ORIGINAL ARTICLE

Dysentery Form Gastroenteritis in Infancy.

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Abstract

Sixty three infants aged below 3 years suffering from dysentery form gastroenteritis were investigated. The investigation included clinical symptoms, course of the disease, stool ova and parasites and stool cultures for enterobacterial pathogens.

Stool examinations revealed: 25.4% Entamoeba histolytica, 22,2% E.E. coli, 15.9% Salmonellae, 1,6% E.E. coli and Salmonella, 1,6% E.E. coli and E. histolytica and 1,6% Staph. aureus.

The etiologic agent in the remaining 31.8% of the patients remained unknown.

The clinical features, the possible pathogenesis and treatment of the discovered pathogens are briefly discussed.

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Introduction

The syndrome of dysentery has clinically been described as diarrhoea containing mucus and blood in the stool, often accompaniod by abdominal pain and tenesmus (Suwignjo, 1979). While the whole description may be valid for adults, it may not allways be the case with infants as tenesmus and abdominal pain are difficult to be evaluated.

Infective diarrhoea has been incriminated as the most common cause of rectal bleeding in infancy (Roy and Silverman, 1975). The pathogenic bacteria may penetrate the mucosa of the distal small intestine and colon to produce morphological abnormalities and dysentery.

In developing countries like Indonesia it is generally thought that Shigella and Amoeba are the main culprits of "dysentery form" enteritis, and that the presence of mucus and blood in the stool suggests this type of diagnosis (Suwignjo, 1979). However, little is known of the nature of the dysentery syndrome in infancy as it is in adults.

Material and methods

Sixty three infants aged below 3 years presented with dysenteriform enteritis underwent investigations.

The investigations consisted of :

— Clinical symptoms and course of the disease.

— Laboratory examination : stool ova and parasites and stool culture for enterobacterial pathogens.

Results

TABLE 1 : *Age distribution*

	0 - 1 mo	2 - 6 mo	7 - 12 mo	> 12 mo	Total
Cult (—)	1	7	7	5	20 (31.8%)
E. histolytica	—	6	5	5	16 (25.4%)
E.E. coli	4	6	1	3	14 (22.2%)
Salmonella	2	7	1	—	10 (15.9%)
E.E. coli + Salm.	—	1	—	—	1 (1.6%)
E.E. coli + histol.	—	1	—	—	1 (1.6%)
Staph. aureus	1	—	—	—	1 (1.6%)

Total no. patients : 63.

TABLE 2: *Course of disease*

	Less than 7 days	More than 7 days
Cult (—)	15	5 (25 %)
E. histolytica	13	3 (18.8%)
E.E. coli	10	4 (28.6%)
Salmonella	4	6 (60 %)
E.E. coli + salmonella	—	1
E.E. coli + histolytica	—	1

TABLE 3: *Clinical symptoms*

	Vomit	Fever	U.R.I.	Conv.	No. of pat.
Cult (—)	2	6	7	—	20
E. histolytica	1	8	3	1	16
E.E. coli	2	2	6	—	14
Salmonella	2	3	3	—	10
E.E. coli + Salm.	—	1	1	—	1
E.E. coli + histol.	—	—	—	—	1
Staph. aureus.	—	—	—	—	1

Table 1 shows the age distribution of the patients suffering from dysenteriform gastroenteritis. E.E. coli and Salmonellae were mostly recovered in patients who were under 6 months of age. Amoebic dysentery was never found in neonates.

Table 2 shows the course of illness. Except for Salmonellae, in most of the cases the duration of illness was less than 7 days.

It is apparent from table 3 that in most instances the infecting agent could not be accurately incriminatel from the

clinical features, and the symptoms and signs might be similar in diseases caused by many of the listed agents.

Discussion

In the present study 68.2% of the cases had positive findings for bacteria and parasites: E. histolytica (25.4%), E.E. coli (22.2%), Salmonellae (15.9%), mixed E.E. coli and Salmonellae (1.6%), mixed E. coli + histolytica (1.6%) and Staphyloc. aureus (1.6%). The etiologic agents in the remaining 31.8% of the patients were unknown.

It is not uncommon for presumed virus induced diarrhoea to mimic the dysentery syndrome in infancy (Roy and Silverman, 1975). It was unfortunate that in the present study viral studies were unable to be performed. In the very young infant, bloody stools may also be due to severe Cow's milk protein allergy (Roy and Silverman, 1975). Yersinia (the examination on which unfortunately could also not be performed in the present study) has also been mentioned as a causative agent in dysenteriform gastroenteritis in infancy.

Enteropathogenic bacteriae may be broadly classified as invasive or non-invasive. Invasive organisms (e.g.: Salmonellae, Shigellae and some strains of E. coli) penetrate the mucosa of the distal small intestine and colon to produce morphological abnormalities and dysentery i.e. :passage of blood, mucus and/or pus in the stools). Dysentery results from mucosal disruption, and diarrhoea from jejunal secretion overwhelming the reabsorptive capacity of the injured colon.

The non-invasive enteropathogens (e. g.: V. cholerae, and E. coli) elaborate enterotoxin in the small intestine and induce secretion without affecting mucosal structure, the absorptive defect is confined to the small bowel and colonic function is normal (Harries, 1977).

The pathogenesis and clinical syndromes associated with bacterial diarrhoea are summarized by Roy and Silverman (1975) as follows:

Organisms.	Pathogenesis.	Syndrome.	Incubation.
V. cholerae.	Enterotoxin	Profuse	
E. coli	production.	diarrhoea	24 — 72 hr.
Shigella dys.	(Small-bowel)	(Cholera like).	
Shigella	Invasion and	Dysentery	24 — 72 hr.
E. coli	destruction	(Colitis)	24 — 72 hr.
Yersinia	(small bowel and colon).		7 — 10 days ?
Salmonella.	Penetration and systematic invasion (Small bowel and colon).	Mostly cholera like, Colitis less frequent, bacteriaemia.	12 — 36 hr.

It is apparent from the present study that there was considerable variation in the clinical manifestation of individuals affected by the same microorganisms. In other words, the infecting microorganisms cannot be precisely incriminated in the clinical features and course of the illness.

The symptoms and signs may thus be similar in diseases caused by various types of microorganisms.

Clinical distinction between gastroenteritis produced by E. coli and that caused by Salmonellae, Shigellosis or Viruses is indeed difficult (Roy and Silverman 1975).

It was impossible without the aid of bacteriologic or serologic examinations to differentiate dysenteric diarrhoea caused by various types of microorganisms and possibly also by unknown viruses (virus dysentery).

It is also apparent from the present study that patients affected by Salmonellae tend to have longer cause of illness than those affected by other microorganisms.

As for E. coli, Salmonellae were frequently found under the age of 6 months (see table 1).

Salmonellae invade the intestinal epithelium but there is no extensive destruction, the epithelial is left intact, and the organisms reach the lamina propria, where they set up an inflammatory response. The nature of this inflammatory response determines the pathogenesis and resultant symptomatology. The inflammatory response of the lamina propria is acute and involves the distal small bowel as well as the colonic mucosa (Roy and Silverman, 1975).

Antibiotics prolong the duration of uncomplicated Salmonella enteritis and the likehood of a carrier state development is increased, but in infections which have resulted in bacteriaemia or septicaemia, the use appropriate antibiotics is unquestioned and essential for the control of the diseases (Harries, 1977).

In rare cases in which the E. coli strain is invasive, it may set up widespread mucosal damage with acute inflammation.

The organisms penetrate the cells of the intestinal epithelium and cause a dysentery syndrome. The colon seems to be a predominant site for the multiplication of invasive E. coli (Roy and Silverman, 1975). Most of the patients who excreted E. coli and Salmonella were under 6 months of age (see table 1).

The susceptibility of infants younger than 6 months to certain strains of E. coli is related to the fact that maternal Ig M is not transfered to the fetus because of its molecular weight (mol wt 900.000). It is however known that antibodies against 0 antigen of gram negative bacteriae are mostly found in the Ig M fraction of human immunoglobulin. Fully breastfed babies are almost completely immune to path. E. coli gastroenteritis. There is no doubt that colostrum and breast milk Ig A prevents

adhesions of bacteriae to the intestinal mucosa and is truly an "antiseptic paint" (Roy and Silverman, 1975).

Because of the possible danger of sepsis and meningitis occurring during the first 3 months of life, a systemic antibiotic effective againt E. coli should probably be given if there is clinical evidence that one is dealing with a dysentery like syndrome indicative of an invasive strain. There is no indication, however, for use of absorbable antibiotics in infants older than 3 months (Roy ant Silverman, 1975).

The development of antibiotics resistant E. coli is a problem that is now better understood. When an antibiotic is given to an infant with E. coli gastroenteritis, the drug effects not only the bacteriall strain responsible for the diarrhoea but all the microorganisms of the gastrointestinal tract. This normal flora may acquire the so called infectious drug resistance, which is then transferred to pathogenic serotypes previously sensitive to the antibiotic in use.

Amoebiasis is an endemic disease transmitted from man to man. Prevalence is highest in the tropics and subtropics but cases have been reported from practically every country.

In the Philippines, amoebiasis has a prevalence rate of 12% and 1 in every 3 dysenteric stools is positive for E.. hystolytica (Latonio, 1976). In the present study E. histolytica was found in 25.4% of cases presenting with dysentery like syndrome.

In the island of Samosir, a western part of Indonesia, the prevalence of E. histolytica was found as high as 7.6%, while in Mataram, an eastern part of Indonesia, E. histolytica was recovered from 8.7% of 14.414 stool specimens (Suwignjo, 1979). There is a tenlency of increasing number of amoebiasis cases among infants (Latonio, 1976).

Recent studies with an electron microscope have revealed the mode of entry of the parasites into the intestinal tissue, At first the amoeba adheres to the plasma lamina of the epithelial cells and destroys the brush border. Subsequentely, it moves into the deeper tissues by separating the epithelial cells and by migrating through the inter-cellular spaces.

Once the amoeba has established in the submucosa it multiplies and produces micro-abscesses. These micro- abscesses grow and may rupture into the lumen giving rise to typical flask shaped ulcers (Zaman, 1976).

Treatment is directed toward (1) eradicating E. histolytica in both the bowel lumen and wall; and (2) protecting the liver from invasion (Latonio, 1976).

Until recently all amoebicides were selective in their sites of action. Thus there were tissue amoebicides like emetine and chloroquine, predominantly luminal amoebicides like emetine bismuth iodide and furadantine and indirect acting bowel spectrum antibiotics like tetracyclines. With such selective action, therapy was not satisfactory and amoe-

biasis has achieved the reputation of being a chronic relapsing condition. The advent of metronidazole in recent years has greatly improved the prospect of affecting a cure (Harnies, 1977). At a dose of 40 - 50 mg/kg daily for 5 days it is effective in amoebiasis of childhood. Good results are also obtained in chronic intestinal amoebiasis and in symptomless cyst passers (Harnies, 1977).

All patients with amoeba in the present study underwent treatment with metronidasole, 81.2% of them were clinically cured within less than 7 days and the remaining patients (18.8%) recovered after they had been treated for more than 7 days.

Metronidasole is rapidly absorbed from the intestine, easy to be administered, practically no side effects (if any slight gastrointestinal irritation) and is effective at all sites and has been indicated for liver abscesses as well (Roy and Silverman, 1975; Latonio, 1976; Harnies, 1977; Suwignjo, 1979).

Those who are clinically free from symptoms but still pass cysts could be given dilozarnide furoate (furoxone) for as long as 10 days (Suwignjo, 1979).

It was striking that no single Shigella was recovered from stools of patients in the present study.

Aswitha Damayanti et al., in 1973 could only isolate 6 patients with Shigella from 45 individuals presenting with dysenteri form gastroenteritis while Van Bauren in 1939 also in Indonesia was still able to isolate Shigella from 55% of dysentery form diarrhoea at the first examination (Aswitha Damayanti et al., 1973).

One could think of a changing pattern of the etiologic agents in infantile gastroenteritis.

Shigella cases in infancy in Indonesia seem to decline in the last years.

In Surabaya in 1976 Shigellosis could still be found in 4,6% of hospitalized and none in ambulatory treated gastroenteritis infants. More recent investigation in 1977 and 1980 on stool of infants with acute gastroenteritis admitted to Dr. Soetomo Hospital Surabaya revealed no single stool specimen containing Shigella

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