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Mini Review

Atiopathogenesis of Acute Diarrhoea

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Abstract

Acute diarrhoeal diseases are caused by a number of bacterial, viral and parasitic microorganisms. The most important are *Vibrios* and Rotavirus. No structural damage occurs in the intestine of cholera patients. In the small intestine, glucose helps Sodium absorption. This is called carrier mediated absorption and paved the development of oral rehydration salt solution (ORS) and this solution saved the lives of millions of children all over the world. *Vibrios* and ETEC produce an exotoxin known as Cholera toxin (CT) or heat-labile toxin, LT) respectfully and are responsible for the pathogenesis of cholera and ETEC. ETEC also produces another Heat-labile toxin (LT) and this toxin also helps in outpouring of secretion in the intestine. The toxins are able to stabilize in its receptor with the help of Tcp. the etiological spectrum of acute diarrhoea varies from one place to another, as for example, cholera is much more prevalent in the developing world. LT-producing ETEC is more common in Eastern Region of India and LT mediated diarrhoea is more severe that ST mediated diarrhoea.

INTRODUCTION

Acute diarrhoeal Diseases comprise of a number of types as the etiology varies and manifestations are variable, e.g. watery diarrhoea and bloody diarrhoea (dysentery). Acute diarrhoea is defined as passage of loose stools at least three times in 24 hours. This may lead to life-threatening severe dehydration. In this situation, acute renal injury (AKI) may set in (< than 1% of total cases seen) and the patient may die. Early and adequate treatment (fluid replacement) saves the life of the moribund patient. These fluids (Normal saline and Ringers' lactate) require to be administered intravenously. This facility may not be available in all places.

There was a revolutionary discovery in the management of acute dehydration (mild to moderate and severe cases in the maintenance phase) when Oral Rehydration Salt Solution (ORS) [1,2] was found to be effective, safe, cheap and easily available in all regions. It saved the life of millions of children all over the world. This fluid is not enough for severe dehydration where initially IV fluid and then both (IV & ORS) are given. When loose stools contain blood and mucous accompanied by abdominal cramps and tenesmus (feeling of a sense of incomplete defecation with pain). In this communication, we will elucidate the causative agents of acute diarrhoea (non-invasive) and Shigellosis (invasive bloody diarrhoea).

ETIOLOGY AND PATHOGENESIS

Cholera is manifested by acute profuse watery diarrhoea, vomiting and dehydration. Enterotoxigenic *Escherichia Coli* [3] also produces similar acute watery diarrhoea. When loose stools contain blood and mucous accompanied by abdominal cramps and tenesmus (feeling of incomplete sense of defecation with pain), it is called dysentery. The etiological agents of acute watery diarrhoea are Vibrio cholerae O1 and O139, and E.TEC3. Bloody diarrhoea is produced by of Salmonellae, Campylobacter jejuni4, Enterotoxigenic Escheria coli (EPEC) and the protozoan parasite *Entamoeba histolytica*; *Campylobacter jejuni* [4], *salmonella*, Enteroinvasive *E. coli* and Enterohaemorrhagic *E. coli, V. Parahaemolyticus, shigellae* and Giardia lamblia (parasite). The most important pathogen for diarrhoea in children (6 months to 5 years, predominately < 2 years) is Rotavirus which is responsible for about 40% of all diarrhea in children. Other viruses like Calicivirus, Norlkwork virus are also causes of diarrhoea. At the Infectious disease Hospital at Kolkata about 20% of all diarrhoeal cases admitted are Cholera although the common perception is that cholera has disappeared from the region [5]. Persistent (diarrhea >2 weeks) and chronic diarrhoea (chronic diarrhoea > 3 months) are not within the scope of this article.

The mechanism of production of watery diarrhoea is described by taking the examples of *V. cholerae* and ETEC. *Vibrio cholerae* O1, O139 and ETEC (which also produces another toxin, LT, heat labile toxin) produce thermo stable exotoxin known as Cholera toxin (CT). By the ligated rabbit ileal loop method, S. N. Dey from Kolkata demonstrated that an exotoxin (CT) is produced inside the gut lumen. The toxin (CT) has two parts- part B (binding part; attaches to its receptor GM1 ganglioside) and the Part A enters inside the cell and initiates a cascade of reactions that results in fluid rich in electrolytes (sodium, potassium and bicarbonate) are lost in the stool. These events occur in the small intestine; when the resultant fluid reaches the colon, and exceeds

the absorptive capacity of the colon, diarrhoea ensues. In cholera stool, flecks of mucous are suspended in the liquid stool giving rise to "rice-watery" stool. It was shown that there is no structural damage to the intestine and that sodium and water are absorbed from small intestine in presence of glucose (carrier-mediated absorption). This understanding of the mechanism of intestinal absorption, the epoch making discovery of Oral Rehydration by Glucose Salt Solution (ORS) was made. The credit of development of ORS goes to the Johns Hopkins Group of Scientists at Calcutta and Seattle group of scientists Dhaka (both working with local doctors). This is called WHO /ORS. Later on, this ORS was found to cause hypernatraemia particularly in neonates and in young children. In view of this, the sodium content in ORS has been reduced. This is called "hypo-osmolar ORS". Hypo-osmolar ORS reduced the need for unscheduled intravenous infusion, stool output during rehydration, and the number of patients with vomiting during oral rehydration treatment and did not seem to increase of developing hyponatraemia [6]. This is an example of how basic research and applied research could do something together tangible for the mankind.

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