with techniques which show better correlation with clinical symptoms (such as the 24 hour continuous oesophageal pH monitoring and ‘milk scan’). The almost perfect agreement between barium examination and ultrasound reported by Naik and Moore suggests, therefore, a high false positive rate regarding clinically important gastro-oesophageal reflux for ultrasound too. This means that the information obtained may not be relied upon and the patient’s symptoms are perhaps unrelated to coexisting incidental reflux. It would have been more informative to compare ultrasound with oesophageal pH monitoring or radioisotope ‘milk scan’.

Moreover, as the authors state, clinical manifestations of reflux (vomiting, failure to thrive, anaemia, aspiration, and perhaps even ‘cot death’) are non-specific. The barium oesophagram has the advantage of offering additional important information such as swallowing incoordination, peptic oesophagitis with strictures, duodenal obstruction, midgut malrotation, or delayed gastric emptying to mention but a few conditions that may cause symptoms similar to those of simple gastro-oesophageal reflux. With current equipment these conditions would be missed were ultrasound to replace barium examinations. Ultrasound is excellent for the diagnosis of pyloric stenosis and in the appropriate clinical set up it should be performed first, at which time one may also look for gastro-oesophageal reflux.

Ultrasound is, therefore, a diagnostic tool which may be used selectively in suspected gastro-oesophageal reflux, but it cannot at present replace barium studies completely.

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Drs Naik and Moore comment:

Our paper indicated that ultrasound is a reliable technique by which to answer the question ‘Does gastro-oesophageal reflux occur?’ Our subsequent experience with the technique has confirmed this. In a patient in whom a clinical diagnosis of gastro-oesophageal reflux has been made ultrasound is an excellent means of confirming this and a barium examination is unnecessary. Ultrasound, however, does not provide comparable anatomical detail to a barium examination and when this is required a barium examination will still be needed. The number of barium examinations for reflux should be few. We are currently trying to define the indications for a barium examination in suspected reflux and would suggest the following:

1. Failure to visualise the oesophagus ultrasonically.
2. Repeated negative ultrasonic findings in a patient with strong clinical indications of reflux.
3. A patient with proved reflux in whom the development of a complication is suspected.
4. Before operation.

We have not determined the ‘false/positive’ rate for our technique; our original criteria were chosen as working guidelines and it may well be that stricter criteria will be necessary to select those patients in whom reflux is ‘clinically important’. We feel this can only be determined after more cases have been studied and the results have been correlated with the clinical findings and response to appropriate treatment.

References

Gaviscon bezoars

Sir,

We read with interest the recent paper by Sinaasappel et al on progressive vomiting in a 5 month old boy caused by a bezoar of Gaviscon. In the past 18 months we have seen three cases of Gaviscon bezoar.

A boy was born by spontaneous vertex delivery at term, her birthweight was 3550 g. At age 6 days Gaviscon was introduced to control persistent vomiting, but there was no improvement. Eight days later a barium meal examination showed gastro-oesophageal reflux plus a large mobile irregular mass in the stomach which was believed to be a Gaviscon bezoar. Gaviscon treatment was stopped and the vomiting settled with the addition of Nestargel to the feeds. A repeat barium meal three weeks later showed that the bezoar had resolved.

The barium meal showed persistent vomiting. Treatment was started with Infant Gaviscon in a dose of one sachet with each feed, but the vomiting persisted and two days later a barium meal examination was carried out. A large mobile mass was seen filling much of the body and antrum of the stomach, and the appearances were thought to be those of a bezoar. Gaviscon treatment was stopped and the vomiting settled with nursing in a chair and the addition of solids to the diet. The bezoar was not found by a repeat barium meal three weeks later.

The third case occurred in a male infant who had lactic acidosis of unknown aetiology. He was admitted at 3 months of age with lethargy, vomiting, and refusal to feed. A barium swallow showed noticeable gastro-oesophageal reflux and treatment was started with one half sachet of Infant Gaviscon with each feed. He developed abdominal distension and the vomiting became more severe. Plain abdominal radiograph and a barium meal showed the presence of two very large mobile bezoars (Figure) and Gaviscon treatment was stopped. Barium studies were not repeated because of his poor clinical condition related to the lactic acidosis.
Gaviscon bezoar was first described in 1976 by Hewitt and Benham and similar complications have also been reported in the neonate from the use of aluminium hydroxide gel. If our experience is typical the condition may be much more common than has been suspected, and may occur after very brief periods of treatment. Sinaasappel et al removed much of the mass with a gastroscope and noted improvement in their patient’s condition after this intervention. Our experience in the two patients where repeat barium studies were performed and that reported elsewhere suggest that such active intervention is unnecessary. The only action required for spontaneous resolution of the bezoar is to stop Gaviscon treatment.

References


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Figure Two Gaviscon bezoars present in the stomach.

Pyridoxine dependent seizures

Sir,

In a recent article in this journal Bankier et al described case reports of four infants with a variety of seizures that responded to large intravenous or oral doses of pyridoxine. The high doses used by these investigators are well within the range that has been shown to be neurotoxic in adults and substantially above the lowest dose observed to result in adult neuropathies (H Schaumburg, personal communication).

Bankier et al administered doses of 100 mg pyridoxine intravenously and then maintained the infants on 75 mg orally. Some form of subsequent impaired mental development was noted in all infants. The ages ranged from 1 day (case 5) to 11 months (case 3). In case 5, the birthweight was 3300 g and pyridoxine was given four hours after birth at a dose of 100 mg intravenously. That is 30-3 mg pyridoxine/kg body weight intravenously in an infant who is undergoing massive and rapid development of the central nervous system. The minimum adult oral dosage shown to result in neuropathies is approximately 7 mg/kg body weight or approximately a quarter of the dose given to these infants. Bankier et al reported in case 3 that ‘The results of investigations for weakness and hypotonia were consistent with spinal muscular atrophy . . .’, symptoms which are consistent with those reported for toxicity in adults and in beagle dogs.

I maintain that the recommendation proposed by Bankier et al that ‘A neonate with seizures, even with documented birth asphyxia, should be given 100 mg of intravenous pyridoxine . . .’ is both potentially harmful to the infant and irresponsible unless there is substantial evidence that potentially toxic doses are absolutely necessary for the survival of the infant. Bankier et al did not investigate the efficacy of maintenance doses less than 1 mg/day on the infants, and their treatment may, in fact, have contributed substantially to the impaired mental development noted by the authors.

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Dr Bankier and co-workers comment:

We thank Dr Reynolds for drawing attention to the potential problems of neurotoxicity after chronic ingestion of high doses of pyridoxine. The only report of neurotoxicity in man postdated our paper on pyridoxine dependent seizures. In reply to Dr Reynolds’ comments:

1) We know of no evidence to suggest that a single dose of pyridoxine is harmful even if the child is not pyridoxine dependent. Pyridoxine given as a diagnostic test in a fitting child is likely to be less harmful than the obvious danger of missing a treatable disorder.

2) There was no evidence of deterioration after pyridoxine treatment in any of our patients. The development mental delay reported was present at the time of the first administered dose. In fact the one patient (case 5) treated from day 1 had an ‘intelligence quotient in the low to normal range’ at 5 years of age. None of these children had peripheral neuropathy. Case 3 developed signs consistent with spinomuscular atrophy. This is an inherited condition which had affected her sibling who had never been exposed to pyridoxine.