
Selected Summaries

Medical Management of Hypertrophic Pyloric Stenosis

[Nagita A, Yagamuchi}, Amimoto K, Yoden A, Takeshi Y, Mino M. Management and Ultrasonographic Appearance of Infantile Hypertrophic Pyloric Stenosis with Intravenous Atropine Sulphate. *J Pediatr Gastroenterol Nutr* 1996; 23:172-177].

Fredet Ramstedt pyloromyotomy first performed in 1911 has long been the standard surgical treatment for infantile hypertrophic pyloric stenosis (HPS). It is the treatment of choice with excellent results, so much so that conservative medical management is invariably neither considered nor offered. A few reports in early sixties pondered over the issue of medical management (1,2) of HPS utilizing methyl scopolamine, atropine and eumydrin but fell into disrepute because of longer duration of therapy, slower resolution of symptoms, higher mortality, unsuccessful oral medication due to continuing vomiting and recurrence of vomiting after initial success in favour of a seemingly simple surgery with negligible mortality but significant complications.

The authors of the present study used oral atropine in some of their patients pre-operatively and found that vomitings resolved in these patients and they could be fed small enteral feeds but there was no change in the hypertrophied pyloric muscle ultrasonographically. This led them to hypothesize that atropine is probably an effective drug for HPS but the gastric outlet obstruction prevents it from reaching the small intestine, its site of absorption. Hence atropine was administered intravenously

in 23 babies with HPS at a dose of 0.04/mg/kg/day divided three hourly (eight doses, 10 min prior to feed administered over a period of 3 min) increasing by 0.01/mg/kg/day until vomitings ceased. Atropine was given orally after the cessation of vomitings at twice the effective intravenous dose and continued for two weeks. Twenty one out of 23 babies did not require surgery, had consistent weight gain, no vomitings, but there was no change in pyloric muscle caliber on ultrasonologic evaluation immediately post treatment. Ultrasonographic normalisation of pyloric muscle caliber was demonstrated by 4-12 months of age. Out of the remaining two babies, one patient's parents opted for surgery and in the other one, the oral dose of atropine was not doubled inadvertently leading to repeated vomitings at the time of discharge which improved on readjustment of the dose but the parents opted for surgery.

Comment

Pyloromyotomy has indeed been the treatment of choice for HPS with excellent results but there is a definite risk of complications ranging from 25-40% in various series. These include infection, wound dehiscence, wound spesis, vomiting, serious hemorrhages and duodenal perforation (3,4). In view of these complications a conservative, medical approach would be welcome. Before even considering having controlled trials with a larger number of subjects (allowing cross over, if medical management fails) we need to understand the pathophysiology of this entity and the role of pharmacologic therapy. The obvious questions that need to be answered in this context are "Does acetyl-choline play a ma-

major role in the pathophysiology of HPS? Is the pyloric muscle hypertrophy worsened by the spasm that occurs in HPS? Or is there an abnormal distribution of nerve terminals in the muscle layer in the pylorus of infants with HPS?"

Well from the results of this study it appears that probably acetyl-choline plays a significant role in the pathophysiology of HPS because atropine which is a cholinergic blocking agent with potent antimuscarinic activity, and which decreases peristaltic contractions by relaxing smooth muscles was able to completely revert the changes characteristic of HPS. However, there was no dramatic change in the caliber of the pyloric muscle which took 4-12 months to normalise. Atropine has traditionally been used as an antispasmodic agent for gastrointestinal disorders hence the principal effect of atropine for HPS can be considered as the control of spasm. This is further highlighted by the fact that with the 3 hourly dosage schedule (prefeed) of intravenous atropine, the smooth muscle at the pylorus relaxed, feeds passed into the duodenum and the vomitings ceased but the pyloric hypertrophy remained unchanged sonographically in all infants whose vomitings were controlled on atropine. The pyloric muscle eventually regressed over a period of 4-12 month at which time the babies were not on atropine suggesting that muscular spasm rather than hypertrophied pyloric muscle accounts for the symptoms of HPS and that this spasm worsens the hypertrophy. Once the spasm is controlled by atropine therapy the muscle caliber starts to regress.

There have been reports showing that the density of nerve terminals, neurofilaments and nerve supporting cells (5,6) was reduced in the muscle layer in the pylorus in patients with HPS. Whether, they

have any bearing on the pathophysiology is not clear with the existing knowledge on the subject.

This study shows excellent results with the use of atropine but the number of patients enrolled were few pending, firm evidence based recommendations, it may be worthwhile attempting a trial of medical therapy in babies with HPS who could subsequently be taken up for surgery if symptoms or hemodynamic disturbance did not improve or worsened.

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Congenital Diaphragmatic Hernia

[de la Hunt MN, Madden N, Scott JES, et al. *Is delayed surgery really better for congenital diaphragmatic hernia?: A prospective randomized clinical trial. J Pediatr Surg* 1996; 31: 1554-1556].

This is one of the few prospective randomized trials which have sought to answer the question of timing of surgery in congenital diaphragmatic hernia (CDH). This study on 54 neonates had two groups; (i) One that underwent surgery early (within 11 h of birth, mean 6 h; n=26), and (ii) the other (n=28) in which surgery was delayed (25-240 h, mean 48 h). The two groups were similar with respect to age at diagnosis, birth weight, gestational age and pre-operative blood gas values. The general supportive and respiratory care including pulmonary vasodilator use was identical in the two groups. Twenty eight babies (51.8%) survived, 46% in the early surgery group and 57% in the delayed surgery group. The difference was not statistically significant ($p=0.42$). When the characteristics of the survivors and non-survivors in either groups were compared, it was observed that the early pre-operative blood gas values were better in the survivors. Large diaphragmatic defects were associated with a higher mortality. Antenatal diagnosis was not a useful prognostic factor. This study highlights the fact that the issue of optimum time for repair of CDH remains unsettled.

[Lewis DA, Rieckert C, Bowerman R, Hirschl RB. *Prenatal ultrasonography frequently fails to diagnose congenital diaphragmatic hernia. J Pediatr Surg* 1997; 32: 352-356.]

This study evaluates 136 cases of CDH which became symptomatic within 24 h of birth. Over a 10 year study period, the use of antenatal ultrasonography for the diag-

nosis of CDH increased from 33% to 100%. The false negative rate, however, remained the same (55%). Ninety two babies (67%) had pre-natal ultrasonography. A correct diagnosis was made in 52% cases. Missed diagnosis in 44 cases was because of technical reasons, or failure to follow established guidelines for the diagnosis of CDH, or missed findings. The paper, however adds that there was no significant difference in the survival rates between those diagnosed antenatally and those which weren't.

[Wilson JM, Lund DP, Lillehei CW, Vacanti JP. *Congenital diaphragmatic hernia-a tale of two cities: The Boston Experience. J Pediatr Surg* 1997; 32: 401-405.]

Cases of CDH treated between 1981 and 1994 were studied. A total of 196 babies presented before 12 h of age and were the subjects for this analysis. The treatment strategy in respect of respiratory support was conventional ventilation in all babies with ECMO rescue in the refractory cases. The overall survival was 53%. Ninety eight babies (50%) required ECMO of which only 44% survived. Associated malformations were seen in 39% cases and this was associated with lower survival rates. Antenatal diagnosis and the side of the diaphragmatic defect had no impact on the outcome. The Apgar score and the pre-operative best post-ductal pO_2 values were important predictors of final outcome.

[Azarow K, Messineo A, Pearl R, Filler R, Barker G, Bohn D. *Congenital diaphragmatic hernia-a tale of two cities: The Toronto Experience. J Pediatr Surg* 1997; 32: 395-400].

In this study 223 babies with CDH diagnosed within 12 h were analyzed. The respiratory support strategy was conventional ventilation with conversion to high frequency oscillation ventilation for refractory hypoxemia or hypercapnia. Since 1985, sur-

gery was deferred until stabilization was achieved. This resulted in a shift in the mortality from postoperative period to the preoperative period with no change in total survival. The overall survival rate in this study was 54.7%. HFOV did not significantly alter overall survival rates. Pulmonary hypoplasia was the principal cause of death. This study confirms the Boston experience that Apgar scores and initial postductal pO₂ values are important to predict outcomes.

Comment

These four studies represent the experience of four major centers in the world treating babies with CDH. The results and conclusions from these studies are important and should determine the future strategies in the management of CDH. While summarizing these studies, it can be clearly said that the treatment of CDH presenting early is still far from satisfactory. There are still a large number of grey areas, but there are also some indisputable conclusions.

- (/) The several treatment strategies in those babies with a poor prognosis include conventional ventilation with permissive hypercapnia, ECMO, high frequency oscillation ventilation, inhaled nitric oxide, liquid ventilation and pre-natal repair. There exists no clear evidence that any of these modalities offers a significant survival advantage over conventional ventilation.
- (it) The issue of optimum timing of surgery remains unsettled. The survival rate is the same whether the babies are operated early or the surgery is delayed. This emphasizes the fact that in the Indian context, where the available ventilatory support is at best very basic, the best chance for the baby is if he is operated early.

(iii) Apgar scores and the pre-operative best post-ductal pO₂ values are important predictors of outcome.

(iv) Antenatal diagnosis and the side of the defect have no impact on the outcome. However, with an antenatal diagnosis, it is safer to transport an affected fetus *in utero* to a surgical facility than to transport a sick neonate.

(v) Pulmonary hypoplasia and ventilation induced barotrauma are the most important causes of death.

EMPYEMA THORACIS

[Chan W, Keyser-Gauvin E, Davis GM, Nguyen LT, Laberge f-M. Empyema thoracis in children: a 26-year review of the Montreal Children's Hospital Experience, *J Pediatr Surg* 1997; 32: 870-872].

This is a study on 47 patients spanning a 26 year period (1968-94). The mean patient age was 5.6 yr (M:F 1:1). The presenting features were cough, chest pain, dyspnea and fever. The patients were classified as acute (pleural fluid clear or slightly cloudy, sterile with increased WBC count); or fibrinopurulent (fluid thick and opaque, culture positive). There were 7 acute empyemas. All responded to antibiotics with or without tube thoracostomy. Of the 39 fibrinopurulent empyemas 32 responded to antibiotics and tube drainage; 25 of which responded in spite of incomplete drainage of loculations. Only 7 patients required decortication for a very thick peel or recurrent empyemas. The overall complication rate was 42% including pneumothorax, abscess, hemoptysis and scoliosis. The scoliosis recovered in all patients.

Comment

There is a paucity of literature on the treatment of acute empyemas in children.

The condition is seen commonly in India and in the absence of any set protocol for their treatment these unfortunate babies are often neglected or improperly treated. Centers in India that see this condition frequently would do us all a favor by writing their experiences in the management of this

condition particularly if they have a well determined treatment protocol.

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