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Editorial

Can a Mathematical Model Be Used to Estimate the Contribution of Acute Gastroenteritis to the Overall Prevalence of Irritable Bowel Syndrome?

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Article: Estimating the contribution of acute gastroenteritis to the overall prevalence of irritable bowel syndrome
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There is a close association between bacterial gut infection, subsequent emergence of persistent intestinal symptoms and the development of irritable bowel syndrome (IBS). IBS symptoms, in particular diarrhea and pain, may persist following an acute episode of bacterial gastroenteritis, and this is commonly recognized as post-infectious IBS (PI-IBS). The infection may set off changes in gut physiology that predisposes an individual to develope gastrointestinal symptoms. Predisposing factors such as stress (recent traumatic life events and neurotic personality traits) observed in association with the occurrence of mucosal inflammatory changes and presence of psychological disturbances, suggest a bio-psychophysiological model for PI-IBS.[1]

Several leading studies on PI-IBS were initially reported from the UK. Following these, other key studies from the USA, Canada and Asia have described similar findings of PI-IBS following both bacterial and viral infections. Three meta-analysis of published studies provided good supporting evidence for PI-IBS following infectious gastroenteritis.[2-4] It was estimated that there was a 6 to 7-fold increase in the odds of developing IBS following acute infectious gastroenteritis. Younger age group, female gender, prolonged fever, severity of initial bacterial infection, duration of enteritis, depression and anxiety are all risk factors for developing PI-IBS. These data do suggest a multifactorial involvement in the pathophysiology of PI-IBS but the exact mechanismis yet to be elucidated.

It has been estimated that 7% to 31% of people develop PI-IBS following an attack of acute gastroenteritis.[5,6] However, the real incidence and prevalence of PI-IBS in the general population is still unclear. Similarly the overall contribution of acute gastroenteritis to the total IBS population remains unknown. This is because most of the epidemiological studies conducted up till now either lack a proper control population, or limited by a short follow-up assessment period post infection.[7] It would be useful if determinination of the contribution of acute gastro-

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enteritis to PI-IBS is possible, as this will assist us in calculating long-term health care costs and convince policy makers that long-term benefits come from primary prevention of infectious gastroenteritis. In this issue of the Journal Shah et al[8] described a basic and empiric population based approach, using mathematical models to illustrate the theoretical extent of the contribution of infectious gastroenteritis to IBS over time.

The IBS prevalence is calculated using mathematical models and calculations are based on existing epidemiology data of PI-IBS incidence, rates of IBS remission after acute gastroenteritis over time, incidence of gastroenteritis in a given population, and characteristics of IBS illness and duration in the population derived from currently available published literature. The first model gave an unrealistically high prevalence rate and was thus non acceptable and invalid. Using the most conservative model, the steady state prevalence of PI-IBS was predicted to be around 9%. This prediction from the theoretical model was very close to the published prevalence rates of IBS which ranged from 10% to 15%,[9-11] suggesting that PI-IBS could be responsible for the majority of IBS cases in the American population over time. In countries at higher risk for enteric infection, this become more relevant as it implies that an increased incidence of IBS is expected, resulting in a step up burden on health care costs.

Unfortunately the expected prediction using the mathematical model was not observed in the Indian subcontinent, where the incidence of enteric infection is high but reported IBS prevalence rate is low.[12,13] This observed inconsistency and apparent weakness of the models could be due to a failure of recognition, and or reporting of enteric infections and IBS symptoms, as explained by the authors, who suggested that good prospective epidemiological studies from these areas will be essential.

One other unique observation that we can gather from developing countries is that even though enteric infections are common in certain local population IBS symptoms are rarely reported. This could be explained by the "hygiene hypothesis" where the possibility of an early exposure to certain pathogens especially helminthes may confer to the person an innate resistance to the development of PI-IBS later in life. Conversely, when daily survival is of the utmost importance, minor irritating symptoms are generally ignored.

One of the main weakness of the mathematical models is that their predictive accuracy depended mainly on several correct assumptions, so they are as precise as the data available on PI-IBS. In developing countries, this may be a problem, as in many local populations such data may not be available. Furthermore, even if the incidence rates are available, it may not be reliable since mild form of acute gastroenteritis tends not to be reported. It has been observed that even mild forms of gastroenteritis can progress to IBS. This may result in an underestimation of the prevalence rate. However, as suggested by the authors, these models are only used to illustrate that acute gastroenteritis plays a significant role in the development of IBS over time, and contribute considerably to the majority of the IBS population. Notably the authors never recommended that their models to be used to provide exact projections of current or future IBS prevalence rates.

The major advantage of these models is that they allow us to predict accurately the contribution of acute gastroenteritis to the overall IBS population, without which, we would have to conduct large and expensive prospective observational studies to arrive at similar conclusions.

Despite the fact that in certain situations there will be limitations, the hypothetical model does implicate infectious gastroenteritis as the main cause of IBS. Further studies are now needed to corroborate these findings and also to consider the impact of enteric infections on other functional gastrointestinal motility disorders. The implications from these observations are obvious and accordingly serious considerations should be undertaken by the health care authorities to implement measures to prevent transmissions and outbreaks of acute gastroenteritis. Such measures in the long term can decrease the significant burden of IBS to the overall health care costs.

The authors have shown us convincingly, a method of using a mathematical model to estimate the contribution of an infectious disease to the prevalence of a chronic illness at a later time. This model can be of use to the World Health Organization where it can be utilized to predict the impact of chronic illnesses, in populations on whom it is not possible to conduct proper studies.

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