Intravenous atropine treatment in infantile hypertrophic pyloric stenosis

H Kawahara, K Imura, M Nishikawa, M Yagi, A Kubota

Aims: To assess the efficacy of a new regimen of intravenous atropine treatment for infantile hypertrophic pyloric stenosis (IHPS) with special reference to regression of pyloric hypertrophy.

Methods: Atropine was given intravenously at a dose of 0.01 mg/kg six times a day before feeding in 19 patients with IHPS diagnosed from radiographic and ultrasonographic findings. When vomiting ceased and the infants were able to ingest 150 ml/kg/day formula after stepwise increases in feeding volume, they were given 0.02 mg/kg atropine six times a day orally and the dose was decreased stepwise.

Results: Of the 19 infants, 17 (89%) ceased projectile vomiting after treatment with intravenous (median seven days) and subsequent oral (median 44 days) atropine administration. The remaining two infants required surgery. No significant complications were encountered. Ultrasonography showed a significant (p < 0.05) decrease in pyloric muscle thickness, but no significant shortening of the pyloric canal after completion of the atropine treatment. The patients exhibited failure to thrive at presentation, but were thriving at 6 months of age (p < 0.01).

Conclusions: This atropine therapy resulted in satisfactory clinical recovery. Pyloric muscle thickness was significantly reduced.

F redet-Ramstedt pyloromyotomy has been regarded as the optimal treatment for infantile hypertrophic pyloric stenosis (IHPS), although surgical complications have been reported. Medical treatment with oral antispasmodics such as atropine sulphate or methyl scopolamine nitrate have virtually been abandoned since the mid-1960s. Nagita et al. have reported a high success rate using intravenous atropine treatment. In their regimen, the intravenous dose of atropine was increased stepwise until vomiting was controlled, so the final dose administered varied between patients. We devised a new regimen with a fixed dose of atropine. This study was designed to assess the clinical outcome of our regimen and to look for evidence of regression of pyloric hypertrophy using ultrasonography.

PATIENTS AND METHODS

Patients

Ethical approval was obtained from the departmental committee, and informed consent was obtained from the patients’ guardians. The study population consisted of 19 consecutive infants (male/female 16:3) with IHPS, who were in Osaka Medical Centre and Research Institute for Maternal and Child Health in the period November 1996 to March 1998. All fulfilled the following diagnostic criteria for IHPS:

(a) repeated projectile vomiting more than twice a day;
(b) gastric outlet obstruction and characteristic narrow, long pyloric canal on upper gastrointestinal series;
(c) pyloric canal length ≥ 15 mm and pyloric muscle thickening ≥ 4 mm on ultrasonography.

Table 1 shows the patient characteristics. The median (range) age was 40 (22–85) days at admission. Their median (range) weights at birth and admission were 3086 (2480–4430) g and 3970 (3200–5410) g respectively.
Ultrasonographic evaluation of the pylorus
Ultrasonographic evaluations were conducted at presentation, at three weeks and six months after completion of oral atropine administration, and at 1 year of age. Ultrasonography was performed by the same paediatric radiologist (MN) using a YOKOGAWA Medical RT4600 and 7.5 MHz linear probe. The patients were fasted for more than three hours before ultrasonographic examination and were not sedated. The examination was performed on several planes. The thickness of the pyloric muscle and the length of the pyloric canal were measured on transverse views, which showed the pylorus along its long axis. Transpyloric flow of gastric contents was also examined. All patients were followed up until 2 years of age.

Statistical analysis
All data are presented as median (range). Tests for statistically significant differences in weight SDSs and ultrasonographic measurements were analysed using the Kruskal-Wallis test. Categorical variables were compared using the Tukey test.

RESULTS
At the time of presentation, 12 patients had previously received infusions at other hospitals, and dehydration and/or electrolyte imbalance were not noted. Of the 19 patients, 17 became free from projectile vomiting during atropine treatment. The remaining two required surgery after 9 and 18 days respectively. Total hospital stay was 13 (6–20) days.

Of the 17 patients, 13 experienced little vomiting three weeks after completion of oral atropine treatment (p < 0.01) (fig 1). There was a significant (p < 0.01) increase at 6 months of age (−0.3 (−1.7– +1.8), but not at 3 months (−0.6 (−3.1– 0)), compared with that at presentation (−1.4 (−4.1– −0.2)). There was no further significant change at 1 year (+0.2 (−1.7– +2.1)) and 2 years of age (−0.4 (−1.2– +2.4)).

Ultrasonography showed significant (p < 0.01) changes during atropine treatment. Figure 2 shows serial changes in pyloric muscle thickness and pyloric canal length. Pyloric muscle thickness decreased significantly (p < 0.05) from 5 (4–6) mm at presentation to 3 (2–5) mm three weeks after completion of oral atropine. Pyloric canal length was 19 (15–25) mm at presentation and 15 (12–20) mm three weeks after completion of oral atropine (not significant). Transpyloric flow of intragastric fluid was not observed by ultrasound in any patient before treatment, but was detected in all three weeks after completion of oral atropine. Pyloric muscle thickness was 2 (2–4) mm six months after completion of oral atropine and 2 (2–3) mm at 1 year of age, both of which were significantly (p < 0.01) less than that at presentation. Pyloric canal length

Table 1 Characteristics of subjects

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Table 1: Characteristics of subjects

Ultrasonographic measurements were made before and three weeks after atropine administration. IV, Intravenous atropine administration.

Figure 1 Serial changes in weight standard deviation scores (SDS) in patients successfully treated with atropine. Box, interquartile range; horizontal bar, median; whiskers, 10th and 90th centile. *p < 0.01, **p < 0.05.
problems. The adverse events in the series of Nagita et al. were maintained in a safe range and there were no serious clinical complications. We therefore used this as a fixed dose before feeds six to ten days at the guardians' request in two patients, they did not improve and required surgery. In contrast, projectile vomiting was controlled within 10 days in 17 patients.

Significant complications were not encountered. One patient had a urinary tract infection and one a transient slight increase in serum aspartate aminotransferase and alanine aminotransferase (< 60 IU/l) during intravenous atropine administration. Facial flushing was not reported during treatment. The resting heart rate remained less than 160 beats/min.

DISCUSSION
Medical treatment with atropine has been reappraised as an option for IHPS treatment, using a step up dosage technique with intravenous atropine administration and was associated with a successful short term outcome. The clusters of tonic and phasic pyloric contractions characteristic of IHPS recently reported are transiently abolished by an intravenous atropine injection of 0.01 mg/kg. We thought that this dose of atropine would improve transpyloric flow by inhibiting the contractions. We therefore used this as a fixed dose before feeds six to ten days a time. This was in contrast with the regimen of Nagita et al., in which the dose of atropine was increased until projectile vomiting was controlled. In that study, the dose of atropine actually given was 0.07 (0.04–0.11) mg/kg/day, and 12 of 22 patients received a dose greater than that used in our study. The success rate in our regimen (89%) was similar to that at presentation. The duration of intravenous atropine was 14 (11–19) mm six months after completion of oral atropine and 12 (9–19) mm at 1 year of age, both of which were significantly (p < 0.01) less than that at presentation.

The present study indicated that pyloric muscle thickness was significantly less on completion of oral atropine treatment, and preceded normalisation of pyloric canal length. The patient had a urinary tract infection and one a transient slight increase in serum aspartate aminotransferase and alanine aminotransferase (< 60 IU/l) during intravenous atropine administration. Facial flushing was not reported during treatment. The present study indicated that pyloric muscle thickness was significantly less on completion of oral atropine treatment, and preceded normalisation of pyloric canal length.

The value of intravenous atropine as a treatment for IHPS remains controversial. It has not gained wide acceptance mainly because it requires a prolonged hospital stay and treatment at home. Although pyloromyotomy has been regarded as the optimum treatment of IHPS, the actual and potential consequences of surgery cannot be ignored. The patient has an abdominal scar, and there is a small long term risk of complications due to adhesion. Expert paediatric surgery is not universally available. A randomised controlled trial comparing the outcomes and cost effectiveness of intravenous atropine versus surgery is required.

Authors' affiliations
H Kawahara, K Imura, M Nishikawa, M Yagi, A Kubota, Departments of Paediatric Surgery and Radiology, Osaka Medical Centre and Research Institute for Maternal and Child Health, Osaka, Japan

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