DIARRHOEA

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The definition of diarrhoea is controversial as what constitutes diarrhoea to one may be quite normal to the other. We can define diarrhoea as frequent loose or liquid bowel movements that may be urgent or impossible to control. They often may be accompanied by some abdominal pain. Diarrhoea may be brought on by stress or fear, by eating large quantities of foods with laxative properties, such as prunes, beans or mangoes, or by taking certain drugs or antibiotics. For the adult population of well developed countries diarrhoea generally represents, at most, an inconvenience. However, among the very young, the old, the malnourished, and those living in marginal conditions, diarrhoea represents a serious, crippling, life threatening situation. As many as one third ofpaediatric deaths in developing countries are attributed to diarrhoea and resulting dehydration. Diarrhoea has even altered the course of military history by incapacitating large number of men, making them unfit for battle, Acute infectious diarrhoeal illness is recognized as one of the leading causes of morbidity and mortality in developing nations. Infectious gastroenteritis is cited as second only to respiratory infections as a cause of morbidity in childhood, Viral agents recognized during the past decade have been shown to be responsible for a large proportion of the diarrhoea for which an etiologic agent can be defined. Acute viral gastroenteritis affects all age groups of people and may occur in either sporadic or epidemic form. Most such illnesses are self-limiting, and in normal hosts recovery is complete. If severe dehydration occurs, morbidity and even mortality may be substantial.

Holiday tummy, one of the commonest forms of diarrhoea is caused by exposure to bacteria against which the affected person has no natural tolerance or immunity.

Persistent diarrhoea, especially when the stools are accompanied by blood and mucus, may be a sign of other diseases involving the intestines in particular, irritable bowel syndrome and ulcerative colitis. When diarrhoea is persistent, or when bouts of diarrhoea alternate with constipation, it is a symptom of bacterial, viral and, or parasitic enteric agents causing cholera, shigellosis, salmonellosis, yersiniosis, aeromoniosis, plesiomoniosis, amoebiasis, giardiasis and viral gastroenteropathy. It can be caused by infection with Escherichia coli and campylobacter strains, non-cholera vibrios and vibrio parahaemolyticus, other infectious diseases such as malaria and measles and intestinal helminths as well as chemical agents. Approximately 70-80% of diarrhoeal episodes in people visiting treatment facilities can be diagnosed etiologically if all the newer laboratory tests are available and are utilized. Relatively few laboratories have these capabilities however, from a practical clinical standpoint, most of these illnesses may be thought of as a single entity since the basic therapy required to prevent a fatal out-come, fluid and electrolyte replacement, is similar for all.

Acute onset diarrhoea may be caused by a variety of bacterial parasitic and viral agents. Included among bacterial etiologic agents are Bacillus cereus. Campylobacter jejuni, Clostridium difficile, Clostridium perfringens, Enterotoxigenic and enteroinvasive E. coli, Salmonellae, Shigellae, Vibrio cholerae and halophic vibrios, and Yersinia enterocolitica. Among parasitic agents are Giardia lamblia, Entamoeba histolytica, cryptospondium. Viral causes of acute onset diarrhoea are Rotavirus, Norwalk and similar agents, calicivirus, and adenovirus. With certain exceptions, serotypes of E.coli, which were formerly described as “enteropathogenic, have been shown not to be pathogenic and should therefore no longer be routinely identified in stool cultures[1].

To examine stool for the more conventional pathogenic bacteria, the laboratory should receive a freshly passed stool or freshly collected rectal swab. It may be helpful in distinguishing between diarrhoea due to invasive and toxigenic bacteria to examine a fleck of mucous or stool mixed with Loeffler’s methylene blue stain for the presence of leukocytes.[2]

The vast majority of acute gastrointestinal ifinesses do not involve a recognizable inflammatory process[3]. Although there is considerable inflammatory enteritis during summer months in wann areas with poor sanitation, most cases of diarrhoea in these areas are non-inflammatory, suggesting an enterotoxic bacterial, viral or noninvasive parasitic process[5].

Epidemic infantile diarrhoea has long been recognized as a potentially serious problem that occurs in newborn nurseries. The unusual susceptibility of newborns may be explained by their unique host status. They have not yet acquired a normal intestinal flora or specific immunity. Often this situation is compounded by severe underlying diseases such as prematurity or congenital cardiac or pulmonary disease. The consequences of diarrhoea in the newborn are unusually severe because of poorly developed homeostatic mechanisms and limited water and electrolyte reserves. The onset is insidious, with irritability and poor feeding over 3-6 days, vomiting and fever are infrequent, and stools tend to be watery, yellow green and usually without mucus, pus, blood. The greatest attack rate of diarrhoea in the community occurs it the time of weaning, usually between 6 to 24 months Of age. Weaning diarrhoea is a major cause of mortality around the world.

Weaning diarrhoea is usually an acute non-inflammatory process. Acute diarrhoea in children 6-24 months of age has been commonly associated with rotaviruses[6] and with enterotoxigenic E.coli[7]. Children with sporadic diarrhoea may have Kiebsiella Citrobacter, Aeromonas or E.coli that produce an (LT) heat labile, cholera like toxin[8]. The major non-bacterial cause of weaning diarrhoea is rotavirus. In temperate climates, acute non-inflammatory diarrhoea in adults may be caused by rotaviruses or by Norwalk-like viruses. The association of rotaviruses as well as adenoviruses coxsackie viruses and toxigenic clostridium difficile with diarrhoea, abdominal cramps, and a higher mortality among bone marrow transplant recipients has also been noted[9]. Additionally, several agents of food poisoning such as clostridium perfringens or Bacillus cereus commonly cause non-inflammatory diarrhoeal syndromes in adults. In adults living in areas with poor sanitation, several other agents commonly cause sporadic non-inflammatory diarrhoea. In certain areas in Asia, cholera is an endemic cause of severe watery diarrhoea. Outbreaks have been related to contaminated mineral water[10]. One should suspect cholera in any patient who has severe dehydration and watery diarrhoea, especially if the patient has a history of recent travel to a cholera endemic area. The disease can be so fulminant as to cause hypovolemic shock and death from the outpouring of liquid into the upper small bowel before the first diarrhoeal stool occurs[11] Whether it “arouses one from bed with a start at 4 A.M. for a record breaking race to the bath-room to begin a stacatto ballet[12], or it produces the poetry of the psalmist, 12 I am poured out like water, my heart like wax is melted in the midst of my bowels”, travellers diarrhoea has major impact each year on the 250 million international travellers. The global nature of the problem and some suggested casual forces are illustrated by its more euphemistic names:

DELHI BELLY “GYPPI TUMMY”, “GIS”, ROME RUNS, “CREEK GALLOP”, “TURKEY TROTS”, “MONTEZUMA’S REVENGE, AZTEC TWO-STEP”, “ADEN GUT”, “SAN FRANCISCITIS”, “BASRA BELLY”, “IA TURISTA”, “PASSION”, “HONG KONG DOG”, “CASABlANCA CURD”, and many more. The onset of the vast majority of traveller’s diarrhoea is usually between 5 and 15 days after arrival with a range from 3 to 31 days.

The illness typically manifests with malaise, anorexia,and abdominal cramps followed by the sudden onset of watery diarrhoea. Nausea and vomiting may accompany in some patients. The diarrhoea is usually non-inflammatory, without blood or pus. A low grade fever may be present in 30% of the cases. The causes of diarrhoea should be established before treating them with antibiotics and untill then mere fluid replacement therapy should be encouraged otherwise things would complicate.

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