Bowel Syndrome (IBS) believes that their IBS began with an infectious illness. The persistence of these symptoms about the clearance of infecting pathogen is known as post infectious IBS (PI-IBS). The current conceptual framework regarding the patho-physiological mechanism of PI-IBS suggests that PI-IBS is associated with altered motility, increased intestinal permeability, increase numbers of enterochromaffin cells and persistent intestinal inflammation, characterized by increased number of T-lymphocytes and mast cells and increased expression of pro-inflammatory cytokines. This therefore suggests that an exposure to pathogenic organism disrupts intestinal barrier function alter neuromuscular function and triggers chronic inflammation causing sustain IBS symptoms.

There is a scope of future potential intervention to prevent PI-IBS complication in travelers who are well known to at risk of infections diarrhea, often with invasive pathogen i.e. C. Jajunni, Sigella and Solmonella. Number of relatively safe and effective prophylactic regimen exists including addition of refuximin as a chemo-prophylaxis agent against traveler’s diarrhea.

EPIDEMIOLOGY
 Infective diarrhea is one of the most common illness worldwide. A link between IBS and enteric infection was first proposed by Stewart more than 5 decade ago. Since then, various perspective and retrospective studies from various countries have reported that a substantial proportion of patient with IBS reported that onset of their symptoms after an acute episode of gastroenteritis. The incidence or prevalence of PI-IBS range from 5% to 32%. Consistent among these studies is the suggestion that PI-IBS is a global phenomenon and not unique to any ethnic group or environment. Incidence or prevalence of PI-IBS varies in part because of differences in study methodology, including the criteria used to define IBS. In general rome II criteria generate lower estimates then rome I or III.

Clinical feature of PI-IBS
The majority of cases of PI-IBS following C-Jejuni associated enteritis meet the rome II criteria for IBS with diarrhea predominantly, loose stool passed most frequently than normal and with urgency. The constipation, bloating and increased frequency of passing mucus per rectum have also been reported in some studies.

RISK FACTOR FOR PI-IBS
Genetic Factor
Predisposition to deferent type of infection depends on genetic factors, such as polymorphisms in genes that encode cytokines. Genetic studies of predisposition to IBS are limited and non have been specifically related to PI-IBS how ever it is clear that there might be a genetic predisposition toward severe inflammation that contributes to an increased incidence of post-infective symptoms.
**Psychosocial factors**

Childhood rearing practices have a strong effect on the chances that a person will develop IBS. Illness beliefs and behavior are usually established in childhood and might be expected to impact on the response to an infectious IBS. IBS was more common in children who were frequently absent from school. Several psychological factors such as hypochondriasis, somatization, neuroticism and depression have been shown to be effective in development of PI-IBS. One study that has examined this prospectively found that those who developed PI-IBS had greater anxiety, somatization and more negative illness belief.

A study of patients in Nottingham showed that bowel dysfunction after C. Jejuni infection was associated with an increased somatization score; suggesting that preexisting behavior patterns were important predictors of outcome.

Interestingly smoking substantially increases the risk of PI-IBS with an odd ratio of 4.8. There are no obvious mechanisms by which smoking might directly cause PI-IBS. Nicotine has been suggested to have anti-inflammatory effects however smoking is a marker of other factor such as neuroticism that also contribute to the risk of PI-IBS. So the effect could be indirect.

**Bacterial factor**

A wide variety of infections have been associated with PI-IBS with differing incidence rates. The importance of bacterial factor in the pathogenesis of PI-IBS because more clear when just a single organism is studied. One study showed the C. Jejuni infection was associated with more risk of PI-IBS where as risk associated with solmonella-spp infection was substantially low. A surrogate marker for bacterial toxicity is the duration of the initial illness; an illness lasting for more than 3 weeks had more rate ratio for PI-IBS; compared with those with an illness less the 3 days.

**Antibiotics**

Several studies have reported that patients who have received antibiotics had more and longer lasting IBS, symptoms compared to the patient who have not received antibiotics for primary AGE.

**Sex and Age**

The Majority of the studies discussed found that women were at increased risk for PI-IBS compared with man. Female patient have a higher incidence of psychological disorder including anxiety & depression.

Very old or young age does not exclude people from developing PI-IBS however in a large community survey, age older than 60 years was found to be protective.

**MECHANISM & PATHOGENESIS**

Acute infections diarrhea is rarely investigated invasively because the illness often resolve without specific treatment, therefore little is known about its pathogenesis.

**a) Mucosal injury and inflammation**

Spiller found that serial intestinal biopsies from patients recovering from compylo bacter Jejuni showed persistent inflammation with elevated T-lymphocytes and calprotectin -positive macrophages possibly a response to mucosal injury and inflammation. Different part of intestine is affected by different organisms and the symptoms also depend on the part of intestine infected. Giardiasis infection affects proximal part of the small intestine and produces symptoms of dyspepsia, early satiety and anorexia predominantly, whereas in bacterial enteritis inflammation and ulceration occur more distally, with involvement of terminal ileum and proximal colon, inducing diarrhea as a predominant symptoms. The persisting increase number of macrophages, lymphocytes, enteroendocrine cells and an increase in the proportion that were serotonin positive in serial intestinal biopsy of patient having persistent symptoms of IBS are directly related to the recovery of patients. This recovery process depends on severity of entero-mucosal damage.

**b) Mast cell hyperplasia**

Mast cell hyperplasia is commonly observed following infection with T. spiralis and shigella spp. The severity of abdominal pain in patients of IBS is because of increase in mast cell and their proximity to enteric nerves. Analysis of mucosal biopsy samples have shown that mucosal related mediators that activates afferent nerve and might therefore mediates the visceral hypersensitivity which is characteristics of PI-IBS.

**c) Inflammatory cytokines**

T-lymphocyte infiltration has been reported in several studies of patients with PI-IBS. It is not very clear weather these increase production of cytokines are from inflammation in the GIT or resulted from psychological stressors because peripheral blood mono-nuclear cell production of cytokines is altered by psychological stress. It will be interesting for future studies to include analysis of both psychological and immunological parameters in cases of PI-IBS.
d) Changes in gastrointestinal microbiota following infective enteritis.

The acute diarrhea that is associated with infective gastroenteritis leads to a profound depletion of the commensal flora followed by a loss of normal fermentation products, particularly short chain fatty acids with an increase in luminal pH. This change allows overgrowth of organism which are usually inhibited. This overgrowth of organism is responsible for the majority of symptoms of IBS.

DIFFERENTIAL DIAGNOSIS

The acute acquired hypolactasia, bile acid mal-absorption, infectious gastroenteritis, ulcerative colitis and diverticulitis are some of the mimic diseases to PI-IBS.

Acute acquired hypolactasia is usually serious in children and very short lived. Lactose intolerance usually does not occur in adult after infectious AGE.

The occurrence of bile acid malabsorption following PI-IBS is well documented. One characteristic feature that can distinguish cases of bile acid mal absorption from those of PI-IBS is nocturnal, high volume diarrhea. These patients have positive responses to the bile salt binding agent cholestyramine.

Ulcerative colitis is rarely a disease of acute onset. Diverticulitis is another AGE that is frequently followed by recurrent abdominal pain and diarrhea and can mimic IBS. Tropical sprue is another diarrhea disease that might mimic PI-IBS and has been reported in epidemics, but this is not well documented and further studies are needed.

PROGNOSIS OF PIIBS

Prolonged follow up studies of PI-IBS are scarce one of the earliest studies reported that 5year after onset 5 of 11 patients with PIIBS after S-enteritidies infection still had diarrhea. A recent meta-analysis is suggesting that patients with PIIBS can expect a gradual recovery.

MANAGEMENT OF PIIBS

The therapy for PI-IBS should be adjusted to the severity of patients symptoms assurance that he is going to improve is the corner stone of treatment.

Diarrhea should be controlled with the use of lopramide orally. Pain symptoms need to be addressed with antitriptyline. Corticosteroids have shown no improvement except as an anti-inflammatory effects.

Recently mesalazine has been proposed as a possible effective drug it is a anti inflammatory agent, preliminary results from a small randomized clinical trial showed that it reduces pain and stool frequency and consistency in patients with PI-IBS.

Alcetron, a 5 HT receptor antagonist was shown to be effective in patients with unselected PI-IBS. Antidepressants and psychotherapy might be indicated when psychological issues are prominent.

Pro and prebiotics or poorly absorbed antibiotics may be of help in correcting disturbed colonic microbiota and may show some promise in future management of this problem.

Additional high quality randomized controlled trials are needed to enable gastroenterologist to recommend effective evidence based treatment for patient with PI-IBS.

CONCLUSION

PI-IBS is a common complication of acute enteric infection while the epidemiology & natural history of this clinical phenomenon have been well characterized our understanding of its patho-physiology remain limited. A systemic review is required to provide baseline estimates of risk and disease burden that could be used to inform individuals and medical decision makers. About the current and potential future utility of primary and secondary interventions to mitigate this disease, particularly in populations such as travelers, whose risk of bacterial infectious diarrhea is very high; hence a safe and effective prophylactic interventions can be provided to them.

REFERENCES