

Case Report

Treating Pyloric Stenosis Medically in a Resource Poor Setting

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Abstract

Infantile hypertrophic pyloric stenosis (IHPS) is one of the most common gastrointestinal disorders requiring abdominal surgery in infancy. Whilst the operation is relatively routine and complications are low in developed countries, in resource poor settings, the condition can be fatal due to lack of paediatric surgeons and neonatal anaesthetists in most hospitals. However, current WHO guidance advises only to refer urgently for paediatric surgery if available.

In this case report we present a case of pyloric stenosis in a medium sized (district, 150 beds) hospital in Tanzania. The case was managed medically given the lack of paediatric surgery possibilities and availability of transfer. The patient was successfully managed with Atropine. We propose medical management of pyloric stenosis could be an effective solution for the condition in resource poor countries.

ABBREVIATIONS

PS: Pyloric Stenosis; **IHPS:** Infantile Hypertrophic Pyloric Stenosis; **WHO:** World Health Organisation; **IV:** Intravenous

INTRODUCTION

Infantile hypertrophic pyloric stenosis (IHPS) is one of the most common gastrointestinal disorders requiring abdominal surgery in infancy [1], with an incidence of 1.06 to 4.33 per 1000 births [2-4]. Infants typically present in the first 4-6 weeks of life with frequent projectile non-bilious vomiting and dehydration, with clinical diagnosis being supported by ultrasound confirmation.

The aetiology of IHPS is still unclear. Standing on some prospective studies the pyloric muscle is not initially hypertrophied but appears to hypertrophy after birth, leading to gastric outlet obstruction [5]. Some authors suggest that there is a localised deficiency of pyloric innervation [6-8], others reporting the normalisation of pyloric muscle thickness after pyloromyotomy or atropine administration, suggest that IHPS is a self-limiting and reversible disorder of muscarinic receptors in pyloric muscle [9]. Kawahara et al found that the frequency of contractions in the pylorus was suppressed temporarily for 20 to 30 minutes by intravenous atropine [10]. These contractions also were reduced significantly soon after pyloromyotomy, suggesting that the role of pyloromyotomy is not so much the creation of

a wider pyloric lumen but the inhibition of circumferential contractions of the pyloric muscle by incising the muscle fibre of the pylorus. Thus, it is supposed that muscular spasm rather than hypertrophic pyloric muscle accounts for the symptoms typical of IHPS and that prolonged spasm induces hypertrophy of the pyloric muscle [11].

Higher incidence in white male infants [12] and association with genetic syndromes suggest a genetic susceptibility; several genetic loci seem to be involved and contribute to the disease however, a solitary causative gene has not yet been identified [12]. The Xq chromosome seems to be involved and it might explain the male predominance of the disease [13]. Other chromosomes harbour interesting candidate genes including glucagon-like peptide 2, nitric oxidase synthase 1, motilin and neuropeptide Y, which may help regulate smooth muscle tone and gastric motility [13]. Moreover the association with macrolide antibiotics has been strongly linked to IHPS risk suggesting that chemical exposures (to mother or infant) can lead to hypertrophy of the pylorus [14].

The diagnosis is usually clinical; it is based on a suggestive history of recurrent non bilious projectile vomiting accompanied by strong appetite and confirmed by palpation of the hypertrophied pylorus, or "olive", which has a positive predictive value of 99% [15] and is felt as a firm mass at the lateral edge of the rectus abdominis muscle in the right upper quadrant of

the abdomen. A recent study highlighted the significance also of gastric peristalsis that was visible in 100% patients [16]. Ultrasonography is now the modality of choice for diagnosis at most institutions, because it is safe and allows repeated examination. Upper gastrointestinal series show a gastric outlet obstruction and characteristic narrow, long pyloric canal, with the string sign (narrow pyloric channel) and shoulder sign (caused by bulging of pyloric muscle into the barium-filled antrum at the juncture of the stomach wall and hypertrophic pyloric muscle). The Haller-Cohen ultrasound accepted criteria [17] are listed as: pyloric muscle thickness >4 mm, pyloric diameter >15 mm and pyloric canal length >18 mm.

Treatment commences with the stabilisation and correction of possible metabolic complications of intractable vomiting (electrolyte imbalances and dehydration) followed by treatment of the stenosis itself. In 1912 the Fredet-Ramstedt pyloromyotomy was first described, but until the late 1960's infantile hypertrophic pyloric stenosis was commonly treated medically, using oral antispasmodics, such as atropine sulphate or methyl scopolamine nitrate [18,19], while the surgery was reserved for complicated or refractory cases.

However, after the late 1960's the pyloromyotomy became the gold standard treatment of infantile hypertrophic pyloric stenosis and the medical treatment was almost abandoned, thanks to the high success rate, the prompt resolution of symptoms obtained from pyloromyotomy and its low associated morbidity, yielding excellent results and fast recovery of patients. Indeed, in Western countries, the Fredet-Ramstedt operative technique is easy for a skilled paediatric surgeon to perform, with both classical and laparoscopic approaches, and postoperatively the child can usually be fed within 3 to 6 hours and discharged within 1-2 days of surgery.

Atropine is a cholinergic blocking agent with potent anti-muscarinic activity that decreases peristaltic contractions and temporarily suppress spastic contractions of pyloric muscle, thus atropine controls the spasms, which seem to be the cause of the condition.

Because of the uncertainty of the results and the prolonged treatment and hospitalisation, nowadays intravenous atropine therapy is not widely accepted in Western countries [20,21,22]. Although, in some settings, medical treatment with intravenous atropine has recently been reappraised as an option for IHPS and trials of medical management with intravenous atropine sulphate and oral atropine have shown encouraging results.

Different treatment regimens have been studied [9,10,21-30]. Since Kawahara et al found that clusters of tonic and phasic pyloric contractions characteristics of IHPS were transiently abolished by the intravenous injection of 0.01 mg/Kg of atropine [31], this dose has been frequently used in following studies. Most of the studies start with intravenous atropine, shifting to oral atropine when the patient is able to tolerate the full volume of the meal without vomiting more than twice a day (or without vomiting at all) and the patient shows a steady weight gain. In some studies only oral atropine was investigated.

The intravenous atropine starting dose ranges from a minimum of 0,04 mg/Kg/day to a maximum of 0,1 mg/Kg/day,

administered from 3 to 10 minutes before the meal; while the oral dose ranges from 0,12 mg/Kg/day to a maximum of 0,24 mg/Kg/day, given from 15 to 30 minutes before feeding the infant. The oral dose is much higher than the intravenous dose assuming that the atropine effective oral dose is double of intravenous dose; moreover atropine sulphate is absorbed from the intestine and therefore atropine given orally could be diluted with gastric contents and delayed emptying might not allow desirable amount of atropine to reach intestine in desirable time [26].

Tosamaganga Hospital is situated in the Southern Highlands district of Iringa in Tanzania. It is a medium sized district hospital with 150 beds. It has running water and electricity and an operating theatre with surgeons able to complete routine operations such as Caesarian section, appendicectomy etc in adults and older children.

CASE PRESENTATION

A 4 week old male baby presented with a history of projectile vomiting to our service at Tosamaganga District Hospital, Iringa-Tanzania. He was born by spontaneous vaginal delivery, with a birth weight of 3.5kg, with no documented complications.

He was vomiting milk, there was no fever and despite vomiting the patient was then eager to breast feed. He had previous history of passing normal yellow paste stool. His body weight on admission was 3.05kg, he was afebrile, appeared dehydrated and his abdomen was distended but soft, visible peristaltic movements were observed and there was presence of smooth 1-2cm mass in the right hypochondrium. Full blood picture was unremarkable. Abdominal X-ray revealed non specific findings with an enlarged stomach. Treatment was commenced with IV fluids and IV antibiotics (gentamicin & ampicillin), however the condition did not improve over the following 4 days and diagnosis of pyloric stenosis was made. The antibiotics were commenced on admission due to initial suspicion of sepsis. Unfortunately there was no Ultrasound capabilities at the hospital.

In the setting there was no neonatal surgical options- and no possibility of referral to an appropriate centre (nearest Hospital offering possible neonatal surgery is in the capital, Dar-es-salaam, approximately 10 hours drive). Therefore, the patient was commenced on medical treatment for pyloric stenosis.

The patient was then started on IV Atropine 10% 0.01mg/kg, given slowly shortly before giving small amounts of expressed breast milk (10mls). This treatment was repeated 4 hourly, meanwhile IV fluids were continued. The initial day the patient tolerated all milk, and so the treatment was continued and feeds were increased slightly (20mls). By the third day the patient only vomited twice and atropine was changed to oral (0.02mg/kg) preparation. The baby was allowed to breast feed, with an administration of atropine each time prior. The IV fluids were weaned off over the course of 4 days. Over the course of the following week the condition gradually improved, the patient was gaining weight and the episodes of vomiting were few 1-2/day, and thus oral atropine was continued and gradually the dose was reduced. The baby experienced flushing and tachycardia when the IV atropine was administered however this was a brief <2 minutes and self limiting. The patient was discharged on day 10 with continued oral atropine to be taken at home on a weaning dose over the following 2 weeks.

At follow-up one week after the discharge the patient was well, symptoms were nearly resolved- only 2 small vomits since discharge. He was tolerating breast feeding and gaining weight. Atropine was thus stopped and we asked to come back again in two weeks but unfortunately the mother and the baby didn't come. However we managed to trace the patient via the community health worker and through telephone consultation a month later, discovered that he was well, and symptom free – which is why the mother did not bring him back to follow-up.

DISCUSSION

The case presented is of the successful medical management of pyloric stenosis in a resource poor setting. Pylorotomy has tended to become the favoured method of treatment as with expert paediatric, surgical, anaesthetic and nursing services and specialised accommodation for infants, the outcome is good with low mortality, short stay in hospital and few complications.

However, a variety of recent studies of medical treatment with anticholinergic drugs (atropine) have produced successful outcomes. It is cheap and relatively easy to administer, safe and an effective alternative to surgical management [21].

Standing on these studies the success rate of atropine treatment in pyloric stenosis is promising: it ranges from 66.7% to 91.6% with no severe adverse events. Only transient tachycardia, flushed skin and increased alanine aminotransferase have been reported, after intravenous injection of atropine, but these side effects are rare and self limiting [23]. Intravenous atropine usually takes from 1 to 9 days to be effective, but more frequently it takes 7 days; then oral atropine must be continued, also after the discharge. Thus the length of hospital stay is usually longer for the medical treatment than for the surgery [21,20].

The major limit of the medical treatment with atropine is the length of stay and the need to continue the treatment after the discharge, which are not neglectable points in a poor setting. Meanwhile, in this setting the infection risk and moreover the lack of paediatric surgical and anaesthetic expertise availability, restrict the surgical approach. Thus, medical treatment with atropine can be considered as a suitable alternative and perhaps its use promoted in international guidance (WHO paediatric pocket book).

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