VOMITING AND HYPOGLYCAEMIA

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Patients admitted with diarrhoea and vomiting are occasionally found to be hypoglycaemic. This is not an uncommon finding particularly in children who are suffering from malnutrition and diarrhoea.

In November and December, 1974, 12 cases were admitted into C.R.L. Hospital with hypoglycaemia associated with diarrhoea. During the same period, 11 patients were admitted with vomiting only associated with profound hypoglycaemia. The clinical presentation and other features of these patients with vomiting were different from those associated with diarrhoea. Bacteriologically all of them were negative for any pathogen. As this was a small epidemic, proved fatal in most of the cases, investigations are in progress to explain the pathogenesis of this type of hypoglycaemia associated with vomiting only. Some interesting features of this particular cases will be described.

<u>Materials</u>

Twelve cases of diarrhoea and 11 cases of vomiting all with hypoglycaemia are taken in this discussion.

Methods

All patients were admitted into the hospital. The following investigations were carried out on admission:

1. Blood:

Total WBC count, Differential count, Haematocrit percentage. Electrolyte, Sugar, Specific gravity, Creatinine, Culture.

2. Stool:

Rectal swab for culture, stool for Microscopy, and culture.

3. Urine:

Routine analysis, bile salt and pigment, culture. Heavy metals.

4. Lumber puncture for CSF examination and culture.

Management

Usual nursing care was maintained. Intake and output was recorded. Fluid and electrolytes were maintained. A broad spectrum antibiotic cover was given in all cases as felt appropriate. I.V. glucose was maintained regularly.

Results

Vomiting group

Out of 11, 9 were children and 2 were adult. Ten were unconscious and 6 were convulsive. One was conscious but was drowsy. Dehydration was mild in 8 and moderate in Radial pulse was not perceptable in 3. None had temperature and lungs were congested in 7; pupils were dilated but reactive to light. Liver and spleen were not palpable. Nutrition was good in all the cases. History of onset varied from 2½ hours to 19 hours till the time of admission. Eight had no detectable glucose in the blood and 3 had blood sugar below 50 mg%. Seven were moderately acidotic. Other electrolytes were normal in all of them. Blood count was done in 9 and all had high total WBC count with high polys. All had a total WBC count above 12,000/cmm, 3 cases above 20,000/cmm and 4 cases above 30,000/cmm. Blood culture was taken in 3 cases and grew no pathogen. Cerebrospinal fluid was available in 6 cases and were negative for Pandy's, cell and pathogen. Stool and rectal swab culture were negative in all the cases. Serum cholinestearase activity done on 5 cases and were within normal range. Needle liver autopsy was done on 2 cases and the results are awaited. There were 7 deaths (5 with 0 mg/s sugar), 2 signed out and 2 survived. Two patients had 4+ proteinurea and 1 was pregnant with normal blood pressure.

Those 2 patients who survived, had an eventful hospital course. One remained unconscious for 5 days, developed temperature on the 2nd day of hospitalization which gradually subsided. The other regained consciousness after 4 hours but remained lethergic and restless for 3 days with abdominal pain and tenderness. One was discharged on the 7th day and the other was discharged on the 9th day of hospitalization.

Diarrhoeal group

All 12 cases were children with a mean age of 3.2 years. Eleven cases were pulseless, 3 with mild dehydration, 4 with moderate dehydration and 4 with severe dehydration. All had varying degree of malnutrition and 6 of them were severely malnourished. Mean plasma specific gravity was 1.020. Five were semiconscious and 5 unconscious associated with convulsion. Severe acidosis was in 2 and hypokalaemia in 3 cases. Other electrolytes were normal in all. Blood glucose level was below 20 mg% in all, out of them 6 had no detectable glucose. Bacteriologically 6 patients had Shigella flexneri in the stool, 1 had cholera and one had bronchopneumonia. Six patients expired, of them 4 were with Shigellosis.

Discussion

Hypoglycaemia associated with diarrhoea is not uncommon in children with malnutrition. In the recent years the nutritional status of our children has gone down and it is met more frequently in them. Prolonged diarrhoea, electrolytes imbalance, infection and other factors, complicated with malnutrition were probably the main causes of death in them. Prolonged vomiting with restricted food intake may sometimes cause hypoglycaemia? The 11 patients remaining well till the onset of vomiting died within several hours. There was no complaint of temperature, abdominal pain or tenderness but developed drowsiness, followed by convulsion, unconsciousness. No pathogen could be isolated from the blood, stool and CSF. There were reports of vomiting in the household in 3 of the cases and in each, a single member was affected and one died.

Deaths due to vomiting in the villages were heard. There were 20 cases reported in Matlab. Dietary history of taking curry of a green leafy vegetable called locally 'Ghagra' was obtained in 2 and in the rest there was nothing particular. Two other cases who took Ghagra, developed vomiting, convulsion and unconsciousness, and expired in the hospital. They were not hypoglycaemic and are excluded from this discussion. Ghagra usually grows on the banks of the river and canals in November and December and the green tender leaves are used as curry in the villages. The leaves of the older plants are not used. An alkaloid of such plant is obtained and further investigations / with the alkaloid is pending.

There is another plant growing together with Ghagra. It may be possible that the other plant also was picked up with Ghagra and cooked together by mistake. The clinical presentation and other features of these cases resemble the vomiting sickness of Jamaica due to Ackee poisoning or due to some other herbs used as Bush Tea. Ackee is not grown in Bangladesh but the herbs mentioned above might be playing a role in such sickness. On the otherhand, the clinical presentation and other features close resemble Reye's syndrome. The cases reported by Lloyd C. Olson et al(7) and MAJ. M. Glasgow et al(7,8) from Thailand, closely resembled our cases. About the other findings mentioned by them we are not aware of it as the facilities for special biochemical tests were limited at the time. Moreover they have (6,7,8) suggested Aflotoxin by certain strains of Asperigillus flavus as the causative factor.

The whole epidemic was for a limited period of time and appears seasonal. So plans have been drawn for further studies of such cases in future. The following tests will be done to explain the pathogens.

- 1. Liver biopsy
- 2. Liver function tests-SGOT, SGPT, Serum Total Bilurubin
- 3. Blood for Ammonia level, Prothrombin activity
- 4. Autopsy in cases when available
- 5. Study of the plant alkaloid
- 6. Epidemiological studies

References

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- 5. Evans, K.L., Arnold, L.E. Experimental Studies of Poisoning with Ackee. Trans. Roy. Sci. Trop. Med. Hyg. 32, 355, 1938.
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- 7. Encephalopathy and Fatty degeneration of the viscera in Northern Thailand, Clinical Syndrome and Epidemiology Lloyd C. Olson et al. Paediatrics, p.707, Vol. 47, No.4, April 1971.
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PATIENTS IN DIARRHOEA GROUP

Age Yr.	Body wt.	Clinical Observation			Electrolyte	Serum	Blood	TWBC	Bacteriological	Outcome
		Pulse	Dehydration	Mental status	abnormality (mEq/1)	sp.gr.	Sugar (ng%)		diagnosis	Outcode
3/12	3.3	0	Moderate	Drawzy	No	1.013	3	N-D	0	Expired
6	11.6	0	Moderate	Unconscious seizure	CO ₂ -9 mEqpl	1.023	17	H-D	Sh.Flex	Expired
5/12	4.8	±	Severe	Unconscious	Na-126 "	1.018	5	8500	Sh.Flex	Expired
6	7.4	±	Moderate	Unconscious 🔑	K-2.6 "	1.018	16	42000	Sh.Flex	Expired
21/2	5.0	0	Moderate	Semoconscious gasping	No	1.021	0	N-D	Sh.Flex	Expired
14/12	6.0	0	Mild	-do-	K-1.4	1.015	4	12500	0	Expired
8	11.0	0	Severe	Semiconscious seizure	No	1.021	0	N-D	0	Survived
3	8.4	±	Severe	O.K.	CO ₂ -11	1.026	0	3400	Sh.Flex	Survived
4	9.0	o	Severe	Unconscious	No	1.024	11	18500	Cholera	Survived
2	, 7.8	0	Mild	Semiconscious	K-2.1	1.014*	0	10000	Sh.Flex	Survived
11	5.8	±	Mild	Unconscious	lio	1.024	0	27650	O(Br.Pnuemo -nia)	Survived
4	7.8	100	Hoderate	Semiconscious	No	1.027	0	H-m	0	Survived

*Human plasma infused.

HYPOGLYCAEMIC PATIENTS IN VONITING GROUP

Age yr.	B.Wt. kg.	Clinical of Fulse	observation o	n arrival Mental status	Pl.sp.gr.	Electrolyte abnormality mEq/1		TwBC/cmm	Outcome
3	9.5	Present	Moderate	Convulsion unconscious	1.022	None	0	29,000	Expired
12	24	0	Mila	Unconscious convulsion	1.028	^{CO} 2 ^{-10.5}	0	42,000	Expired
7	16	0	Mild	Unconscious convulsion	1.024	002 18	49	33,000	Expired
8	16	Present	Mild .	Unconscious	1.026	^{CO} 2 14	0	31,000	Expired
11	23	Present	Mild	Unconscious	1.024	^{CO} 2 17	32	-ND	Expired
16	28	Present	Moderate	Unconscious gasping	1.030	002 16	0	36,000	Expired
30	Not taken	0	Mild	Unconscious convulsion pregnant 28 wk	1.026	002 15	0	24,600	Expired
8	14.5	<u> </u>	Moderate	Unconscious convulsion	1.028	None	7 mg	17,100	DORB
7	18	±	Moderate	Uncare lous On alcion	1.031	^{CO} ₂ 13.6	0	-ND+	DORB
12	⇒24	Present	Mild	Droney	1.029	⁰⁰ 2 13	0	13,000	Survived
12 .	23	Present	Hila	Unconscious convulcion		co ₂ 15	0	29,900	Survived

TABLE 1

Vomiting only group Diarrhoeal group

Total No.11

Total No.12

Children: 9

Children : 12

Age: 1-5 = 1

Age: 0-5 = 9

6-15= 8

6-10= 3

Adult:-2

TABLE 2

Vomiting only group	Diarrhoeal group
Total case = 11 .	Total case = 12
Blood sugar 0 = 8	Blood sugar 0 = 6
Blood sugar < 50 mg% = 3	Blood sugar < 20 mg% = 6
 OUTCOME	
Expired - 7	Expired - 6
DORB - 2	Survived - 6
Survived - 2	

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