Value of ultrasound in differentiating causes of persistent vomiting in infants

M D Rollins, M D Shields, R J M Quinn, M A W Wooldridge

Abstract
A prospective study of abdominal ultrasound was undertaken in 100 consecutive infants who presented with a history of persistent vomiting aged 5 to 90 days. Each infant had a ‘test feed’ followed by an ultrasonographic scan of the pylorus at the cotside. On test feeding a palpable tumour was evident in 38 infants. On real time ultrasound using the criteria for diagnosing pyloric stenosis, these 38 infants as well as six others were documented as having pyloric stenosis. In the other 56 patients the vomiting settled in six and a barium examination was performed on the remaining 50. This confirmed gastro-oesophageal reflux in 46, two of whom had an associated hiatus hernia, one with a duodenal malrotation, and three were reported as normal. Ultrasound of the abdomen is an accurate, reliable, and rapid screening method to differentiate the causes of severe vomiting in infancy.

Since the demonstration by Teele and Smith in 1977 of the ultrasonographic appearance of infantile hypertrophic pyloric stenosis on static B scan several papers have been published assessing the accuracy and predictive value of this technique. With the widespread use of high resolution real time equipment this technique has now proved much easier, with greater visualisation and assessment of the pyloric region.

In the past many of the infants exposed to abdominal ultrasound will have presented with the classical history of projectile vomiting, visible gastric peristalsis, and a palpable tumour on test feeding thus indicating the presence of the underlying pyloric stenosis. These patients will not necessarily benefit from a sonographic examination except that it will confirm the diagnosis and document the size of the hypertrophied muscle. The group of patients that will, however, benefit from an ultrasound examination are young infants who present with severe vomiting but with no palpable mass on initial test feeding. The diagnosis may well be infantile hypertrophic pyloric stenosis but could be due to differing conditions associated with vomiting in infancy.

Our aims in this prospective study were firstly to confirm that pyloric muscle dimensions are an accurate indicator of underlying pyloric stenosis with no evidence of false positive or false negative results. Secondly, to find whether by using this ultrasound technique one can rapidly and reliably exclude pyloric stenosis at the cotside before deciding whether radiological methods are required to identify other causes of vomiting in infancy.

Methods
The study was carried out in the Paediatric Unit of Altnagelvin Area Hospital which serves the population of Londonderry and adjacent areas of Northern Ireland. Between 1 December 1986 and 30 June 1988 100 consecutive infants up to the age of 3 months were admitted to the infant unit with a history of severe persistent vomiting. None of these infants showed clinical evidence of associated infection or had a known metabolic disorder which may give rise to such vomiting.

On admission a careful history was taken to establish the type and amount of feeds taken by each infant, the age of onset of vomiting, its frequency, and its relation to feeds. The baby’s birth weight in relation to weight on admission was documented as well as a general assessment of the infant. Of the infants studied, there were

(A) Transverse sonogram. Normal pylorus of newborn. (B) Transverse sonogram. Characteristic ‘target sign’ of infantile hypertrophic pyloric stenosis. The distance between the crosses shows the thickness of the hypoechoic muscle mass. The cross section of the pylorus was also measured (anteroposterior diameter).
Value of ultrasound in differentiating causes of persistent vomiting in infants

60 boys and 40 girls. Their ages ranged from 5 to 90 days (mean (SD) 35 (20) days). The duration of vomiting on presentation to hospital ranged from one to 86 days (mean 12 (16) days). Before performing a test feed a complete blood picture, electrolyte analysis, and arterial blood gas samples were routinely taken.

Each infant had a ‘test feed’ performed at the bedside shortly after admission to assess evidence of projectile vomiting, visible gastric peristalsis, and a palpable tumour. This was immediately followed by ultrasound of the pyloric region using a portable ADR 4000 mechanical sector scanner with a 5-5 MHz head. The patient was laid in a supine position with the transducer head placed on the anterior abdominal wall anterior to the right kidney, medial to the gall bladder, and lateral to the head of the pancreas.

Using this technique the flexibility of the antpyloric muscle was observed and a record made of the pyloric muscle dimensions. Photographs of static scans were taken directly if there was any doubt about borderline measurements and a video recording of antpyloric movements taken if required for further scrutiny (Figure). Two paediatric consultants and two resident paediatric registrars, all of whom had considerable experience with the use of this ultrasound technique, performed both the test feed and the ultrasonic examination. The period of scanning time varied from one to five minutes. If the ultrasonographic criteria were fulfilled no further investigations for diagnosing pyloric stenosis was thought necessary whether the test feed was positive or not. If the pyloric muscle size on ultrasound seemed to be within the normal range— that is, anteroposterior diameter <15 mm or muscle size <4 mm, or both—further radiological studies were performed if the infant continued to vomit feeds.

Results

Of the 100 infants presenting with persistent vomiting between 1 December 1986 and 30 June 1988, 44 were diagnosed by ultrasound criteria as having infantile hypertrophic pyloric stenosis (Table I). Thirty five were boys and nine were girls. Of these, 38 (86%) were ‘test feed’ positive— that is, they showed projectile vomiting, visible gastric peristalsis, and palpable tumour. The clinical assessment thus failed to show pyloric stenosis in six (14%) (Table II). All ‘scan positive infants’ had confirmation of a significantly hypertrophied pylorus at surgery (mean (SD) anteroposterior diameter 15-7 (0-2) mm and mean muscle thickness 5-2 (0-9) mm) with no false positive findings (Table III). The incidence of pyloric stenosis in this study was 5·3 per 1000 live births, which again confirms the high incidence of pyloric stenosis in this area.

Among the 56 remaining infants, vomiting settled after admission in six. A barium upper gastrointestinal series was performed in 50, 44 of which confirmed gastro-oesophageal reflux of varying degree (eight mild, 30 moderate, and six severe). Two infants were shown to have a hiatus hernia with moderate degrees of association reflux, one infant had a duodenal malrotation, and three were reported as normal (Table I). One of the last three was later diagnosed as having primary lactose intolerance. None of the barium examinations performed on infants who were ‘scan negative’ showed any evidence of delay in gastric emptying, and none of these infants subsequently developed pyloric stenosis at a later follow up. A barium examination had been performed on one infant in a peripheral hospital before transfer which suggested delay in gastric emptying consistent with pyloric stenosis. Subsequent ultrasound examination in our unit, however, showed a normal pylorus and the vomiting soon settled. There was no significant correlation between the type, brand, amount, or frequency of feeds taken by infants who developed pyloric stenosis and those who did not. A positive family history of pyloric stenosis was present in only four (9%) infants who developed it.

As a consequence of persistent vomiting pyloric stenosis produces a state of hypochloremic alkalosis. On reviewing our biochemical findings, 42 of 44 infants (96% with pyloric stenosis) had an arterial pH >7-45. Thirty five of 56 infants (62% of the group with no pyloric stenosis), however, also showed a pH >7-45. Appreciable hypochloremia (a chloride concentration of <90 mmol/l) was present in only six (14%) of 44 infants with pyloric stenosis and three (4%) of the others. It seems that an arterial pH <7-45 would not support the diagnosis of pyloric stenosis, although a normal chloride concentration (>90 mmol/l) does not exclude it.

Discussion

This prospective study shows that real time ultrasound is a rapid, reliable, and accurate

---

**Table 1**

<table>
<thead>
<tr>
<th>Ultrasound diagnosis</th>
<th>Final clinical diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>IHPS (44)</td>
<td>IHPS (44)</td>
</tr>
<tr>
<td>Non-IHPS (56)</td>
<td>Gastro-oesophageal reflux (44)</td>
</tr>
<tr>
<td>Vomiting undefined</td>
<td>Hernia hiatus + gastro-oesophageal reflux (8)</td>
</tr>
<tr>
<td>Malrotation</td>
<td>Primary lactose intolerance (1)</td>
</tr>
</tbody>
</table>

IHPS = idiopathic hypertrophic pyloric stenosis.

*Partial thoracic stomach.

**Table 2**

<table>
<thead>
<tr>
<th>Anteroposterior diameter (mm)</th>
<th>Muscle thickness (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test feed negative but ultrasound positive</td>
<td>13-8 (0-7)</td>
</tr>
<tr>
<td>Test feed positive and ultrasound positive</td>
<td>16-0 (0-3)</td>
</tr>
</tbody>
</table>

**Table 3**

<table>
<thead>
<tr>
<th>Anteroposterior diameter (mm)</th>
<th>Muscle thickness (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-IHPS IHPS IHPS IHPS IHPS</td>
<td>Non-IHPS IHPS IHPS</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>11·3 (1·7)</td>
</tr>
<tr>
<td>Range</td>
<td>7·0-14·0</td>
</tr>
</tbody>
</table>

IHPS = idiopathic hypertrophic pyloric stenosis.
method of confirming and diagnosing infantile hypertrophic pyloric stenosis. No other radiological investigation is required to diagnose the condition, thus preventing undue diagnostic delay and exposing infants to unnecessary irradiation and possible risk of aspiration by other contrast methods. The ultrasound scan seems to be more accurate than clinical assessment of the infant (100% compared to 86%) in our study. Ideally, paediatric radiologists should perform ultrasound scans to diagnose pyloric stenosis because they may be able to identify other disorders more readily than doctors with less experience. Such operators, however, are few and are not always attached to a teaching hospital let alone a peripheral one. General paediatricians who come across this common clinical dilemma in differentiating among causes of persistent vomiting in infancy would really need to be at hand to perform the scans at the bedside at any time during the day. This results in minimal disturbance to the infant and the presumptive diagnosis can be relayed to the parents immediately. The use of paediatric ultrasound imaging techniques is operator dependent, hence the more scans one performs the more one becomes aware of normal and abnormal findings. Previous publications have shown an overlap between the ultrasound measurements of the pyloric diameter, muscle thickness, and pyloric length, suggesting that there are grounds for uncertainty in diagnosing borderline cases of suspected pyloric stenosis. Despite the measurement of the pyloric volume being recently advocated,10 to overcome these uncertainties we still find that a rapid assessment of the pyloric diameter and muscle thickness maintains a degree of accuracy of 100% with no false positive or false negative results. In previous studies we have found that the muscle thickness is a better criterion for pyloric stenosis than the overall anteroposterior diameter. As we found in this study, no tumour was evident if the muscle thickness was <4 mm, whereas an anteroposterior diameter of <15 mm could still be associated with a pyloric tumour. Many paediatricians have stated that the diagnosis of pyloric stenosis should be entirely clinical by detecting a palpable tumour during the test feed. Certainly, if infants are examined at a late stage it should be possible to palpate the tumour in all cases. If, however, the infant is examined at an earlier stage, when the tumour is developing, the diagnosis can be missed. On reviewing the six infants in our study with pyloric stenosis who were test feed negative but ultrasound positive we found that their pyloric dimensions were significantly smaller than those of infants who were both test feed positive and ultrasound positive (Table II). The difference between the mean (SD) diameter in the test feed negative but ultrasound positive group (13.8 (0.7) mm) compared to that in the normal infants (11.3 (1.7) mm) was slight. But the difference in mean muscle thickness in this group (4.4 (0.4) mm v 2.2 (0.5) mm) was highly significant (p<0.01). This again confirms that the anteroposterior diameter is a questionable measurement for confirming infantile hypertrophic pyloric stenosis, particularly in a small or early tumour. We therefore will make the diagnosis entirely on a muscle thickness of >4.0 mm:an anteroposterior diameter of >15 mm.

We again emphasise that no moderate or large pyloric tumours were 'missed' by clinical interpretation of the test feed; however, smaller degrees of muscle hypertrophy indicating early pyloric stenosis would have been difficult to detect.

The other major cause of persistent vomiting in infancy, as shown in this study, was gastro-oesophageal reflux. This can be diagnosed using abdominal ultrasound,2 and indeed we were able to identify degrees of reflux during the scanning period. It has even been suggested that a higher rate of detection of reflux can be obtained by this technique compared with the barium swallow examination.3 Showing reflux by ultrasound in inexperienced hands, however, can be time consuming, and even with radiology it is difficult to interpret as reflux can be seen in some normal infants.10

For our study the barium swallow was thought to be a more definitive examination to investigate causes of vomiting in the group with no pyloric stenosis. This test not only identifies the presence of reflux but also shows or excludes rarer abnormalities such as hiatus hernia, stricture, duodenal obstruction, and intestinal malrotation. We emphasise, however, that the barium test should only be reserved for infants who continue to vomit despite a negative ultrasound of the pyloric region. None of the infants who had negative ultrasound scans and who subsequently stopped vomiting during their hospital stay were found to have appreciable disease on follow up.

Real time ultrasound is now routinely performed at the bedside after the test feed by our resident paediatric staff on infants who present with persistent vomiting. We have shown that it is an accurate diagnostic tool providing us with an efficient means to evaluate infants who present to hospital with this common diagnostic problem.

Value of ultrasound in differentiating causes of persistent vomiting in infants.

M D Rollins, M D Shields, R J Quinn and M A Wooldridge

Gut 1991 32: 612-614
doi: 10.1136/gut.32.6.612

Updated information and services can be found at:
http://gut.bmj.com/content/32/6/612

Email alerting service

These include:
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Topic Collections
Articles on similar topics can be found in the following collections
- Stomach and duodenum (1689)
- Gastro-oesophageal reflux (351)

Notes