Increased sodium requirement following early postnatal surgical correction of congenital uropathies in infants

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Abstract. Serum electrolyte equilibrium and plasma aldosterone concentrations were monitored in 19 infants who had severe obstructive uropathy or grade 5 vesico-ureteral reflux and were undergoing surgical correction in the first 2 months of life. Before surgery high plasma aldosterone levels were observed in 8 patients, but serum sodium and potassium concentrations were normal. Plasma concentrations of aldosterone were elevated in all patients during the week following surgery and 7 patients developed severe hyponatraemia, hyperkalaemia and weight loss despite very high plasma aldosterone concentrations. As a consequence 5 infants were infused with sodium chloride (4 mEq/kg per day) before and for 36 h after surgery; this prevented metabolic imbalance. We conclude that infants undergoing surgical correction of uropathies may require a high sodium intake to maintain electrolyte balance and adequate growth.

Key words: Obstructive uropathy - Serum electrolytes - Aldosterone - Sodium chloride infusion

Introduction

Surgical correction of congenital uropathies during the neonatal period is being performed with increasing frequency, in part because of prenatal diagnosis. It is hoped that early surgical relief might ameliorate the long-term prognosis. Although the success of the surgical procedure in early life varies surgical complications have been reported only rarely [1–3].

Obstructive uropathy and severe reflux may, however, affect tubular and glomerular functions and cause metabolic disorders. Hyperkalaemic acidosis and pseudohypoaldosteronism with severe salt loss have been reported in the presence or absence of decreased glomerular filtration in both man and animals [4–7]. Derangements in water and electrolyte metabolism have also been described as a consequence of the altered tubular and glomerular functions that may occur in the postobstructive period, and have been called the "postobstructive syndrome" [8–12]. This condition has not been specifically studied in early infancy, to our knowledge.

In the last 3 years 5 infants in our department who had been operated on for uropathies at 5–30 days of life developed acute renal failure characterized by severe weight loss (>15% body weight), hyponatraemia (<120 mEq/l) and oliguria. This occurred 5–20 days postoperatively, in the absence of surgical complications. This observation led us to study prospectively water and electrolyte metabolism in the perioperative period in 24 consecutive infants undergoing surgery in the first 2 months of life.

The specific aim of this study was to ascertain whether tubular resistance to aldosterone might be a cause of electrolyte disorders and lead to acute renal failure in infants, even after correction of the uropathy.

Patients and methods

Twenty-four infants were admitted to this study prospectively over a period of 15 months. All had been referred to our centre after prenatal diagnosis of obstructive uropathy by ultrasound examination. This was confirmed in all cases by cystography, i.e. urography and 99m technetium-DTPA renal scan. The decision to operate was made when severe hydronephrosis was present. Surgery was performed at 4–70 days of life (median 40 days). The diagnoses were pyelo-ureteral obstruction (13 cases, 2 bilateral), ureterovesical obstruction (2 bilateral cases), vesico-ureteral reflux with mega-ureter (4 cases, 2 bilateral), double ureters with obstruction (3 cases, 2 bilateral) and ureteral valves (2 cases).

All infants were born at term. In all cases their pre-operative condition was good. Urine cultures were performed weekly before surgery and were sterile. Infants with reflux were given antibiotic prophylaxis. Serum electrolytes, serum creatinine, acid-base equilibrium, blood urea nitrogen, urinary electrolytes and plasma aldosterone concentrations (P ald) were measured before, as well as 5 and 20 days after operation. The urinary potassium (K) to sodium (Na) ratio (U K/Na) was measured as an index of tubular response to aldosterone.

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Table 1. Selected studies in 19 infants with congenital uropathies undergoing surgery in the first 2 months of life

<table>
<thead>
<tr>
<th></th>
<th>Pre-operative values</th>
<th>Postoperative values</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>5 days</td>
</tr>
<tr>
<td>SCR (mg/dl)</td>
<td>0.5 ± 0.2</td>
<td>0.5 ± 0.3</td>
</tr>
<tr>
<td>SK (mEq/l)</td>
<td>5.3 ± 0.4</td>
<td>6.3 ± 0.6*</td>
</tr>
<tr>
<td>SNa (mEq/l)</td>
<td>138 ± 3</td>
<td>133 ± 6*</td>
</tr>
<tr>
<td>UK/Na</td>
<td>1.8 ± 0.8</td>
<td>2.6 ± 0.7</td>
</tr>
<tr>
<td>P aldo (pg/ml)</td>
<td>1124 ± 811</td>
<td>5187 ± 5956*</td>
</tr>
</tbody>
</table>

* P <0.05 compared with pre-operative values

SCR, Serum creatinine; SK, serum potassium; SNa, serum sodium; UK/Na, urinary potassium to sodium ratio; P aldo, plasma aldosterone concentration

a Mean ± SD

An infusion with 5% dextrose and 1–2 mEq/kg per day sodium chloride (NaCl) was started 12 h before surgery and continued for 36 h. The infants were then fed with maternal milk or formula. When adequate calories were being taken, the infusion was stopped. This occurred in most cases within 72 h of surgery.

The last 5 patients included in the study were followed as above, but received an i.v. infusion of 4 mEq/kg per day NaCl for 36 h, beginning 12 h before surgery, and an oral NaCl supplement of 2 mEq/kg per day for 1 week.

Results

Table 1 summarizes pre- and postoperative values for serum creatinine, K and Na, UK/Na and P aldo levels in the first 19 infants who were admitted to this study. Before surgery all the infants had normal serum values for creatinine, K and Na. UK/Na ratios were comparable with values found in a group of normal control infants. The mean P aldo was within the normal range, but 8 infants had values exceeding the upper limit (1200 pg/ml) found in control infants in the first 2 months of life.

Postoperatively there was a significant increase in serum K and a significant decrease in serum Na (P <0.05 for both by the paired Student’s t-test). UK/Na did not differ from pre-operative values. The mean P aldo of the group rose significantly. In 7 infants who had severe hyponatraemia (<132 mEq/l) and hyperkalaemia (>6.5 mEq/l, Fig. 1) values for P aldo were extremely high (>2500 pg/ml) and metabolic acidosis was present (serum bicarbonate <18 mEq/l). Their UK/Na ratios were markedly decreased. Infusion with normal saline and bicarbonate was necessary for these infants: oral supplementation with 2 mEq/kg per day was required to allow good weight gain. This was continued for 2 months and then withdrawn progressively.

The data for the group of 5 infants who received a NaCl supplement are shown in Fig. 2. There was no change postoperatively in serum electrolytes or P aldo. The acid-base equilibrium remained normal in all cases.

Fig. 1. Plasma aldosterone concentrations, serum creatinine (SCR), serum potassium (SK) and serum sodium (SNa) and urinary potassium to sodium ratio (UK/Na) in 7 infants with congenital uropathies undergoing surgical intervention.

a Mean value with (SD); ■ patients with bilateral lesion; □ patients with unilateral lesion; * P <0.01 compared with pre-operative value

Fig. 2. Plasma aldosterone concentration, SCR, SK, SNa and UK/Na in 5 infants with congenital uropathies undergoing surgical correction in the first 2 months of life. These infants received an increased Na supplement.

a Mean value with (SD); ■ patients with bilateral lesion; □ patients with unilateral lesion; * P <0.01 compared with pre-operative value