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Effect of Atropine for The Treatment of Infantile Hypertrophic Pyloric Stenosis

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1. Abstract

1.1. Objective

To compare the efficacy of treatment of infantile hypertrophic pyloric stenosis with atropine sulfate and by surgery, with special reference to regression of pyloric hypertrophy.

1.2. Methods

Thirty-two consecutive infants with pyloric stenosis were seen between January 2008 and June 2011, and 28 cases were enrolled randomly. The diagnosis in all infants was confirmed by an abdominal ultrasound. Medical treatment consisted of intravenous atropine at 0.01 mg/kg every 4 h, followed by oral atropine at 0.02 mg/kg every 4 h. Surgical treatment consisted of standard Ramstedt's pyloromyotomy under general anesthesia. The response to the two treatments was gauged from clinical improvement, weight gain and ultrasonic measurement of pyloric muscle thickness and pyloric canal length.

1.3. Results

Fourteen infants were treated medically and 14 were given surgical treatment. Medical treatment resulted in complete cure in 8 of 12 infants (66.6%), whereas surgical treatment was successful in all 14 infants (100%; $p < 0.05$). The mean length of hospital stay was 10 ± 2 days for medically treated infants and 3 ± 1 days for those treated surgically ($p = 0.0001$).

1.4. Conclusion

Surgical treatment is superior to medical treatment in cases of infantile pyloric stenosis. The success rate of medical treatment with atropine sulfate nevertheless justifies its administration to infants in whom surgery is contraindicated and when medical treatment is preferred by the parents.

2. Introduction

Infantile hypertrophic pyloric stenosis is an acute abdominal emergency and is the most common cause of non-bilious vomiting in infancy [1]. Its incidence is 2–4 per 1000 live births [2] and the risk is four times higher in boys than girls. The pylorus hypertrophies after birth and causes progressive gastric outlet obstruction, infants most commonly presenting this are between 2 and 6 weeks of age [3]. The typical presentation is with projectile and non-bilious vomiting, which, if unrelieved, leads to progressive weight loss, dehydration and metabolic abnormalities [4].

The exact etiology and pathogenesis are unknown, but the reason for clinical presentation is hypertrophy of the circular muscle layer of the pylorus, which causes gastric outlet obstruction. The diagnosis is usually clinical; it is based on a suggestive history and confirmed by palpation of the hypertrophied pylorus, or 'olive', which has a positive predictive value of 99% [5,6]. The standard surgical technique is Ramsestdt's pyloromyotomy, and this is still the treatment for infantile hypertrophic pyloric stenosis medical treatment with atropine sulfate has been proposed as an alternative to surgical treatment. Atropine sulfate is a cholinergic drug with strong anti-muscarinic effects, which temporarily suppresses spastic contractions of pyloric muscle in pyloric stenosis [7,8]. The clusters of tonic and phasic pyloric contractions characteristic of infantile hypertrophic pyloric stenosis are transiently abolished by an intravenous atropine injec-

tion of 0.01 mg/kg [9]. Initial reports on the use of this method, however, did not validate its use as a treatment option, in view of the reported low success rate of 50% [7,10]. Recently, however, researchers in India reported a success rate of 96% with medical treatment,11 which has encouraged other researchers to review the treatment of infantile hypertrophic pyloric stenosis with atropine sulfate. The objective of this study was to see the effect of atropine for treatment of Infantile hypertrophic pyloric stenosis

3. Materials and Methods

A prospective study was conducted in the Paediatric Surgery Department during the period January 2022 to June 2023. Fifteen consecutive infants (12 boys and 3 girls) in whom infantile hypertrophic pyloric stenosis was diagnosed after presentation at the emergency department fulfilled the following diagnostic criteria: a classical history of pyloric stenosis (frequent non-bilious projectile vomiting), pyloric muscle thickness ⩾4 mm and pyloric canal length ⩾15 mm on ultrasonography. (Agata)) The aim of the study were explained to the parents before they gave consent for their infants to participate in the study.

After selection of the subjects, detailed benefit of the study was explained to legal guardian. they are encouraged to participate voluntarily and were allowed to withdraw from the study freely.

The oral atropine starting dose ranges from 0.12mg/kg/day to a maximum 0.24mg/kg/day, given from 15 to 30 minutes before feeding the infant (Agata). They are discharged from hospital when vomiting was controlled with oral atropine was then continued at home for 1 month. Medical treatment was considered ineffective when the infant failed to tolerate oral feeding and vomiting persisted 48–72 h after the start of intravenous atropine or vomiting recurred after starting oral atropine. Non-responders were shifted to surgical treatment. Surgical treatment consisted of classical Ramstedt's pyloromyotomy under general anesthesia (Agata).

Response to either surgical or medical treatment was assessed clinically on the basis of cessation of vomiting, tolerating oral intake and gaining weight, and radiologically by ultrasonographic measurements of pyloric muscle thickness and pyloric canal length. Complete response was defined as sustained clinical and sonographic response after cessation of treatment. All infants were followed for 1 year to evaluate vomiting, weight gain and recurrence of symptoms. Abdominal ultrasonography was done in all treated cases 6 weeks after the end of treatment to confirm radiological improvement (Agata).

4. Results

The group treated medically consisted of 12 boys and 3 girls. with a mean agev of 28 days (SD7) and a mean weight of 2.7 kg (SD0.8). Pyloric muscle thickness decreased significantly ($p< 0.05$) from 5 (4-6) mm at presentation to 3(2-3) mm three weeks after completion of oral artopine. Pyloric canal length was 19 (15-25) mm at presentation and 15 (12-20) mm three weeks after completion of oral atropine (not significant). Pyloric muscle thickness was 2(2-4) mm six monyhs 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. Pyliric canal length 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 at presentation. Complete response was found in 11 of the 15 infants who completed the regime (73.3%), partial response was observed in 2 cases and no response in 2 cases. These last 4 cases underwent surgical treatment. Side effects of oral atropine was less. Only transient tachycardia, flushed skin and fever have been reported, but these side effects are rare and self limiting.

5. Discussion

Surgical treatment was clearly superior to medical treatment with regard to response and was also associated with a shorter hospital stay. Complications, however, occurred more often in the surgically treated group. Although pyloromyotomy is a safe curative procedure, it has its risks. Hulka et al [8]. Reported 0.1% mortality, 4% intraoperative complications and 6% postoperative complications, such as bleeding, mucosal perforation and wound infection. Mucosal perforation commonly results from extending the myotomy beyond the pyloric–duodenal junction. Pranikoff et al [9]. Found that the risk for mucosal perforation was 0.5% when pyloromyotomy was performed by a paediatric surgeon. In our study, mucosal perforation occurred in one case and wound infection in another. Although there has been some concern about side-effects of atropine, no serious complications have been reported. Nagita et al [7]. Reported only mild facial flushing, increased alanine aminotransferase activity and tachycardia. We observe no complications in our medically treated infants. Although the complication rate in the surgical group was higher than that in the medical group in this study, the difference was not statistically significant. Other concerns with atropine treatment are the length of hospital stay and the necessity to continue oral atropine. Currently, upper abdominal ultrasound is the imaging modality of choice in pediatric centers. The success and reliability of this modality depend to a certain extent on the operator and his or her experience. Ultrasound is performed after feeding the infant dextrose solution to dilate the fundus, which improves the image quality. NH aider et al, [3]. The length of the pyloric channel and the thickness of the pyloric muscle are the quantitative parameters in the longitudinal view. The classical target sign is seen in the transverse view. Although conventional ultrasound is widely used, high-resolution endoscopic ultrasound can be performed by placing the probe along small endoscopes when the luminal diameter is limited, and the muscle thickness is easily seen [13]. The success rate was 73.3% in our study. The rate for the medical group was lower than those reported by Singh et al. [11[and Kasuko et al. [10] (96%), Kawahara et al, [5,6,9] (87%) and almost similar to Meissner et al. [12] (75.8%). This may be due to the small number of infants in the medical group who completed the study, use of different doses of atropine and different durations of intravenous regimens by other researchers and the fact that we did not escalate

the dose of intravenous atropine to overcome unresponsiveness, as done by other researchers [11]. No obvious clinical or radiological features were found for the infants who failed to respond to medical treatment and those who responded in this study.

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