paragonimiasis was not conclusively established by identifying ova in the specimens, the clinical, radiological, serological, and epidemiological evidence supported this diagnosis and the patient was treated with Bitin-S [Bis (2-hydroxy-3, 5-dichlorophenol) sulfoxide] 15 mg/kg on alternate days for 15 doses. Bitin-S is a new derivative of Bithionol (2, 2'-Thiobis (4, 6-dichlorophenol)).

Two weeks after treatment began a second, nontender swelling developed in the left inguinal region. One month after treatment had stopped both swellings had disappeared, chest x-rays were normal, the ESR had fallen to 4 mm in 1st hour and the eosinophil count was only 0.6 \times 10^9/1 (600/mm^3).

Discussion

Human paragonimiasis is mainly a disease of the lung and the presenting complaints are usually respiratory—such as blood-stained sputum. Extrapulmonary paragonimiasis may occur in many sites including the pleura, lymph nodes, muscle, and skin, and there can be CNS complications especially with *P. westermani* (Oh, 1967, 1968). In most patients chest x-rays show changes similar to those in pulmonary tuberculosis (Ogakwu and Nwokolo, 1973). Occasionally unilocular or multilocular cysts may be seen (Nwokolo, 1972a). Diagnosis is usually established by demonstrating paragonimus ova in sputum but care must be taken to exclude tuberculosis which may coexist. Ova may also be seen in stools and pleural exudates.

The presentation of our patient was atypical because of the absence of respiratory symptoms and the presence of the soft tissue swellings which are not normally a feature of the disease. Failure to demonstrate the ova in sputum was disappointing but it was not unexpected in view of the absence of respiratory symptoms. The following criteria helped to establish the diagnosis of paragonimiasis: (1) the patient came from an area where paragonimiasis is endemic and gave a history of eating crayfish, (2) the radiological findings and eosinophilia were consistent with paragonimiasis, (3) excluding the doubtful positive Mantoux test (previous BCG vaccination) there was no evidence for tuberculosis, (4) the fasciola CFT (usually positive in paragonimiasis) was positive in serum and pleural aspirate, (5) treatment with Bitin-S was followed by complete clinical, radiological, and haematological cure.

Summary

A 2½-year-old girl recently arrived from eastern Nigeria presented with a soft tissue swelling of the infraclavicular region. Subsequent investigation revealed a cavity in the left lung associated with a small pleural effusion and leucocytosis with pronounced eosinophilia. Clinical and serological findings were compatible with the diagnosis of paragonimiasis. After a course of Bitin-S the chest x-ray returned to normal, the soft tissue changes disappeared, and the eosinophil count fell.

We thank Professor J. A. Davis for allowing us to report on his patient and Professor Nwokolo for helpful comments.

References


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Faecal excretion of oligosaccharides and other carbohydrates in normal neonates

Sugar intolerance is a common cause or complication of diarrhoea in infants. If watery diarrhoea is present from birth, or if chronic diarrhoea impairs growth in the newborn period, a diagnosis of primary lactasia may be considered. Simple tests may be used to assess the carbohydrate (Kerry and Anderson, 1964) and acid content; the presence of >0·5% reducing substances and pH < 6 may
prompt the clinician to investigate the stool for the presence of sugars by chromatography (Soearto et al., 1972; Ament, 1973).

One such investigation, in which the presence of >2% reducing substances in the acid stools of a breast-feeding infant (Case 1) had suggested a diagnosis of primary alactasia, showed large quantities of an oligosaccharide in a stool from which other sugars were absent. This finding prompted us to examine the stools of normal newborn infants to determine how often oligosaccharides and other sugars were present.

**Patients and methods**

Stools were collected from 40 normal newborn infants in the first 8 days of life. 22 were breast fed and 18 were fed on gold cap SMA. There was no significant difference between the ages in the two groups.

Stools were collected from plastic-backed disposable nappies and deep frozen within 2 hours of collection. The concentration of reducing substances was estimated with Clinitest (Ames) tablets using the method described by Kerry and Anderson (1964). The pH was estimated with pH papers (Whatman-BDH, narrow range).

Stool samples were homogenised with an approximately equal volume of distilled water then centrifuged for 10 min at 3000 rev/min in a bench centrifuge (MSE minor). An aliquot of the supernatant (10 μl) was seeded as a streak approximately 1 cm long and 1 cm from the edge of a 20 x 20 cm silica gel 60 chromatographic plate (E. Merck, Darmstadt). The chromatogram was developed for 6 hours in N-butanol:glacial acetic acid:water (75:25:6) and then dried with warm air. Sugars were identified by spraying the plate with 1% diphenylamine: 1% aniline in acetone and heating for 5 min at 150°C. The mobility and colour of bands appearing in test samples were compared with a standard solution containing 500 mg/l each of xylose, glucose, fructose, galactose, lactose, maltose, and sucrose, which was cochromatographed.

Fresh expressed breast milk, pooled and pasteurised expressed breast milk, SMA, Galactomin 17, 18, and 19, and cows’ milk were also examined for the presence of sugars by thin-layer chromatography without dilution and at a dilution of 1 in 30 in water.

**Results**

**Reducing substances and pH.** Reducing substances were present in amounts >0.5% in 14 (64%) out of 22 stools of breast-fed infants, and in 2 (11%) out of 18 stools from artificially-fed infants. This difference is significant by χ² test with Yates’s correction for small numbers (χ² = 3.94, P < 0.05). Amounts >1% were found in 4 samples from breast-fed infants and in none from artificially-fed ones. All stools had a pH between 5.0 and 6.5, and there was no difference in stool pH between the two groups.

**Oligosaccharides.** On stool chromatography a prominent band was present in a position characteristic of an oligosaccharide (oligosaccharide I Rf 8.0) in 34 (85%) samples. Hydrolysis, of stools containing only this substance in 0.1 M sulphuric acid, produced lactose, galactose, and fucose (6-deoxygalactose). The presence of the last was confirmed by thin-layer chromatography, compared with authentic 6-deoxygalactose, and by gas-liquid chromatography (Murphy et al., 1974). There was no correlation between the presence of oligosaccharide I and the presence of reducing substances or other sugars in the chromatogram. In 16 samples a less prominent second oligosaccharide (oligosaccharide II) was identifiable close to the origin with an Rf value of 3.5. Its presence did not appear to be related to the presence of oligosaccharide I, and it has not been investigated further. Both oligosaccharides were present in the stool collected from Case 1.

**Monosaccharides and disaccharides.** Galactose was the most common substance and occurred in 25 stool samples. Lactose was present in 17, and was observed in 15 of the 23 samples from infants aged less than 5 days. Fucose or glucose was present in 17 samples. In 15 of these both were present and, whereas they did not always coexist, neither was present in the absence of galactose. Fructose was present in one sample containing more than 2% reducing substances.

**Analysis of milks.** In addition to lactose, both oligosaccharides found in the stools were also present in fresh expressed breast milk, pooled pasteurised human milk, Galactomin 18, and cows’ milk although they were difficult to separate in the presence of excess lactose. Oligosaccharide bands could no longer be detected at dilution of 1 in 30. SMA and Galactomin 17 and 19 did not contain any oligosaccharides.

**Discussion**

The most common sugar identified in the stools of infants in the first 8 days of life is an oligosaccharide consisting of glucose, galactose, and fucose. Davidson and Mullinger (1970) reported the
frequent presence of substances, often in high concentrations, which they did not investigate but suggested might be oligosaccharides. Some fucose-containing oligosaccharides have been described in milk, particularly fucosyl lactose, which is present in concentrations of between 150 and 300 mg/l (Stacey, 1962). It is reasonable to assume that the predominant oligosaccharide seen in 85% of newborn infants’ stools is fucosyl lactose and, as we have shown that oligosaccharide is present in both human and cows’ milk, it is likely that its presence in the stools is of dietary origin. The infant (Case 1) was put on a lactose-free diet consisting of Galactomin 17, which does not contain oligosaccharide I, and the oligosaccharide I disappeared from the stool. This would seem to confirm the dietary origin of the oligosaccharide, and we do not agree with Counahan and Walker-Smith (1976) that stool oligosaccharides are rarely found and are primarily of bacterial origin. As fucose and glucose were seen only in the presence of galactose, it is likely that these 3 sugars are the products of the intestinal hydrolysis of fucosyl lactose as they were also found on acid hydrolysis of a stool containing oligosaccharide I. This process may be responsible for at least some of the lactose found in the stools of newborn babies.

Our findings confirm those of previous workers who have shown the frequent presence of large amounts of reducing substances in the stools of infants in the first week of life, particularly in babies who are breast fed (Davidson and Mullinger, 1970; Counahan and Walker-Smith, 1976). All but two samples had a pH < 6, and we would not recommend that stools from infants in this age group be examined for the presence of reducing substances or pH as a screening test for carbohydrate malabsorption. Previous workers have not detected fucose, and infrequently have identified oligosaccharides. Our findings may be the result of a better chromatography method for separation of these substances from other sugars. Oligosaccharides are clearly present in a large proportion of faecal samples in normal neonates. Stool chromatography may help to identify sugars in stools, but unless the source of these substances can be accurately identified, they cannot be relied upon to make a diagnosis of carbohydrate intolerance. It would seem more reliable to investigate infants for specific malabsorption of carbohydrates by giving the infant a specific carbohydrate load rather than examine randomly collected faeces which yield results not open to accurate interpretation.

**Summary**

Chromatographic analysis of sugars in the stools of 40 normal newborn infants has shown the presence of an oligosaccharide in 85% of samples. The oligosaccharide has been shown to contain fucose (6-deoxygalactose), glucose, and galactose and is present in normal breast milk. In addition fucose has also been found in the presence of galactose in some samples and is probably a breakdown product of milk oligosaccharides. These findings, which affect the interpretation of faecal sugar excretion, have not been described previously.

**References**


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