Short reports

Pyloric stenosis: congenital or acquired?

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SUMMARY Pyloric muscle dimensions were measured in 1400 consecutive newborn infants. Nine of these subsequently developed pyloric stenosis. Their pyloric measurements at birth were all within the normal range. Congenital preformed muscular hypertrophy does not appear to be present in babies who later develop pyloric stenosis.

Infantile hypertrophic pyloric stenosis is usually classified as a congenital disorder. The condition is almost unknown in stillbirths, however, and usually presents with vomiting starting after the second week of life; this suggests that it may be acquired. Wallgren performed radiological examinations of the upper gastrointestinal tract of 1000 asymptomatic newborn infants and found that pyloric stenosis subsequently developed in five of them.¹ Barium studies performed in the newborn period showed no difference between the five who developed infantile hypertrophic pyloric stenosis and the others. The radiological studies, however, could not exclude a congenital preformed 'soft' hypertrophy of the pyloric muscle causing no constriction. Recently,

two dimensional ultrasonography has proved reliable for confirming the diagnosis of infantile hypertrophic pyloric stenosis.² The purpose of this prospective study was to establish whether pyloric hypertrophy detected by ultrasonography is present at birth in infants who subsequently develop this condition.

Subjects and methods

Altnagelvin Hospital serves the population of Londonderry and adjacent areas of Northern Ireland. The study was carried out in the maternity and paediatric units of the hospital during the five month period from 1 January 1987 to 31 May 1987. The pyloric dimensions of 1400 consecutive newborn babies were measured at the time of their routine postnatal examination using an ADR 4000 scanner with 5.5 MHz transducer. This provided the normal range of newborn pyloric dimensions and a baseline from which to assess those who would later develop pyloric stenosis and present to this hospital. The ultrasound criteria for infantile hypertrophic pyloric stenosis used was of a pyloric anterior to posterior diameter ≥ 15 mm or muscle thickness ≥ 4 mm (figure).^{3,4} Student's paired *t* test was used to

Figure (a) Transverse sonogram. Normal pylorus of newborn. (b) Transverse sonogram. Characteristic 'target sign' of infantile hypertrophic pyloric stenosis. The distance between the crosses shows the thickness of the hypoechoic muscle mass. The cross section of the pylorus was also measured (anterior-posterior diameter).

Table Pyloric dimensions in infants studied. Results are mean (± 2 SD)

	Anterior-posterior diameter (mm)	Muscle thickness (mm)
Newborn infants (n=1391)*	8.8 (± 2.2)	2.1 (± 0.6)
Range	7.0–11.0	1.0–3.0
Infantile hypertrophic pyloric stenosis (n=9)	15.7 (± 0.6)	4.9 (± 0.14)
Range	13.0–23.0	4.0–6.0
Infantile hypertrophic pyloric stenosis (values on day 3) (n=9)	8.3 (± 0.17)	1.8 (± 0.09)
Range	7.0–10.0	1.0–2.0

*The nine infants later developing infantile hypertrophic pyloric stenosis were excluded from the normal range.

determine the significance of the change in pyloric dimensions from birth to presentation with infantile hypertrophic pyloric stenosis.

Results

A total of 1400 consecutive newborn infants had pyloric dimensions measured at routine postnatal examination. Nine of these infants subsequently presented to this hospital with infantile hypertrophic pyloric stenosis, which in all cases was confirmed by surgery. The pyloric dimensions recorded are summarised in the table.

The initial pyloric dimensions of the nine infants who presented with infantile hypertrophic pyloric stenosis were no different from the other 1391 healthy newborns. However, there was a highly significant increase ($p < 0.001$) in the size of the

pylorus by the time infantile hypertrophic pyloric stenosis was diagnosed.

Discussion

This study shows that pyloric muscle hypertrophy is not present in the early newborn period of infants who later develop infantile hypertrophic pyloric stenosis. It excludes the possibility of a congenital preformed 'soft' hypertrophy of pyloric muscle without constricting effects suggested by Wallgren. The incidence of infantile hypertrophic pyloric stenosis in this study group was 6.4/1000 live births and is in keeping with the previously reported high rising incidence in this area.⁵ We conclude that infantile hypertrophic pyloric stenosis should no longer be referred to as 'congenital' pyloric stenosis, although the true aetiology of this acquired condition remains to be elucidated.

References

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Plasma aldosterone and renin activity

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SUMMARY Eight infants had paired measurements of plasma aldosterone and plasma renin activity while being treated for congestive heart failure. There is parallelism with aldosterone and renin activity in the presence of a hyperactive renin-angiotensin-aldosterone system. Six patients had plasma renin activity and plasma aldosterone measured after commencing captopril and we have shown biochemical blockade of the renin-angiotensin-aldosterone system.

We have recently reported raised plasma renin activity in infants being treated for congestive heart failure due to congenital heart disease.¹ In that study we were unable to report the results of plasma aldosterone because concentrations fell outside the range of the assay in most instances. We have performed an additional study to determine whether parallelism between plasma renin activity and plasma aldosterone, which has been described in normal infants,² continues when the renin-angiotensin-aldosterone system is hyperactive, and whether this