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Acute Gastroenteritis in African and European Adults in Rhodesia

BY

Michael Gelfand

C.B.E., M.D., F.R.C.P., D.P.H., D.M.R.

Professor of Medicine (with special reference to Africa), University of Rhodesia.

For the purpose of this article I would define acute gastroenteritis as a condition with acute onset in which the striking features are vomiting and the frequent passage of watery motions with little or no mucus or blood. Clearly there are a host of different disorders that would fulfil this definition, but my purpose is largely to present what to me as a physician is a most unsatisfactory state of affairs. Although it is known that there exists type specific *E. coli*, which can produce gastroenteritis, we rarely recover any organism in well over 90 per cent. of infants with this condition. Indeed, we can seldom demonstrate a causative organism and only occasionally are able to find a Shigella, Salmonella or Klebsiella one. Again, it is difficult to link or exclude a staphylococcal toxin in our cases. Then sensitivity foods, such as shell fish or eggs, often appear to be most unlikely, as they are not usually consumed by the Shona. Some suspect an enterovirus, but again, as far as I know, no one has yet shown this to be the case.

In the infant acute and serious gastroenteritis often follows upper respiratory and urinary infections or frequently a severe attack of measles. The same may be said of the adult in whom an attack of acute gastroenteritis often occurs during severe chronic debilitating disorders.

It might be of interest to learn how common this disorder is in an African hospital in Rhodesia. If we take the Mpilo hospital records for a period of ten years (1959-68) (Fraser Ross, 1969) and note the order of frequency of discharges, using the coding system employed by the International List of Cases for Tabulation of Morbidity and Mortality (List A), we find out of approximately 145,000 discharges the following:

1.	Diseases of the genito-urinary system	A 114
2.	Fracture of the limbs	AW 140
3.	Non-septic abortion	A 118
4.	*Gastroenteritis and colitis*	*A 104*
5.	Lacerations and other wounds	AN 145
6.	Lobar pneumonia	A 49
7.	Bronchopneumonia	

Taking the records of the Wedza Tribal Trust Land hospitals from 1st April, 1968, to 3rd March, 1969, the code figures are as follows:

1.	Bilharziasis	A 37
2.	Acute respiratory infections	AB 7 plus A 92
3.	*Enteritis and other diarrhoeal diseases*	*A 104*
4.	Avitaminosis and other nutritional deficiencies	A 65
5.	Measles	A 32

(Fraser Ross, 1969)

If we compare the discharge patterns in the urban area with those in the rural one, we find that only four of the diseases in the top 10 are the same in both. The four common to both are:

(1) Diseases of the genito-urinary system.
(2) Gastroenteritis and colitis.
(3) Lobar pneumonia and bronchopneumonia.
(4) Infections of the skin and subcutaneous tissue.

Fraser Ross (1960) working in Bulawayo considered that gastroenteritis there was associated with a seasonal incidence related to the advent of the hot weather with increased evidence of fly breeding and probably to the poor hygiene and storage of food in the indigenous population.

Dr. Mary E. Mackintosh has provided me with the monthly figures of acute gastroenteritis in patients attending the clinic at Wedza from March, 1968, to February, 1969. It will be seen from Table I that there were more cases in the warmer months of the year. Thus out of 2,406 patients seen, 186 were treated for diarrhoeal disease (approximately 8 per cent.), and from March to August there were 44 cases in contrast to 142 from September to February (the warmer half of the year).

Table I

Half Yearly Number of Cases Treated with Diarrhoeal Disease at Wedza Clinic (March, 1968, to February, 1969)

Total number of cases receiving treatment for various diseases	2,406
Number with diarrhoeal disease	186 (8%)
Number with diarrhoeal disease (March to August)	44
Number with diarrhoeal disease (September to February)	142

Having established that gastroenteritis is a very common disorder, the next stage of my investigations was to determine the age distribution of the cases admitted to the wards of Harare Central Hospital.

Table II

Age Distribution of Acute Gastroenteritis at Harare Central Hospital

1967			1968		
Ages in Years			Ages in Years		
0-2	2-19	20 plus	0-2	2-19	20 plus
396	63	115	731	102	228

Table III

Quarterly Records of Cases of Gastroenteritis Admitted to Harare Central Hospital

	Ages of Patients in Years		
	0-2	2-19	20 plus
June to August (coldest quarter)	151	29	45
September to November	135	12	62
December to February	200	39	65
March to May	245	22	56
	731	102	228

From these figures it will be noted that gastroenteritis may occur throughout the year and that it is not rare in the cold season in Rhodesia, although in the hottest quarter, December to February, it was seen more often. However, these figures are not statistically significant (W. Castle, personal communication).

My next step was to determine the cause or causes of gastroenteritis. We are already aware of the great complexity of the problem in the infant. In the adult the cause is equally elusive. For this purpose I studied 48 consecutive cases admitted to the medical unit of the University Teaching section at Harare Central Hospital. The point that stood out very clearly as a result of this investigation was that no organism that could be related to the disorder was demonstrated. The results of the bacteriological tests were uniformly disappointing. However, further investigations in the future may well reveal a viral cause. A study of the clinical picture of the disorder also failed to provide a clue to its causation. There appeared to be two main types. In the first and by far the more common group the disease occurred *de novo* in an otherwise fit individual. In the second it developed as a complication in a patient already ill with diabetes, hypertension or similar chronic condition.

As in cholera, its main important effects were loss of fluid and dehydration, which varied enormously from case to case. At times the fluid loss was quite extensive, causing the skin to feel loose, soft and doughy. The fever was generally mild, rarely moderate and never high. The blood pressure varied and was usually much reduced. A very constant finding was a relatively high haemoglobin, no doubt due to the loss of body fluid (haemoconcentration). Headache was one of the most frequent symptoms mentioned by the patient. Whereas it is unusual for a European to be admitted to hospital with gastroenteritis, this is a common event in Africans. Therefore it would be fair to deduce that an African with gastroenteritis must feel very ill to necessitate his admission to hospital.

The following is a brief account of the more severe form of acute gastroenteritis which may be encountered.

Muvamburi Chiratidzo, aged 24, was admitted because of acute vomiting and severe diarrhoea for 36 hours. No blood or mucus were observed in the stools. On admission he was dehydrated. The blood urea was 43 mg. per cent., but by the next day it had risen to 245 mg. per cent. Haemodialysis was performed. A renal biopsy revealed tubular damage. The patient made a full recovery. Diagnosis: acute renal failure with *complete recovery* following acute gastroenteritis.

In the 48 cases studied there were five deaths (10 per cent.). One of these had cirrhosis of the liver and was jaundiced before death. The other four cases seemed in good health at the time of onset of illness. Autopsies were performed on all five, but nothing special was found.

Table IV

FINDINGS IN THE FIVE PATIENTS WITH GASTROENTERITIS IN WHOM DEATH SUPERVENED

Age	Hospital No.	Blood Urea	Autopsy Report
58	9731	127	Paralytic ileus (? cause)
65	3021	No result	Acute gastroenteritis (no autopsy)
48	16651	285	Acute renal failure
40	40151	27	Liver failure (cirrhosis)
28	8760	76	Acute gastroenteritis

Case 1

Bere Mahari, hospital No. 9731, aged 58 years. 12.9.67.—History of vomiting and abdominal pains. Swelling of abdomen five days. Dehydrated. Blood urea 127 mg. per cent. At autopsy the entire gut including the stomach contained greenish fluid and faeculent material. No volvulus or adhesions. Autopsy paralytic ileus. Cause?

Case 2

Phiri Notiu, hospital No. 3021, aged 65 years. 30.6.68.—Admitted because of watery stools and severe abdominal cramps. The patient looked very ill and he was dehydrated. He deteriorated rapidly and died. No autopsy possible.

Case 3

Alec Vasco, hospital No. 16651, male, 3.3.68.—Admitted with acute diarrhoea and vomiting and abdominal pain over four days. Developed acute renal failure. Blood urea 280 mg. per cent. Autopsy: kidneys showed congestion and extravasation of blood into glomeruli and tubules. The glomeruli showed capsular adhesions and hypercellularity.

Case 4

Sifunduza Rosa, female, hospital No. 40151. 27.1.69.—Three days' duration vomiting and abdominal pain Dehydrated. Liver edge just palpable. Became jaundiced and died on 14.2.69. Massive haemorrhage from oesophageal varices in a subject with post-necrotic scarring of the liver. No ulceration of any part of mucosa of gastrointestinal tract.

Case 5

Gomba Mashura, hospital No. 8760. 16.8.67.—Admitted with severe vomiting and diarrhoea after a meal (five other people had the same complaint and took ill after the same meal). She had lower abdominal cramps and blood was also noticed in the stools. She was quite well before. When seen, she had developed signs of consolidation at the left base. She was exceptionally ill and collapsed and died on the same day of admission. Blood culture and Widal reaction were negative. Stool was dark brown with mucus, but contained no blood. No *E. histolytica* seen. Culture moderate growth of *Candida* species. Blood urea 75 mg./100 ml.

GASTROENTERITIS IN THE EUROPEAN ADULT —A PARTICULAR FORM

On the subject of the acute gastroenteritis in the European, I would like to describe the clinical features of a not uncommon syndrome which I am meeting in Rhodesia. I do not know its cause or indeed whether it is not a condition already well known. For some years I have seen Europeans in consultation with a sprue-like disorder. In most of them it commences abruptly with the passage of watery stools unaccompanied by the mucus and blood. The patient experiences abdominal pain and the temperature may be elevated. The diagnosis made at the time is one of acute gastroenteritis. For a few days or longer the looseness is quite marked, the patient losing much fluid. Then the acuteness lessens, the patient passes fewer stools which are perhaps a little better formed. This might be described as the subacute stage which continues in this vein for two or more weeks. Then the chronic stage is reached in which liquid motions are passed with seldom any mucus and blood. The stools tend to be unformed, soft, doughy in consistency, often offensive in smell and yellow. In other patients it is greenish or brownish in colour and does not resemble those found in the malabsorption syndrome. By this time the patient, still under constant medical attention, becomes concerned in case he has developed malignant disease, as he has lost a great amount of weight. I have not known any pathogen established on culture in any of my cases or those cared for by others. Usually a number of remedies are tried, including broad spectrum antibiotics and sulpha drugs, but the condition seems to continue in this chronic or subacute state, much to the dismay of the patient. Proctoscopy and sigmoidoscopy reveal little of note and a barium enema shows no changes suggestive of proctocolitis. On the other hand, a barium meal sometimes reveals clumping of the barium, suggestive of a small bowel disorder. Only about one-fifth of my cases have shown excessive fat in the stools and the xylose absorption is usually normal. I have not detected the

development of any anaemia or sore tongue. At first I believed this condition was possibly of viral origin with the small bowel as the seat of attack. Placing the patient on a gluten-free diet may be of great help in controlling the diarrhoea. However, I have had only limited success with this treatment and have found small doses of steroids of greater benefit. The condition eventually resolves completely, but it may take from three to nine weeks for recovery to take place. Not infrequently, when the patient is seemingly improving he relapses for one or a few days, but the relapse is not as severe as the illness was at its onset. I have not been able to have a jejunal biopsy done on any of my cases.

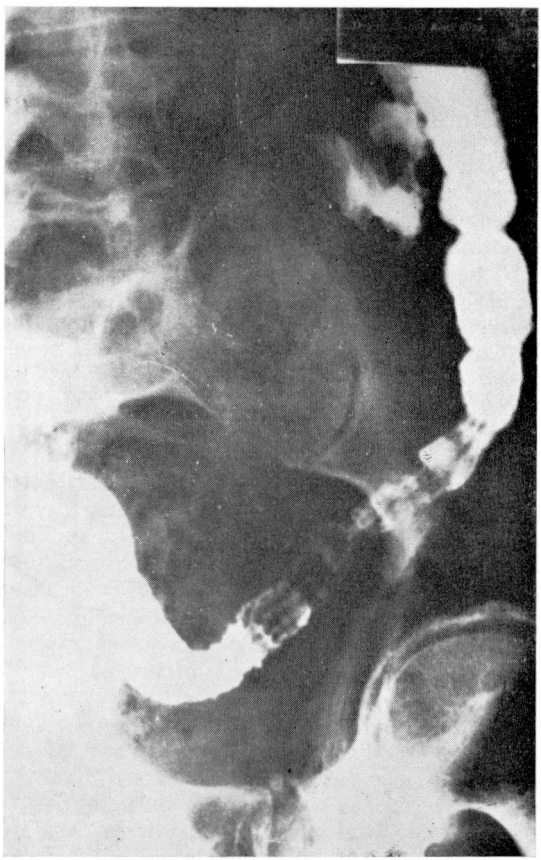

Fig. 1—Changes in the sigmoid region of the colon indicative of an ulcerative colitis.

May I quote some examples of patients seen with this disorder? E.M.T., a farmer, aged 44, started two years ago with acute enteritis. After a while the acute stage subsided and he passed two or three motions in the mornings. He complained of lack of energy. His doctor referred him to me as a case of malabsorption syndrome. A gluten-free diet, broad spectrum antibiotics and folic acid were of no benefit. When I saw him the total fats in his stool over three days were 2.7 grams. The stools were darkish brown. A barium enema showed features rather suggestive of ulcerative colitis and, like most of my other patients, the man noticed no mucus or blood in his stools (Fig. 1). I think that perhaps some of these patients may have a mild form of ulcerative colitis which is not obvious radiologically, although the great majority have no mucus in their stools and the onset of the disorder is very acute. He did extremely well and was cured after being placed on a course of steroids.

Another patient, E.W., aged 26, started with a very acute enteritis, which failed to clear. Her stools became very loose, bulky and were passed any time of the day. They tended to be yellow and offensive. No mucus was noticed. She lost a good deal of weight. No cause was found for the condition, which continued more or less *in status quo*. Later the presence of *E. histolytica* was reported, but the treatment for this had no effect. No changes were seen on sigmoidoscopy and a barium meal and enema showed no abnormality. The fat content of the stool was under 3 grams. Eventually she did quite well on small doses of steroids and the condition cleared after about 18 months.

A third patient, Mr. J.N., aged about 60, developed the condition very acutely with fever. He was given antibiotics and sulphaguanidine by his practitioner. The watery diarrhoea subsided and he passed about a dozen loose yellowish stools, which smelled badly. He lost about 20 pounds in weight. Faecal cultures and radiological investigations were negative. Fat content and xylose absorption were normal. No blood or mucus was ever found. He greatly feared that he might have developed a carcinoma. He seemed to do best on Prednisolone. Eventually a year later when he went to London he sought opinion there; the best diagnosis that could be made was a confirmation of ours that it might be ulcerative colitis. Like the other patients, he made a complete recovery.

In 1964 I published my findings on 16 cases of gastroenteritis, all European, seven of whom had the acute type of illness just described. At the time I linked the disorder with a gluten sensitivity, but I now doubt this view and I would prefer to regard it as an infection in the first instance. My account of the acute type was as follows:

"Fairly typical of the disease in many of the cases is an acute onset indistinguishable from an

acute gastroenteritis. This acute phase is accompanied by watery diarrhoea, but after a few or several days the stools become less watery, more formed and soon assume the yellow, more bulky, soft stool so typical of this disturbance."

Summary

(1) Acute gastroenteritis one of the most common acute disorders encountered in the African. Although it is far more commonly encountered in children, it is by no means uncommon in the adult in whom it may occasionally prove fatal. There were five deaths in a series of 48 cases admitted to the medical department of the University College. Although more adult cases are met with in the warmer months of the year, the disease is by no means uncommon in the cold season.

(2) A peculiar form of gastroenteritis is recorded in the European adult of Rhodesia. It affects the male and female adult equally. It has a characteristic acute onset with vomiting and the passing of watery motions. A little mucus or blood may be noticed later, but this is unusual. After several days the acute diarrhoea becomes less, but the patient continues to pass loose motions a few or more times a day. The condition may last for several or more months, but it always seems to clear entirely.

References

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Acknowledgments

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