Heart failure apparently due to overfeeding in a neonate

Congestive cardiac failure in the newborn period is usually due to congenital heart disease, endocardial fibrolastosis, storage diseases, and occasionally, to infections. Heart failure also occurs iatrogenically due to fluid overload during intravenous therapy. We report what we believe to be the first recorded case of heart failure in infancy due to overfeeding by the mother.

Case report

The baby, a girl, was born by normal delivery at 38 weeks’ gestation after an uncomplicated pregnancy to a healthy 25-year-old woman and was her first child. Birthweight was 2670 g which was between the 10th and 25th centiles for gestational age. There were no problems and she was breast fed, being discharged home on the 5th day weighing 2620 g. The mother took the baby to her local infant welfare clinic and was told by the doctor that as the baby was small she should be fed as much as possible. The mother then supplemented breast feeding by giving the baby 200 ml/kg per day of a mixture of two-thirds cows’ milk and one-third water and, during the next 2 weeks, the baby gained some 500 g (Fig. 1). As the mother felt that breast feeding was inadequate, she changed over completely to bottle feeding with feeds every 3 hours, each lasting one hour, and a total intake of some 300 ml/kg per day. By the 4th week of life the infant had risen from the region of the 10th to the 50th centile (Fig. 1) but she was by now feeding poorly, breathless, and vomiting. She was seen at hospital and immediately transferred to our newborn intensive care unit as she was in severe congestive heart failure.
unremarkable except for a QT interval of 0·3 second. The patient was hypoxic while breathing air but $P_{O_2}$ rose to 32·7 kPa (242 mmHg) breathing oxygen. Blood urea nitrogen was 2·7 mmol/l (16 mg/100 ml), Na 134 mmol/l, and K 5·1 mmol/l. Serum Ca was considerably reduced at 1·33 mmol/l (5·3 mg/100 ml), and P raised at 2·62 mmol/l (8·1 mg/100 ml). Haematocrit was 12 g/100 ml and other haematological values were unremarkable. Cultures of blood and CSF gave no evidence of bacterial or viral infection.

Treatment was begun immediately with digoxin, frusemide, and calcium gluconate IV with total fluid restricted to approximately 100 ml/kg per 24 hours. She rapidly improved during the next 4 days, weight falling to below the 10th centile (Fig. 1), and all signs of heart failure disappearing. Chest x-ray returned to normal (Fig. 2b). Serum Ca rose to 1·8 mmol/l (7·2 mg/100 ml) by 12 hours after starting treatment and calcium therapy was stopped after one week without any problem. The prolonged QT interval had also returned to normal (0·13 second).

The baby was discharged home after 10 days being fed 150 ml/kg per day of humanised milk (Similac) and at follow-up 3 weeks later she was completely well although her weight had again reached the 50th centile, Fig. 1.

She has remained well and was last seen aged 13 months weighing 8·97 kg (25th centile).

**Discussion**

We believe that we excluded the common causes for heart failure in this infant and her recovery was so rapid and complete that we find it difficult to believe that it was due to viral myocarditis. It seems likely that fluid overload was the basic cause but the hypocalcaemia, itself probably due to the excessive intake of cows' milk, may have contributed (Troughton and Singh, 1972). Even allowing for the dilution of the milk used, the much higher concentration of sodium in cows' milk compared with breast milk and the huge amounts given could well have exceeded the renal capacity for excretion. In addition the known inability of the immature kidney to handle a large water intake could have been instrumental in causing the heart failure (Ames, 1953). In this context it is interesting that the infant was subsequently able to gain weight rapidly on a low sodium milk without developing heart failure, presumably because this was a true increase in tissue and not just water retention.

**Summary**

Severe heart failure in a one-month-old infant is described, apparently due to the administration of cows' milk in the region of 300 ml/kg per day. There was also pronounced hypocalcaemia. The infant responded rapidly to treatment with no evidence of intrinsic heart disease.
Adenoidectomy

An evaluation of the indications

For over 100 years it has been accepted that the symptoms associated with disease of the adenoids are: nasal obstruction with mouth breathing, snoring, recurrent earache, anterior and posterior nasal discharge, cough, and speech defect (Meyer, 1870).

Many authorities feel that these symptoms are due to simple hypertrophy of the adenoids; in Gray’s Anatomy it is stated that overgrowth of the adenoid obstructs respiration through the nose and results in mouth breathing, and causes deafness by blocking the eustachian tube (Davies and Davies, 1962). A statistical correlation has been shown between the size of the adenoid (ascertained by clinical examination) and the presence of fluid in the ear in a group of children (Murray et al., 1968).

However, Mawson (1971) postulates that hypertrophy of the adenoid causes only recurrent earache, mouth breathing, and snoring, and that the other symptoms are due to chronic adenoiditis. Others have stated that the concept of an infected adenoid is incorrect, that the symptoms are due to hyperplasia alone, and that septic foci have not often been shown histologically in the adenoid (Guida, 1930; Lemere, 1932; Osborne and Roydhouse, 1976).

A random survey of ENT surgeons (Hibbert, 1977) showed that 80% felt that a history from the mother of nasal obstruction and snoring was important in the diagnosis of enlarged adenoids. 75% of surgeons considered that it was the size of the adenoid which caused the symptoms.

The purpose of the present study was to investigate the relation between the signs and symptoms usually attributed to adenoid hypertrophy and the actual size of the adenoid removed at surgery.

Material and method

76 children were chosen consecutively from the waiting list. There were 50 boys (median age 6 years 9 months; range 2 years 11 months–10 years 9 months) and 26 girls (median age 7 years 6 months; range 4 years 10 months–10 years 10 months). The parents of the children were interviewed by one of us (J.H.) and asked about symptoms. The child was then examined for mouth breathing, abnormality on anterior rhinoscopy, and serous otitis media. The presence of a symptom or sign was scored as +1. No attempt was made to differentiate between degrees of any sign or symptom. The adenoids were then removed (by J.H.) using a standard technique and washed, dried, and weighed.

Analysis of the data. The results were arranged in order of increasing weights of adenoids and then divided into 4 groups by quartiles. The number of positive scores for each sign and symptom was noted for each of these groups. The data were submitted to a χ² test to determine if the total number of positive scores increased with increasing size of adenoids. Each symptom and sign was tested separately to assess whether there was a correlation between scores and increasing size.

The randomness of distribution of the scores was also assessed by the theory of runs using the data arranged in order of weight of the adenoids.

The weight of the adenoid may vary with age and thus interfere with the distribution of the symptoms against the weight. Therefore regression of the adenoidal weight against age was done by the least squares method (using log₁₀ weight since the weight of the adenoid follows a log normal distribution (Hibbert, 1978)).

Results

The incidence of the various symptoms and signs is shown in the Table, divided by quartiles according to weight of the adenoid. Increasing frequency of scores with increasing weight was recorded for snoring only (χ² = 9·32, 3 d.f., P < 0·05). The results for the remaining observations were non-significant. In addition a χ² test for trend showed that there was significant regression for the snoring group (χ²₄₂ = 7·97, 2 d.f., P < 0·05).

The number of runs for any of the symptoms and signs did not show a significant deviation from randomness.

There was no correlation between the log weight and the age (r = -0·06, t = 0·52, 74 d.f.). Age dependent variation in weight was thus excluded as a factor interfering with a random distribution.
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