Table 2. The toxic effect of HL ...om on neutrophil:lymphocytes ratio

Patient	Total WBCs	Neutrophils (%)	Lymphocytes (%)	Other WBCs
1	9000	96	4	—
2	16000	89	10	1 (Eos)
3	7600	88	12	—
4	6200	80	20	—
5	2500	90	7	3 (Eos)
6	9000	90	10	—
7	11800	78	18	3 (bands) + 1(Eos)
8	12000	86	14	—

CHLORPROMAZINE

Chlorpromazine has been used for treatment of nausea, vomiting and other CNS problems. The single case of nocturia responded well to imipramine. Diazepam and phenobarbitone can be administered if the patients have convulsions.

ANTIVENOM

As the venom quantity has been too small to be extracted and lyophilysed, there is no antivenom available so far. Theoretically, the HL venom, in contrast to *Androctonus crassicauda* and other species of the family Buthidae (Radmanesh 1986), can induce antibody formation so it is possible that an effective antivenom could be developed if technical problems were overcome.

Antihistamines are not helpful and their administration may lead to drowsiness and confuse the physician in evaluating the general condition of the patient.

Adequate hydration and alkalinization of the urine by addition of sodium bicarbonate to each i.v. fluid bottle are recommended in haemolytic patients in order to prevent haemoglobin deposition in renal tubules.

BLOOD TRANSFUSION

Blood transfusion may be required in severe haemolytic patients. Packed RBC is preferred in order to prevent volume overload.

DIURETICS

In those who have good urine output, dextrose 5% may act as a diuretic. Some of the patients may have already developed acute renal failure and anuria before admission. In these patients the urine output should be evaluated first. If urine volume is low or they are anuric, then they should not receive more than 250 ml i.v. fluid and should immediately receive 40 mg frusemide i.v. If there is no urine flow, the frusemide dose can be gradually increased to 200 mg or more; if no urination takes place, the patient should be prepared for dialysis. Serum potassium in anuric patients, because of both haemolysis and renal failure, may soon reach a toxic level and so should be monitored. The patients can be discharged as their general condition improves; with no fever, normal appetite, improved skin rash and cleared urine colour. Local oedema and cellulitis may persist for a few weeks and can be followed as outpatients.

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Household faecal contamination and diarrhoea risk

Aung Myo Han and Kyaw Moe

Department of Medical Research, No. 5, Ziwaka Road, Rangoon 11191, Burma

Summary

We conducted a study in a low-ranking socio-economic community in Yangon, Myanmar (Burma) to determine the source of household faecal contamination (HFC), devise an index, and associate this index with diarrhoeal incidence in children. Observation was used to collect information on the sources of household faecal contamination (HFC) and a twice-weekly monitoring scheme was employed for determining diarrhoea incidence. A valid household faecal contamination index (HFCI) was developed using three sources of HFC: going about without footwear, indiscriminate defaecation near or under the house, and absence of latrine. The risk of diarrhoea significantly increased from 4.21 to 8.66 per 1000 child-days ($P < 0.001$) when HFCI increased from 0 to 3. Although the adjusted rate ratios for the three levels of HFCI were approximately equal to 2.00 (2.16, 1.77, 2.14), they were not statistically significant. Further studies are needed to corroborate the results of what seems to be the first study attempting to associate HFC with diarrhoea incidence.

Introduction

In Myanmar, as well as in many developing countries, many people do not have access to adequate methods of human faecal waste disposal (Esrey & Habicht 1986). In these communities, faecal wastes containing enteric pathogens contaminate houses, yards and the neighbourhood through indiscriminate defaecation. This greatly facilitates the spread of major infectious agents of diarrhoea which are shed via the faeces. Although adverse effects of environmental faecal contamination have been speculated upon, we are not aware of any study looking into the association between this contamination and diarrhoeal incidence. In this study, our aim was to determine the sources of household faecal contamination, devise an index, and associate this index with the diarrhoeal incidence in children.

Correspondence: Dr Aung Myo Han, Epidemiology Research Division, Department of Medical Research, No. 5, Ziwaka Road, Yangon 11191, Myanmar (Burma)

Materials and methods

The study was conducted in Lay-Daung-Kan ward of Thin-Gun-Kyun township in Rangoon from January 1986 to December 1987.

After taking the ward census, an age-stratified cohort of 240 children (80 each from age groups 0–5, 6–11, 12–17 months) was selected at the beginning of the study. A dynamic cohort of 240 to 310 children between 0 and 23 months was maintained by taking in new children (approximately 60 to 70) aged 0–5 months every 6 months and taking out those who had reached 24 months of age.

The study included children from 392 households in the ward. The information collected from them were: demographic and socioeconomic data; observational data on household faecal contamination and diarrhoeal occurrence data.

DEMOGRAPHIC AND SOCIOECONOMIC DATA

The demographic and socioeconomic data collected include age and sex of each child, maternal age and education, total monthly family income, paternal education and occupation and child's breast feeding status.

OBSERVATION FOR HOUSEHOLD FAECAL CONTAMINATION

Observations were made of household faecal contamination (HFC) by five properly trained

Table 1. Household faecal contamination index based on the three sources of faecal contamination: each score is 'high' if one or more elements of the contamination source listed is present

Household faecal contamination source	'High' score if
Family member(s) going about outside the house without footwear during the observation period	Present
Latrine type	There is no latrine It is a surface* or a full pit latrine
Faecal contamination	There are one or more faecal mounds present under or near the house

*Surface latrine is a raised platform with a defaecation hole through which excreta fall onto the ground.

observers in a standardized manner. This was done towards the end of 1987 assuming that dirty households will remain so regardless of time and season. Observations were made on (1) whether household drainage existed and whether it was properly maintained; (2) presence or absence of facilities for washing feet before house entry; (3) whether or not any of the family members (including young children) were going about without footwear outside the house within the observation period; (4) latrine type, and (5) presence of human faeces under or near the house (faecal contamination).

These five sources of HFC were initially considered to be important for constructing the household faecal contamination index (HFCI). However, after determining their discriminative power using the item analysis technique (Goode & Hatt 1956), only three items: (1) without footwear; (2) latrine type, and (3) faecal contamination, were found to have high or acceptable levels of discriminative power (i.e., >0.5) with faecal contamination having the highest discriminative power (2.5).

The HFCI was constructed using these three sources of HFC and they were scored as either 'low' or 'high' according to whether one or more element of the contamination source was present (Table 1). We quantify HFC by the total number of high scores. HFC was considered to be great when three sources of HFC were simultaneously rated as high (HFCI = 3).

DIARRHOEA MONITORING

Twice-weekly monitoring for diarrhoea (either Monday and Thursday or Tuesday and Friday) was done on all the study children for 2 years. Monitoring was done by four properly trained field-workers who were not aware of the study hypothesis. The reliability of their surveillance data was cross-checked once with those collected independently by the staff from the Department of Medical Research (DMR) and was found to be quite comparable. Diarrhoea was defined as described by Newell (1965) and at least 7 days of normal stools should separate each episode.

ANALYSIS

The analysis was done in several stages: (1) construction of a numerical index of HFC; (2) investigation of a relationship between HFCI and diarrhoea incidence, and (3) establishment of the HFC-diarrhoea association after controlling for confounding factors.

Groups with HFCI = 1–3 were compared with the reference group having HFCI = 0 after calculating density type incidence rates for these groups. The association of HFCI and diarrhoea was established by determining the crude and adjusted incidence density ratios (Kleinbaum *et al.* 1982).

The important confounders, maternal education (MED) and socioeconomic index (SEI) (an index devised by DMR, taking into account paternal education, occupation and family income and with a scale of 1–4), were controlled by stratified analysis after stratifying the HFCI groups 0–3 according to the arbitrary cut-off points of these confounders (middle school $\geq$ MED *vs* > middle school; SEI = I, II, III *vs* IV). This analysis was done on only 273 households because information for the stratifying variables was not known for the remaining 19 households and also because it makes no difference whether they were excluded or included assuming that they belong to either of the two groups of the stratifying variables. The statistical calculations were made on an HP41CV programmable calculator using the programs on comparison of rates and analysis of $2 \times K$ tables by Abramson and Peritz (1983).

Table 2. Diarrhoea incidence rates (ID per 1000 child–days), crude rate ratios (CRR), adjusted rate ratios (ARR) and 95% confidence intervals (CI) by household faecal contamination index (HFCI) in 392 households with 0–23 month children, Yangon, 1986–1987

HFCI	CD*	ID†	CRR	95% CI	ARR‡	95% CI
0	19021	4.21	1.00		1.00	
1	53950	4.80	1.14	(0.87–1.49)	2.16	(0.75–6.20)
2	54318	4.95	1.18	(0.91–1.53)	1.77	(0.51–6.13)
3	19869	8.66	2.06	(1.58–2.68)	2.14	(0.21–22.02)

*CD, Child–days of observation.
†Chi-square test for trend = 29.80 ($P < 0.001$).
‡Adjusted for socioeconomic index and maternal education.

Results

Over the 2 years, the dynamic cohorts came from 484 households. From these, HFC data were obtainable from only 392 households (81%). The remaining 92 (19%) were either untraceable because they moved out from the ward (88 (18%)) or dropped from the study because of child's death (4 (1%)). However, the demographic and diarrhoea incidence data in the included and excluded households were similar.

Among the households included, 18% had no proper drainage; 34% had one or more household members going about without footwear; 72% had insanitary or no latrine; and 41% had human faeces under or near the house. The percentage distribution among them of HFCI 0, 1, 2, 3 were 15, 36, 36 and 13%, respectively.

RELATIONSHIP BETWEEN DIARRHOEAL INCIDENCE AND HFCI

When the study households were grouped according to the HFCI, the risk was greater when the HFCI increased: when it increased from 0 to 3, the associated diarrhoea incidence rate significantly increased ($\chi^2 = 29.80$; $P < 0.001$) from 4.21 to 8.66 per 1000 child–days. Likewise, the rate ratios increased from 1.14 for the group with HFCI = 1 to 2.06 for HFCI = 3. The crude diarrhoea rate ratio (CRR) in children from households with HFCI = 3 was significantly greater than those with HFCI = 0 (RR = 2.06; 95% confidence interval (CI) = 1.58–2.68).

However, after controlling for confounding, adjusted diarrhoea rate ratios (ARR) (2.16, 1.77 and 2.14) were higher than their crude values but not statistically significant (Table 2).

Discussion

Among the five sources of HFC considered in this study, only without foot-wear, insanitary (or without) latrine, and faecal contamination were found to be important correlates and a HFCI was constructed using these three sources only. The role of not wearing footwear as a source of HFC has not been reported although a study has speculated that footwear may be important in transporting salmonellosis into homes (Haddock & Malilay 1986).

Although the adverse effects of HFC appear in the literature (Esrey *et al.* 1985; Esrey & Habicht 1986; Anonymous 1986) we are not aware of any study associating this variable to diarrhoeal incidence. We have shown that children in households with a high HFCI have a greater risk of diarrhoea.

It is necessary to consider to what extent the study findings might have been due to biases.

Since observation was used there was probably little bias in the measurement of the exposure variables. We did reliability checks as well as correlating the HFCI with the hand-washing behaviour scores of mothers in the households and found that the two were highly correlated ($r = 0.9$; $P < 0.001$). In the same way, bias in the monitoring of diarrhoea was minimal because HFC data were collected towards the end of the study when most of the diarrhoea data had been collected and also because of the reasonably high reliability of the surveillance data.

There was probably no selection bias in the study because characteristics of the low and high HFC groups, as well as those included in and excluded from the study, were similar.

Only MED and SEI were used for the control of confounding because these two variables were thought to be the most important potential confounders. Breast-feeding and hand-washing behaviour were not controlled because the majority of the children were breast fed (96%) and most of the mothers did not practice hand-washing (80%).

The major limitation of this study might be the collection of HFC data towards the end of the study rather than at the beginning. We assume that those who have poor personal and domestic hygiene will remain so regardless of time and season. This is supported by the fact that HFCI was highly correlated to the hand-washing behaviour (taken as a proxy for personal hygiene). Furthermore, it is unlikely that diarrhoea occurrence might have influenced the collection of HFC data because exposure information was collected by observation rather than by recall.

We have developed a HFCI that seems to be valid and that can be used in identifying children in households with a higher risk of diarrhoea. We have identified three important sources of HFC: not wearing footwear, indiscriminate defaecation near or under the house, and absence of latrine. The risk of diarrhoea was greater among those with all the three sources of HFC than those having either two or one. Our findings need to be corroborated by further studies.

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Glutathione and related enzymes in fascioliasis before and after treatment with bithionol

S. Z. Abdel-Rahman,* A. M. El-Sharkawy,* L. M. Abou-Bachat and A. Salemt

*Departments of *Applied Medical Chemistry and †Parasitology, Medical Research Institute, Alexandria University, Egypt*

Summary

The present work is an update evaluation of the glutathione status in patients with established fascioliasis before and after treatment with bithionol. Blood glutathione (GSH), erythrocyte glutathione S-transferase (GST) and serum γ-glutamyl transferase (GGT) activities were studied. After treatment, the variations observed in these parameters were restored to the corresponding normal control values confirming the toxic features resulting from fascioliasis and suggesting no adverse effect of bithionol on the parameters studied. We recommend the use of serum GGT, blood GSH and erythrocyte GST for the early detection of therapeutic response in fascioliasis.

Introduction

Fasciola hepatica and *gigantica* are trematodes that inhabit the bile ducts of various mammals including man. The parasite causes the destruction of the liver tissue and induces hyperplasia of the bile ducts causing localized or generalized toxic and allergic reactions (Facey & Marsden 1960; Wei 1984).

Several drugs have been considered for the treatment of fascioliasis. Recently in Egypt, bithionol (2,2-thio-bis-4,6-dichlorophenol) was recommended for the treatment of the parasite (Farag *et al.* 1988).

Glutathione (GSH), a tripeptide thiol found in virtually all cells, functions in metabolism, amino acid transport and cellular protection. It participates in the reduction of disulphides of other molecules and conjugates with compounds of exogenous and endogenous origin (Meister 1983). Erythrocyte GSH protects haemoglobin and other critical proteins from peroxidative injury (Fairbanks & Klee 1986).

It is possible that GSH and its metabolic activities play an important role in the development of the toxic features associated with fascioliasis. This led us to investigate the GSH content and glutathione-S-transferase (GST) enzyme activity in erythrocytes of patients with fascioliasis as well as γ-glutamyl transferase (GGT) activity in the sera of these patients. The latter enzyme reflects, at least in part, the glutathione metabolism in the liver. The same parameters were studied after bithionol therapy.

Dr Sherif Abdel-Rahman, Department of Pathology; F-05, University of Texas Medical Branch, Galveston, Texas 77550, USA.

Materials and methods

CHEMICALS

All fine chemicals used in this study were Sigma products (Sigma, St Louis, MO, USA). All other chemicals were analytical grade and purchased from Merck (Darmstadt, FRG) unless otherwise specified.

COLLECTION OF SAMPLES

Ten patients aged from 5 to 12 years old were enrolled in the present study. They were referred from the Medical Centre in Abis and were admitted to the Medical Research Institute Hospital. They were examined clinically and their stools searched for parasites. After establishing the diagnosis of true fascioliasis by repeated faecal examinations they were subjected to the following tests together with 10 control subjects, who were healthy children of workers in the M.R.I. and in the same age group as the patients.