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Gastroenterology

AN EPIDEMIC OF ACUTE GASTROENTERITIS -RAYAPUDI VILLAGE

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ABSTRACT

BACKGROUND: Gastroenteritis is the leading cause of morbidity and mortality. Causes include infectious &non infectious

AIM & OBJECTIVES: The aim of present study is to identify the pathogen causing the out break of this acute diarrhea and to manage, prevent complications that occurs due to acute gastroenteritis.

MATERIALS & METHODS: Various Biochemical, Radiological, Microbiological parameters are analyzed clinically correlated and expressed as percentages of the individual parameters.

RESULTS: Only Males are effected in this point source epidemic from Rayapudi village among them 50% have severe hypotension, 37% have severe dehydration, 25%have Tachycardia,2% have bradycardia, 30% have Metabolic acidosis, 62% have raised creatinine, 25%have dyselectrolytemia, 7% cases went for dialysis , 2% are dead.

CONCLUSION:- The severe diarrhea due to Escherichia coli occurred because of low sanitation, and due to transmission of recombinant strains. Timely intervention and treatment with Intravenous fluids and antibiotics prevented major complications and death

KEYWORDS : Hypotension, Tachycardia, Dyselectrolytemia, Renal failure.

INTRODUCTION

Gastroenteritis is the leading cause of mortality and morbidity in developing and developed countries .Aetiological agents of Gastroenteritis can be viral, bacterial or protozoans;and bacteria can be either enteropathogenic,toxigenic or both.Gastroenteritis is most often self-limiting, most authorities are of opinion that stool cultures should be restricted to patients who are severely dehydrated, toxic or immunocompromised .

For most common encountered pathogen specific antibiotics is not needed if symptoms have improved by the time the culture become available.Main stay of treatment is to maintain hydration, relieve symptoms, prevent spread of infection and to give antibiotics empirically in indicated case.

Identification of etiological agent by bacterial stool cultures is required for severe prolonged diarrhea; regardless of pathogen detected or not optimal management with oral and intravenous fluids and quinolones minimize the risk of dehydration and helps in faster improvement of symptoms.

Peak incidence of Infectious Gastroenteritis are found in younger age groups(<5yrs), while severe disease leading to hospitalization and resulting death seen in elderly >60yrs (2)

AIM & OBJECTIVES

The aim of present study is to identify the pathogen causing the out break of this acute diarrhea and to manage,prevent complications that occurs due to acute gastroenteritis.

MATERIALS & METHODS

Various Biochemical, Radiological, Microbiological parameters are analyzed clinically correlated and expressed as percentages of the individual parameters.

RESULTS

Table:1 Includes Dehydration Status And Vitals Of The Patients

	MILD	MODERATE	SEVERE
Dehydration	7	18	15
Hypotension	8	12	20
	>100bpm		<60bpm
Heart rate	10		1

Table 2 Includes Dyselecrolytemias, Acidosis, Creatinine, And About Dialysis Requirement.

Serum Sodium	135 - 125	124- 120	<120
	7	2	1
Serum Pottasium	3.5-3	2.9- 2.5	<2.5
	3	6	2
Metabolic Acidosis	MILD	MODERATE	SEVERE
	4	2	6
Serum Creatinine	1.4-3	3 -10	>10
	11	8	6
Dialysis	Done		Not done
	3		11

Only Males are effected in this point source epidemic from Rayapudi village among them. 50% have severe hypotension, 37% have severe dehydration, 25%have Tachycardia,2% have bradycardia, 30% have Metabolic acidosis, 62% have raised creatinine, 25%have dyselectrolytemia, 7% cases went for dialysis , 2% are dead.

DISCUSSION

This acute episode of gastroenteritis was caused by diarrheogenic E.COLI.

There are five strains of E.COLI-ETEC, EPEC,EIEC,EAEC,EHEC.

ETEC-15-20% of community acquired and hospital acquired diarrhea are due to this strain which spreads via feco-oral route.

TYPES OF BACTERIAL GE

PARAMETER	SECRETORY GE	INFLAMATORY GE	INVASIVE GE
LOCATION	PROXIMAL SI	COLON	DISTAL SI
TYPE OF ILLNESS	WATERY DIARRHEA	DESENTRY	ENTERIC FEVER
STOOL EXAMINATION	NO FECAL	FECAL LEUCOCYTES	MONONUCLEAR
	LEUCOCYTES	PRESENT-PMN	LEUCOCYTES
MECHANISM	BACT.ENTERO TOXIN/	BACT.INVASION/ CYT	PENETRATE MUCOS
	ADHERENCE CAUSES	OTOXINS CAUSE	AND INVADE RES
	SHIFT IN WATER AND	MUCOSAL DAMAGE-	
	ELECTROLYTE	INFLAMATION	
	EXCRETION		
CLASSIC PATHOGENS	VIBRIO,ETEC,C .PERFE,	SHIGELLA,SALMONEL	SALMONELLA ,YERSIN
	B.CEREUS,STAPH	LA,C.DIFFICLE	IA

- EPEC- The patogenecity is due to activation of LEE(locus enterocyte effacement)genes and secretes effector substances likeTIR(translocatable intimin receptor)<which leads to further attachment ofEPECleading to flatenning of brush border of small intestine=>loss of absorption of dissacharides like sucrose< maltose etc> leading to osmotic diarrhea

Pathogenecity of ETEC- they colonise small intestine by means adhesive fimbiae or pilli called CFA(colonisation factor antigen) ,which binds to specific receptor on enterocyte surface which release heat labile LT-TOXIN(which is similar in structure to cholera toxin),binds to GM-1ganglioside on enterocyte surface leading to secretory diarrhea.

EAEC-due to adherence of organism to HEP-2 type epithelial cells leading to formation of stacked brick pattern on intestinal mucosal cells;leading to persistent diarrhea.

EIEC-invades and kills colonic enterocytes which resembles shigella "O" ANTIGEN leading to inflammatory diarrhea . Diagnosis is by stool culture on Mac Conkey agar. Serogrouping, DNA Hybridization or PCR

- Treatment:-fluid And Electrolytes, flouroquinolones/ azithromycin, probiotics, Rifaximin, Dialysis For Hus

CONCLUSION:-

The severe diarrhea due to Escherichia coli occurred because of low sanitation, and due to transmission of recombinant strains. Timely intervention and treatment with Intravenous fluids and antibiotics prevented major complications and death.

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CONFLICTS OF INTREST:

There are no conflicts of intrest.

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