

Prevalence of Infantile Hypertrophic Pyloric Stenosis and relation to breast feeding trends in the Maltese Islands between 1995 and 2007

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Abstract

Introduction: Several studies have indicated that breastfeeding may protect against the development of infantile hypertrophic pyloric stenosis (IHPS). This study investigated trends in IHPS over the period 1995-2007 in Malta and analysed the relationship of IHPS and changes in infant feeding practices during the same time period.

Methods: Patients with IHPS were identified from Hospital Activity Analysis reports. The case notes were obtained and the following data were collected: birth order of the patient, gender and type of feeding. Breast feeding information trends were obtained from published reports. Population data were obtained from publications of the National Statistics Office.

Results: A total of 125 patients were operated for IHPS. Case records were available for 86. The incidence was 2.26 per 1000 live births. There was an expected preponderance of males with no mortality. Seventy one patients (82.5%) had been formula fed from day one, while ten patients were exclusively breast fed. Five patients were breast fed and supplemented with formula at presentation. Forty-six patients were first born infants. A nonsignificant downward trend was noted. IHPS was significantly less common in breast fed infants (X^2 72.4, $p < 0.001$).

Conclusion: These findings further support the hypothesis that breast feeding protects against the development of IHPS

Keywords

Epidemiology, Infantile pyloric stenosis, Malta

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Introduction

Infantile hypertrophic pyloric stenosis (IHPS) is a condition of early infancy in which gastric outlet obstruction occurs due to circular muscle hypertrophy at the pylorus. Although classed as a congenital defect, there is evidence that the condition develops after birth.¹ The aetiology of IHPS has not been fully elucidated. Although there is a genetic component,² some evidence suggests that postnatal factors may also play an important part. Clinical symptoms develop most frequently at 2–8 weeks of age and the typical presentation is the onset of initially non-bloody, always non-bilious vomiting at 4-8 weeks. Although vomiting may initially be infrequent, over several days it becomes more predictable, occurring at nearly every feed. Vomiting intensity also increases until the typical projectile vomiting ensues. Slight haematemesis of either bright red flecks or a coffee-ground appearance is sometimes observed. Patients are usually not unwell or febrile. The infant in the early stage of the disease remains hungry and sucks vigorously after episodes of vomiting. Prolonged delay in diagnosis can lead to dehydration, poor weight gain, malnutrition, metabolic alterations, and lethargy. Parents often report trying several different baby formulas because they assume vomiting is due to intolerance.^{2,3} Standard treatment consists of surgical pyloromyotomy and is generally curative.

Epidemiologically, IHPS is the most common condition requiring surgical intervention in the first year of life. It is most common in Caucasians and is relatively uncommon in Afro-Caribbeans and Asians. The condition has a reported incidence of 1–8 per 1000 live births. A recent decline in its incidence has been reported in a number of countries.⁴

A study on this condition has never been carried out in Malta and the aim of this study was to review the incidence of IHPS with time and attempt to identify associations with feeding practices.

Methods

A total of one hundred and twenty-five patients with infantile IHPS were identified between 1995 and 2007. Records were obtained from Hospital Activity Analysis reports which record all activity within the only general hospital on the island of Malta. The case notes of 86 patients were obtained (the rest could not be traced) and a questionnaire was filled in for each patient with regard to presentation and feeding methods (Figure 1).

The birth order of the patient, gender and the type of feeding were obtained from the case notes. Breast feeding information trends were obtained from published reports.⁵ Population data were obtained from publications of the National Statistics Office.⁶

The data were arbitrarily divided into 3 eras to reflect the years for which breast feeding statistics could be obtained: 1996-9, 2002-3, 2004-7.

Data were entered into an Excel spreadsheet and analyzed using X² test and X² test for trend. Tests were performed with Statcalc. A p value <0.05 was taken to represent a statistically significant result.

Results

The overall incidence of IHPS was calculated at 2.26 per 1000 live births which is within the incidence range for Europe. Cases were fairly evenly distributed over the 13 year period with the exception of one year in which no cases of IHPS were recorded (Table 1). Overall the incidence dropped from 2.7 per 1000 live births before 1999 to 1.6 per 1000 live births after 2005.

Seventy patients were male (81%) and sixteen were female (19%). There was no mortality. Over 95% of the patients presented with typical projectile vomiting by the fifth week of life. Poor weight gain, weight loss, constipation, irritability and jaundice were other presentations. Seventy one patients (82.5%) had been formula fed from day one, while ten patients

Table 1: Distribution of cases over period under investigation

Year	Pyloric stenosis (n)	Live births (n)
1995	10	4950
1996	11	4944
1997	17	4835
1998	12	4670
1999	12	4403
2000	9	4392
2001	7	3957
2002	11	3918
2003	11	4050
2004	8	3887
2005	0	3858
2006	5	3885
2007	12	3871

were exclusively breast fed. Five patients were breast fed and supplemented with formula at presentation. Forty-six patients were first born infants.

The years between 1996 and 2007 were divided into three four-year periods and the incidence rates were calculated for each of the periods. There was a downward trend from 1996-1999 to 2004-2007 (Figure 2) but this was not statistically significant, even when analysed by individual year for trend (Odds ratio = 0.13 [95% CL 0.07-0.23], p=0.098). The overall declining trend in IHPS incidence was associated with an increase in the incidence of breast feeding from 48.2% in the first period to 59% in the last period (Figure 2).

However, a 2 by 2 table (X²) for the entire group, comparing with total live births, with and without IHPS, and with and without breast feeding, was highly significant (Table 2 – X² with Yates continuity correction 72.4, p<0.001).

Discussion

Our 4:1 ratio of male to female cases has been well described in the literature.^{7,8}

This study showed no mortality and indeed, the combination of preoperative rehydration, skilled anaesthesia, and the use of the Fredet-Ramstedt operation (pyloromyotomy) have virtually eliminated mortality from uncomplicated infantile hypertrophic pyloric stenosis.^{8,9} Pharmacological agents for the treatment of IHPS including atropine have been tried successfully but treatment is prolonged and surgery remains the management of choice. Oral or intravenous atropine should be reserved for use in infants with significant co-morbidities. However, there is not enough evidence to support routine use of pharmacological agents instead of surgery.

The causes of infantile hypertrophic pyloric stenosis are multifactorial. Both environmental factors and hereditary

Figure 1: Pyloric stenosis audit sheet

Case Notes No: _____

Sex: Male Female

Birth order: 1 2 3 4 5 6

Year of operation: _____

Age at presentation: _____

Type of feeding: BF Formula BF+Formula

Duration of BF: _____

Figure 2: Rates for pyloric stenosis and breast feeding: 1996-2007

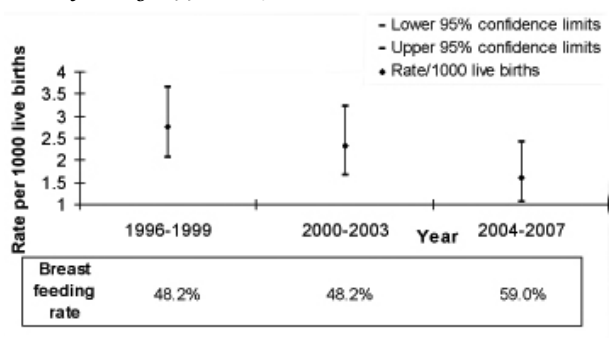


Table 2: Breast feeding and infantile pyloric stenosis (IPS)

	Breast feeding and mixed feeding	Formula feeding
With IHPS	15	71
Without IHPS	30904	18536

(type of feeding not known n=1172)⁵

factors are believed to be contributory. It has been suggested that persisting duodenal hyperacidity secondary to a high parietal cell mass, and loss of gastrin control produces pyloric stenosis from repeated pyloric contraction in response to hyperacidity.¹⁰ Explanations for a protective effect of breast feeding in pyloric stenosis can only be speculative but include the presence in human milk of high levels of hormones like vasoactive intestinal peptide that favour pyloric relaxation. Increased plasma gastrin concentrations in infants wholly or partially fed by bottle have also been found, and these may be associated with pylorospasm, pyloric hypertrophy, and consequent damage of peptide containing nerve fibres.¹¹ Finally, the presence of unknown confounders may also play a role in this condition.⁹

Our study has an important but unavoidable limitation insofar as only 68% of recorded cases of the condition could be traced. There is no reason to believe that this implies bias in that case notes are randomly lost. Moreover, breast feeding statistics in the early part of the study were incompletely kept and only quoted discharge breastfeeding rates and not rates at, for example, 6 weeks or 3 months. However, despite these limitations, our data showed a non-significant downward trend from 1996-1999 to 2004-2007 related to a drop in formula feeding with an Odds Ratio = 0.13 (95% CL 0.07-0.23).

This study is in agreement with most recent European studies relating IHPS to feeding practices. Several reports have shown a decreasing incidence in IHPS in the last several years.³ Some have postulated that the decrease in incidence is related to a decrease in formula feeding and a concurrent increase in breast feeding. In the absence of supporting biological data, a causal interpretation for this association is not yet warranted.⁸

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