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Diarrhoea is one of the most important public health problems and a major cause of death among infants in the developing world. In vast majority of cases, diarrhoea in infants is self limited and caused by viral or known bacterial pathogens which are identified by routine stool culture. In some infants, however, inspite of the ordinary supportive measures instituted during the diarrhoeal episode, diarrhoea continues for protracted period. Some of these children are found to have developed intolerance to carbohydrate component(s) of milk at the same time, leading to perpetuation of diarrhoea with its consequences.

According to present study, conducted at the departments of Pediatrics & Biochemistry, M.L.B. Medical College, Jhansi, prevalence of carbohydrate intolerance in protracted diarrhoea was 46.67%. In 1969, Chandra et al used stool pH estimation along with oral lactose feeding test to diagnose lactose intolerance. Thus, he detected lactose intolerance in 54% of cases.

In 1972, Reddy et al used similar methods and detected intolerance in 37% of cases. In 1976, Udani
et al reported a prevalence rate of 9.32%. Larcher et al in 1977, using stool pH estimation along with estimation of reducing agent content of stool, reported a prevalence rate of 12%. In 1982 Ghai et al reported a prevalence rate of 24% for carbohydrate intolerance.

Bhave et al in 1983, employed stool chromatography and reported a figure of 35.7% prevalence rate. In 1984 Davidson et al used the much acclaimed Breath hydrogen test and detected 50% prevalence rate.

These studies only show that the incidence of carbohydrate intolerance is highly variable and depends on the sensitivity of the diagnostic procedures employed as well as the season of study. Perhaps the breath hydrogen estimation test will in future be established as the most sensitive test for early and correct diagnosis of carbohydrate intolerance, but it is very difficult to use in infants.

According to Chandra et al (1969) viral diarrhoeas are more often implicated in the causation of lactose intolerance, and these commonly occur during the winter months. But in this study which was conducted over a period of one year there was uniform distribution of cases with minimal clustering during the summer months.
The present study showed that carbohydrate intolerance was, predominantly, a problem of the latter half of infancy. 52% of the cases in this study were between 6-12 months of age with peak incidence at 9 months. In the study conducted by Ghai et al in 1982 the peak incidence of carbohydrate intolerance was 10.63 months.

That male children are more predisposed to develop carbohydrate intolerance, has been found in several other studies. The male to female ratio as obtained in this study was 2.2 : 1. Trounce et al (1985) found a male to female ratio of 3 : 2 in cases of carbohydrate intolerance. The male preponderance could be a reflection of the traditional Indian family taking more interest in the male sib in all spheres including medical attention. It could also perhaps be explained by the fact that the gene controlling synthesis of immunoglobulin is located in the X chromosome. Since females are homozygous for X chromosome, they have higher levels of immunoglobulin with subsequent higher levels of resistance against invasive micro-organisms.

Out of 75 cases of chronic diarrhoea, 71.4% belonged to the lower socio-economic strata. This is, possibly, because poverty is associated with malnutrition, higher incidence of infection, lack of education and proper hygiene. Parental ignorance and poverty lies at
the root of the problem. 52% of the patients in this study had either normal nutritional status or they fell in grade I malnutrition. Grade II to IV malnutrition was present in the remaining 48% cases.

In 14.88% of the infants above 6 months of age, grade IV malnutrition was observed. The above results emphasize the increased incidence of malnutrition above 6 months of age, probably associated with weaning diarrhoea and the fact that sugar intolerance can occur in all nutritional groups.


Protein energy malnutrition is associated with diminished neutrophil function, chemotaxis and phagocytosis, decreased opsonisation, diminished T cell response, diminished secretory IgA levels which tend to perpetuate the infection and further aggravate malnutrition. The lower levels of proteins in protein energy malnutrition
prevent quick regeneration of the destroyed intestinal epithelium.

Antibiotics had been administered to 74 percent of the cases who developed carbohydrate intolerance prior to admission. Kumar et al (1975) reported that 71 percent of infants had received one or more antibiotics. Davidson (1984) reported that 37% had received antibiotics. Perhaps the antibiotics by altering the flora of the intestine, and by destroying the brush border epithelium also had a role to play in the perpetuation of the diarrhoea. Administration of antibiotics indiscriminately during an episode of diarrhoea induces destruction of the intestinal epithelium and subsequent sugar intolerance.

Nearly 12% of children in this study were on breast alone during the diarrhoeal episode, while 57 percent received Cow's or buffalow milk in addition to breast milk. Since the lactose component of breast milk is higher, infants on breast milk are, probably, more prone to develop sugar intolerance. Two infants who were exclusively given artificial feeds had also developed sugar intolerance.

This study also showed that breast feeding did not protect an infant from developing carbohydrate intolerance.
Majority of the children (85%) with sugar intolerance had diarrhoea of watery consistency, while it was semi-solid in consistency in the rest of cases under study. Other important symptoms were: fever, vomiting and abdominal distension which together were seen in 40% cases. Stool frequency was more than 10/day in 57% cases. Diarrhoea and frequency of stool in sugar intolerance has been attributed to bacterial fermentation of undigested intestinal contents and the osmotic action of large amount of unabsorbed sugar.

Perianal excoriation was found to be a sensitive indicator of the presence of sugar in stool. It was present in all the cases which were positive for sugar, in the present study. Ghai et al (1982) had emphasized this fact. However, the severity of perianal excoriation did not correlate with the extent of sugar malabsorption. Perianal excoriation subsided spontaneously on withdrawal of the offending sugar.

Associated systemic disease, at the time of admission, was seen in 17% of cases. However, the disease showed no correlation to the development of sugar intolerance. The associated diseases were mainly: Asthmatic bronchitis, Bronchopneumonia and severe anemia. Systemic complications would have, probably, contributed to sugar intolerance by necessitating further administration of antibiotics.
Lifshitz et al (1971) emphasized the fact that systemic infection prevented the gut from regenerating destroyed epithelium. Destroyed gut epithelium, besides increasing the sugar intolerance, facilitated rapid entry of pathogenic micro-organisms and their toxins into the blood stream and produced septicemia, endotoxemia, hypersensitivity to various proteins etc.

The macroscopic examination of stool showed that stool were greenish yellow, foul smelling and frothy in 65.7% of cases. Udani et al (1976) had emphasized that stool are large, watery, at times greenish or yellowish in colour, usually had sour smell and often contained mucous in cases of sugar intolerance. Further, authors observed that stool was passed explosively and in the fresh state it was frothy. Ansari et al (1979) have reported that in 76% of cases stool were large, frothy and sour smelling.

Microscopic examination did not revealed any fat globules or pus cells in any of the case (in the present study).

Presence of large numbers of fat globules on stool microscopy in children with sugar intolerance would have suggest concomitant fat malabsorption. Lifshitz et al (1971) reported that steatorrhoea could sometimes be associated with sugar intolerance.
Giardiasis was not seen in any of the cases. It is an important protozoon which may cause disaccharide malabsorption and prolongation of diarrhoea.

Fat malabsorption could be due to deficiency of the enzyme glucosyl ceramidase, which splits glucosyl ceramide present in milk fat vesicles and exists as a complex with lactase, or due to deconjugation of bile salts as a result of colonisation of small intestine by anaerobes.

The pH of the stool was found to be a useful guideline towards the diagnosis of sugar intolerance as observed in the present study. The mean pH of the stool in this study was 5 in 45.7% and 6 in 31.4% cases.

Durand et al (1961) and Davidson (1967) stated that stool pH of less than 6 was characteristic of disaccharide malabsorption and that this was a reliable indicator. Lifshitz (1971) who has done exhaustive work on the problems of sugar intolerance emphasized that stool pH was a reliable indicator. Udani et al (1976) in their study reported that stool pH was below 6 in 61% of cases. It was also noted by the authors that greater the amount of sugar that was detected in the stool, lesser was the pH value.

Ansari et al (1979) in their study reported that pH was less than 6 in 67% of cases of sugar intolerance.
However, oral intake of furazolidine and neomycin, and
delay in the performance of test tended to increase the
stool pH despite the existence of sugar intolerance.
On the other hand infants with metabolic acidosis
passed stool with acid pH, that gave false positive
indication of sugar intolerance.

McMichael et al (1980) reported that stool pH
was highly fluctuant and unreliable in sugar intolerance.
Bhave et al (1983) also agreed with the above statement
of McMichael. However, consensus of opinion at present
is that stool pH could be used as a rough and quick
screening test for other diagnostic measures to follow.

Benedict's test for reducing agent in stool
showed 0.25 - 0.99 gm% sugar in 71.4% cases. However,
presence of reducing agent in stool was not synonymous
with sugar intolerance as nine false positive cases were
seen which ultimately did not show evidence of sugar on
chromatography.

Udani et al (1976) has pointed that that there
are many factors which affect the outcome of this test.
During an episode of diarrhoea, if the child is on oral
rehydration solution some amount of glucose contained
in the oral rehydration solution would be excreted in
the stool as a result of intestinal hurry. Moreover,
if the lactase deficiency of the intestinal epithelium
was only partial, part of the lactose was split into its component monosaccharides that appeared in the stool to give a positive test with Benedict's reagent. In all the cases of present study examination of acid hydrolysed specimen of stool filtrate was done in order to detect sucrose in stool. In this particular study, the sensitivity of Benedict's test was found to be 94.6% while specificity was only 80.6%. Singh et al (1991) in their study pointed out that the specificity of Benedict's test was 78%.

It was found in the present study that Rubner's test was a more specific test as compared to Benedict's. It's specificity in sugar intolerance was 95.1% as compared to 80.6% in Benedict's test. But, it was found to be less sensitive test as compared to Benedict's. Sensitivity was 87.5% as compared to 94.6% in Benedict's test. It was also a simple test requiring two reagents and could be done on the bed side of the patient. Singh et al (1991) reported that modified Rubner's test was more specific test for the detection of sugar in stool.

In this study oral lactose loading test was not done, as many authors have pointed out various disadvantages which limit the use of this diagnostic test. Lactose loading test may result in severe bouts of diarrhoea and requires repeated collection of blood samples which would be unacceptable to many parents.
Udani et al (1976) pointed out that sugar loading test was rarely necessary in routine practice and would be useful only in cases which showed a clinical picture of sugar intolerance and responded to withdrawal of the sugars from the diet, but did not show evidence of sugar in the stool.

Stool chromatography, though a very tedious procedure was found to be an excellent, highly sensitive and reliable indicator of the presence of sugar intolerance. Even mild degrees of sugar intolerance could be detected by this method. In this study lactose alone was present in the stools in 65.7% of cases; 14.3% had lactose in addition to galactose. Lactose was observed in 11.6% cases in addition to glucose, lactose and sucrose were seen in 2.8% cases. In only 5.6% of the cases was triple sugar intolerance detected. In half of the cases it was lactose, sucrose and galactose intolerance, responsible for the clinical condition.

Stool chromatography has been hailed as a cornerstone procedure for the diagnosis of sugar intolerance by Durand (1961) and Haworth (1963). Moreover, since the development of breath hydrogen test, chromatography has received scant attention.
Bowie et al (1965) studied acquired disaccharide intolerance in malnutrition. Disaccharide intolerance were seen in 52% of the cases which was diagnosed by chromatography. The predominant sugars detected were lactose, glucose and galactose in varying amounts.

Udani et al (1976) in their study detected lactose in stool in 30% of cases and lactose with other sugars in 30% of cases. Sucrose alone was seen in 20% of cases, and glucose alone in the remaining 20% of cases. The intolerance to maltose and fructose was rare, in their study.

If the child was intolerant to lactose alone, it suggested a disturbance in enzymatic hydrolysis of lactose by lactase in the brush border lining the luminal surface of the intestinal epithelium. If the child was intolerant to galactose alone, that suggested complete enzymatic hydrolysis of lactose in the brush border and galactose malabsorption without glucose malabsorption. When lactose and galactose both were detected in the stool, there was, probably, partial enzymatic hydrolysis of lactose resulting in lactose and galactose malabsorption and no glucose malabsorption. When all the three sugars - lactose, galactose and glucose were detected in the stool, there was partial enzymatic hydrolysis of lactose and malabsorption of
all the three sugars. When glucose alone was detected in the stool it was due to the disturbance in active transport mechanism of glucose (Udani et al, 1976).

Joseph et al (1976) reported in their study an incidence of 30 percent sugar intolerance in refractory diarrhoea, diagnosed by Thin Layer Chromatography. Lactose alone was seen in 80% of cases while in the rest it was lactose with sucrose.

Vincent et al (1979) reported an incidence of 59% for sugar intolerance in acute diarrhoea. In 40% of cases it was single sugar intolerance being lactose while in the rest multiple sugar intolerance viz., glucose, sucrose and galactose. Furthermore, authors opined that glucose and fructose could not be differentiated by Thin Layer Chromatography.

Ansari et al (1979) reported in their study that lactose was detected by chromatography in all the cases of sugar intolerance. Besides, there was intolerance to sucrose and galactose in 40% and 20% cases respectively.

Bhave et al (1983) using chromatography reported the incidence of sugar intolerance as 35.7%. They observed single sugar intolerance in all the cases. In 70% it was lactose intolerance while glucose, and sucrose intolerance was seen in 15% cases each.
Ashoka et al (1988) reported an incidence of 68% sugar intolerance using chromatography in protracted diarrhoea. Intolerance of lactose with galactose was seen in 3% of cases. Lactose with glucose was seen in 4% of cases. Triple sugar intolerance with lactose, glucose and galactose was seen in 16% of cases. Only monosaccharides were seen in 17% of cases.