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# A pediatric patient with hyponatremic hypertensive syndrome without persistent hypertension in acute phase: A case report and review of literature

**Nodoka Hinokuma[1], Shunsuke Sakurai[1], Atsutoshi Shiratori[1], Keiko Nagahara[1], Yoshifusa Abe[2], Takeshi Shimizu[3], Takanari Fujii[3], Katsumi Mizuno[1] and Hideshi Tomita[3]**

**Abstract**

Hyponatremic hypertensive syndrome is characterized by hypertension, hyponatremia, and hypokalemia due to unilateral renal artery stenosis. We herein report a 1-year-old hyponatremic hypertensive syndrome infant without persistent hypertension in the acute phase. On the ninth hospital day, his systolic and diastolic blood pressure increased up to 154–160 and 70–84 mmHg, respectively. Acute gastroenteritis and dehydration might transiently mask his hypertension. By percutaneous transluminal balloon angioplasty for right renal artery, his blood pressure finally normalized without antihypertensive drugs. We reviewed 23 previously reported pediatric patients with hyponatremic hypertensive syndrome under the age of 15 years. Including our patient, there are only three reports on hyponatremic hypertensive syndrome without persistent hypertension in the acute phase. Hyponatremic hypertensive syndrome is curable with proper diagnosis and timely intervention. Therefore, pediatricians should pay attention to the signs and symptoms associated with hyponatremic hypertensive syndrome, even if persistent hypertension was absent in the acute phase.

**Keywords**

Hyponatremic hypertension syndrome, renovascular hypertension, children

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## Introduction

Hyponatremic hypertensive syndrome (HHS), which was first named by Brown et al. in 1965, is characterized by hypertension, hyponatremia, and hypokalemia due to unilateral renal artery stenosis or other kidney diseases.[1] By renin release from kidney on stenosis side, renin–angiotensin–aldosterone system (RAAS) is increased. Acceleration of RAAS causes hypertension, glomerular hyperfiltration leading to proteinuria, and pressure diuresis leading to hyponatremia from the healthy side kidney.[2] In adults, HHS patients with renal artery stenosis are not so rare,[3] but reports involving children are few.[4] We report herein a 1-year-old HHS infant without remarkable hypertension in the acute phase.

## Case report

The patient was a 12-month-old boy who was the first child of healthy non-consanguineous Japanese parents. He had egg allergy requiring elimination. He was delivered spontaneously at 40 weeks and 2 days of gestation. His birth weight was 3084 g (+0.21 standard deviation (SD), 50–75th percentile in Japan), body length was 50 cm (+0.48 SD, 50–75th percentile in Japan), and head circumference was 33.0 cm (−0.27 SD, 25–50th percentile in Japan). Phototherapy was performed due to jaundice at 1, 2, and 4 days of age. Poor weight gain was noticed during health examination at 3–4 and 6–7 months. He had fever 4 days before admission. The next day after fever onset, he was taken to a local physician because of vomiting and referred to our hospital. At 2, 3, and 5 days after fever onset, drip infusion therapy was repeatedly

[1]Department of Pediatrics, Showa University School of Medicine, Tokyo, Japan
[2]Children's Medical Center, Showa University Koto Toyosu Hospital, Tokyo, Japan
[3]Pediatric Heart Disease and Adult Congenital Heart Disease Center, Showa University Hospital, Tokyo, Japan

**Corresponding Author:**
Yoshifusa Abe, Children's Medical Center, Showa University Koto Toyosu Hospital, 5-1-38 Toyosu, Koto-ku, Tokyo 135-8577, Japan.
Email: yoshifusa@med.showa-u.ac.jp

performed at the outpatient service without dramatic improvement. Hence, he was finally admitted to our hospital.

On admission, his height was 73 cm (−0.77 SD, 10–25th percentile in Japan), his body weight was 7.3 kg, which was 6% lower than the pre-illness weight 7.8 kg (−1.3 SD, 3–10th percentile in Japan), body temperature, 37.0°C; heart rate, 124 bpm; respiratory rate, 42/min; and systolic blood pressure, 124 mmHg (≥95th percentile + 12 mmHg). It did not persist and spontaneously decreased and his systolic blood pressure was around 90th percentile until the eighth hospital day even after correction of dehydration, as shown in Table 1. Facial skin showed pallor, and enophthalmos was present, indicating dehydration. Hematological values were as follows: white blood cell count, 5600/μL; hemoglobin, 15.5 g/dL; and platelet, $9.4 \times 10^4$/μL. Blood chemistry and serologic findings were as follows: total protein, 7.3 g/dL; albumin, 4.7 g/dL; total bilirubin, 2.0 mg/dL; urea nitrogen, 19.7 mg/dL; creatinine, 0.29 mg/dL; uric acid, 9.3 mg/dL; aspartate aminotransferase, 69 U/L; alanine aminotransferase, 26 U/L; lactic dehydrogenase, 767 U/L; creatine kinase, 117 U/L; sodium, 127 mEq/L; potassium, 3.3 mEq/L; chloride, 85.2 mEq/L; glucose, 55 mg/dL; and C-reactive protein, 0.09 mg/dL. Venous blood gas analysis showed the following: pH, 7.456; $pCO_2$, 28.9 mmHg; $pO_2$, 39.7 mmHg; $HCO_3^-$, 19.9 mmol/L; base excess, −2.6 mmol/L; anion gap, 18.8 mmol/L; and lactate, 2.6 mmol/L. Urinalysis revealed a 2+ test for urine protein, 2+ for occult blood, and 1+ for ketone, but negative for urine sugar. Urinary β-2 microglobulin was 1086 μg/L (normal: ≤250 μg/L); α-1 microglobulin, 10.6 μg/mL (normal: ≤5 μg/mL); and *N*-acetyl-β-D-glucosaminidase (NAG), 13.2 U/L (normal: ≤5 U/L). Fractional excretion of sodium (FENa) and potassium (FEK) were 3.8% and 27.9%, respectively. Ultrasonographic study of the kidneys showed a difference in long axis diameter, where right and left was 4.7 and 6.7 cm, respectively. Echocardiography on 33th day of admission showed pulmonary hypertension, left ventricular hypertrophy, and cardiomegaly. Left ventricular mass index (LVMI) was 112.5 g/m$^2$ and left ventricular ejection fraction was 85%.

Hypoglycemia was treated with intravenous 12 mL of 10% glucose. Fluid therapy with isotonic solution was performed. At 12 h after admission, the amount of drip infusion was about 450 mL. His body temperature was 37.5°C; heart rate, 118 bpm; and respiratory rate, 28 breath/min. Assessed by blood gas analysis sample, sodium and potassium levels decreased to 122.8 and 2.72 mEq/L, respectively. Urinary osmolality was 327 mOsm/kg. To avoid further lowering of sodium level, syndrome of inappropriate secretion of antidiuretic hormone (SIADH) was taken into consideration. Hence, fluid intake was restricted. Approximately 30 h later, sodium improved to 135.7 mEq/L. Hypokalemia improved by 5.75 mEq/kg/day of potassium oral administration. On the sixth hospital day, pre-illness weight was achieved. Until the eighth hospital day, systolic blood pressure and urine volume were 100–116 mmHg and 278–1165 mL/day, respectively. On the ninth hospital day, it was revealed that he had no ophthalmologic complication such as cataract or coloboma associated with renal tubular dysfunction. Those were screened by an ophthalmologist using mydriatic agent. In the afternoon, his blood pressure suddenly elevated. Systolic and diastolic pressures were 154–160 and 70–84 mmHg, respectively. Urine volume increased to 2290 mL/day. To investigate electrolyte and endocrinological problems, renin and aldosterone values were measured. Plasma renin activity (PRA) was 66.6 ng/mL/h (normal: 0.2–3.9 ng/mL/h) and plasma aldosterone level was 1112 pg/mL (normal: 35.7–240.0 pg/mL) on the second day of hospitalization.

As shown in Figure 1(a), the diuretic renogram showed right kidney dysfunction. Contrast computed tomography did not detect renal artery stenosis (Figure 1(b)), but selective angiography revealed right renal artery stenosis at 2/3 of the site from aorta to branch (Figure 1(c)); the stenosis ratio was 99%, indicating that it led to increased plasma renin and aldosterone levels and hypertension. Brain magnetic resonance imaging (MRI) and MR angiography showed no other vascular lesion. As shown in Figure 1(d), continuous intravenous infusion of nicardipine and oral administration of doxazosin were required to manage hypertension. We also added oral administration of furosemide, spironolactone, and carvedilol for myocardial hypertrophy and tachycardia. He needed intravenous nicardipine up to 10.5 μg/kg/min. It was finally switched to oral use.

On the 65th hospital day, percutaneous transluminal balloon angioplasty (PTA) for right renal artery was performed. After PTA, proteinuria disappeared on 84th hospital day. Increased PRA (19.3–320.3 ng/mL/h) and plasma aldosterone level (2188.8–4518.0 pg/mL) lasted up to 16 months of age. Hence, doses of antihypertensive drugs were gradually decreased. Intravenous nicardipine was stopped on 88th hospital day (at the age of 15 months). At the age of 22 months, it was revealed that PRA and plasma aldosterone level were within normal limits and there was no right renal artery restenosis evaluated by angiography. At about 1 year and 2 months post-catheter intervention (at the age of 2 years and 4 months), we finally stopped the prescription of all antihypertensive drugs. His blood pressure was 110/60 mmHg; height, 89.2 cm (+0.28 SD, 50–75th percentile in Japan); and body weight, 13.9 kg (+1.23 SD, 75–90th percentile in Japan). At the age of 2 years and 5 months, LVMI was 63.2 g/m$^2$. At the age of 2 years and 8 months, his blood pressure was 100/50 mmHg (<90th percentile).

## Discussion

Our patient had hypertension, hyponatremia, hypokalemia, metabolic alkalosis, and proteinuria secondary to right renal artery stenosis. Although his blood pressure was transiently high on admission, subsequently he did not show remarkable hypertension in acute phase.

Our patient had hypertension, hyponatremia, hypokalemia, increased PRA, hyperaldosteronemia, proteinuria, renal tubular dysfunction, and renal artery stenosis in the right kidney. Urinary potassium level more than 20 mEq/L and FEK more than 7% suggested that hypokalemia was due to renal loss. Urinary chloride level more than 15 mEq/L

**Table 1.** Blood pressure and biochemical data in our patient.

| Hospital day | −3 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | Normal values |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Blood pressure | | | | | | | | | | | | | |
| Systolic (mmHg) | | 124 | 92–116 | 96–102 | 98–118 | 80–104 | 90–110 | 102–112 | 108–116 | 112–160 | 107–169 | 102–150 | 98[a] |
| Diastolic (mmHg) | | | 54–68 | 58–60 | 50–56 | 52–56 | 54–70 | 53–58 | 52–58 | 48–84 | 41–94 | 37–78 | 52[a] |
| The number of measurement | | 1 | 2 | 3 | 5 | 4 | 4 | 4 | 4 | 11 | 25 | 7 | |
| Laboratory data | | | | | | | | | | | | | |
| Blood | | | | | | | | | | | | | |
| Sodium (mEq/L) | 133.5 | 126.7–132 | 134.4–138.7 | 140.3 | 136.5 | 138 | | | | 140.3 | 145 | 146 | 135–145 |
| Potassium (mEq/L) | 3.3 | 2.8–3.3 | 3.2–3.9 | 4.9 | 3.5 | 3.7 | | | | 2.9 | 4.1 | 3.6 | 3.6–4.8 |
| Chrolide (mEq/L) | 93.7 | 85.2–92.1 | 100.8–105.8 | 109.1 | 104.1 | 104.8 | | | | 106 | 111.2 | 112.7 | 98–108 |
| Magnesium (mg/dL) | | 2.1 | 1.8–2.0 | 2.0 | | | | | | 2.6 | 2.4 | 2.0 | 1.8–2.5 |
| Blood urea nitrogen (mg/dL) | 11.1 | 13.6–19.7 | 4.3–6.3 | 7.0 | 3.8 | 4.0 | | | | 5.2 | 10.4 | 7.4 | 6–20 |
| Uric acid (mg/dL) | 5.3 | 7.3–9.4 | 2.9–3.6 | 2.9 | 2.9 | 3.2 | | | | 3.0 | 5.1 | 4.9 | 2.3–5.8 |
| Creatinine (mg/dL) | 0.26 | 0.22–0.29 | 0.21–0.23 | 0.22 | 0.22 | 0.23 | | | | 0.19 | 0.28 | 0.29 | 0.16–0.32 |
| Glucose (mg/dL) | 111 | 55–101 | 96–126 | 93 | 100 | 93 | | | | 137 | 97 | 96 | 70–109 |
| PRA (ng/mL/h) | | | 66.6 | | | | | | | 188.2 | | | 0.2–3.9 |
| Plasma aldosterone (pg/mL) | | | 1112 | | | | | | | 831.9 | | | 35.7–240.0 |
| Osmolality (mOsm/kg) | | | 266 | | | | | | | | | | 275–290 |
| pH | 7.511 | 7.456–7.547 | 7.468–7.500 | 7.456 | 7.502 | 7.481 | | 7.459 | | 7.443 | 7.456 | 7.45 | 7.350–7.450 |
| $pCO_2$ (Torr) | 30.9 | 28.4–28.9 | 34.4–38.8 | 36.9 | 33.8 | 33.2 | | 31.8 | | 34.9 | 32.2 | 32.9 | 35–45 |
| Bicarbonate (mmol/L) | 24.2 | 19.9–24.1 | 24.4–28.1 | 25.4 | 25.9 | 24.2 | | 22.1 | | 23.3 | 22.2 | 22.4 | 22–26 |
| Base excess (mmol/L) | 2.0 | −2.6 to 2.6 | 1.2–4.3 | 1.7 | 3.1 | 1.3 | | −1.2 | | −0.3 | −0.8 | −1.1 | −2.0 to 2.0 |
| Urine | | | | | | | | | | | | | |
| Protein creatinine ratio (g/gCr) | | | 8.6 | 4.4 | 2.1 | | | 0.82 | 1.5 | | | Negative | <0.15 |
| Sodium (mEq/L) | | 47.2 | 40.6 | 47.8 | 31.5 | | 45.2 | 64.5 | 82.1 | | | 121.3 | Not available |
| Potassium (mEq/L) | | 8.9 | 57 | 97.2 | 25.7 | | 9 | 11.3 | 10.3 | | | 18.1 | ⩽15[b] |
| Chrolide (mEq/L) | | 49.7 | 74.7 | 89.1 | 48.5 | | 49.2 | 69.8 | 86.4 | | | 118.7 | ⩽15[c] |
| Calcium creatinine ratio (g/gCr) | | | 0.55 | 0.21 | | | | 0.56 | | | | 0.94 | 0.40–0.67 |
| β-2 microglobulin (μg/L) | | 1086 | 4868 | | | | 230 | | | | | 129 | ⩽250 |
| α-1 microglobulin (μg/mL) | | | 10.6 | | | | 1.5 | | | | | 0.7 | ⩽5 |
| NAG (U/L) | | | 13.2 | 28.9 | | | 7.8 | | | | | 2.5 | ⩽5 |
| Specific gravity | | 1.004 | 1.012 | 1.018 | 1.006 | 1.003 | 1.003 | 1.006 | 1.006 | | 1.006 | 1.007 | 1.002–1.030 |
| Osmolality (mOsm/kg) | | | 327 | 473 | | | | 230 | | | | 315 | 40–1200 |
| FENa (%) | | 2.8–3.8 | 0.35 | 0.16 | 0.25 | | | | | | | 2.2 | <1 |
| FEK (%) | | 22.6–27.9 | 20.9 | 9.1 | 8.1 | | | | | | | 13.3 | ⩽6[d] |
| FEUN (%) | | | 33.7 | 20.8 | | | | | | | | 48.1 | <35 |

PRA: plasma renin activity; NAG: *N*-acetyl-β-D-glucosaminidase; FENa: fractional excretion of sodium; FEK: fractional excretion of potassium.

[a]Blood pressure < 90th percentile for 1-year-old boy.[5]

[b]>15 mEq/L indicates renal loss of potassium under hypokalemia.[6]

[c]>15 mEq/L indicates chloride-resistant metabolic alkalosis involving increased PRA and hyperaldosteronemia without volume loss.[6]

[d]>6% indicates renal loss of potassium under hypokalemia.[7]

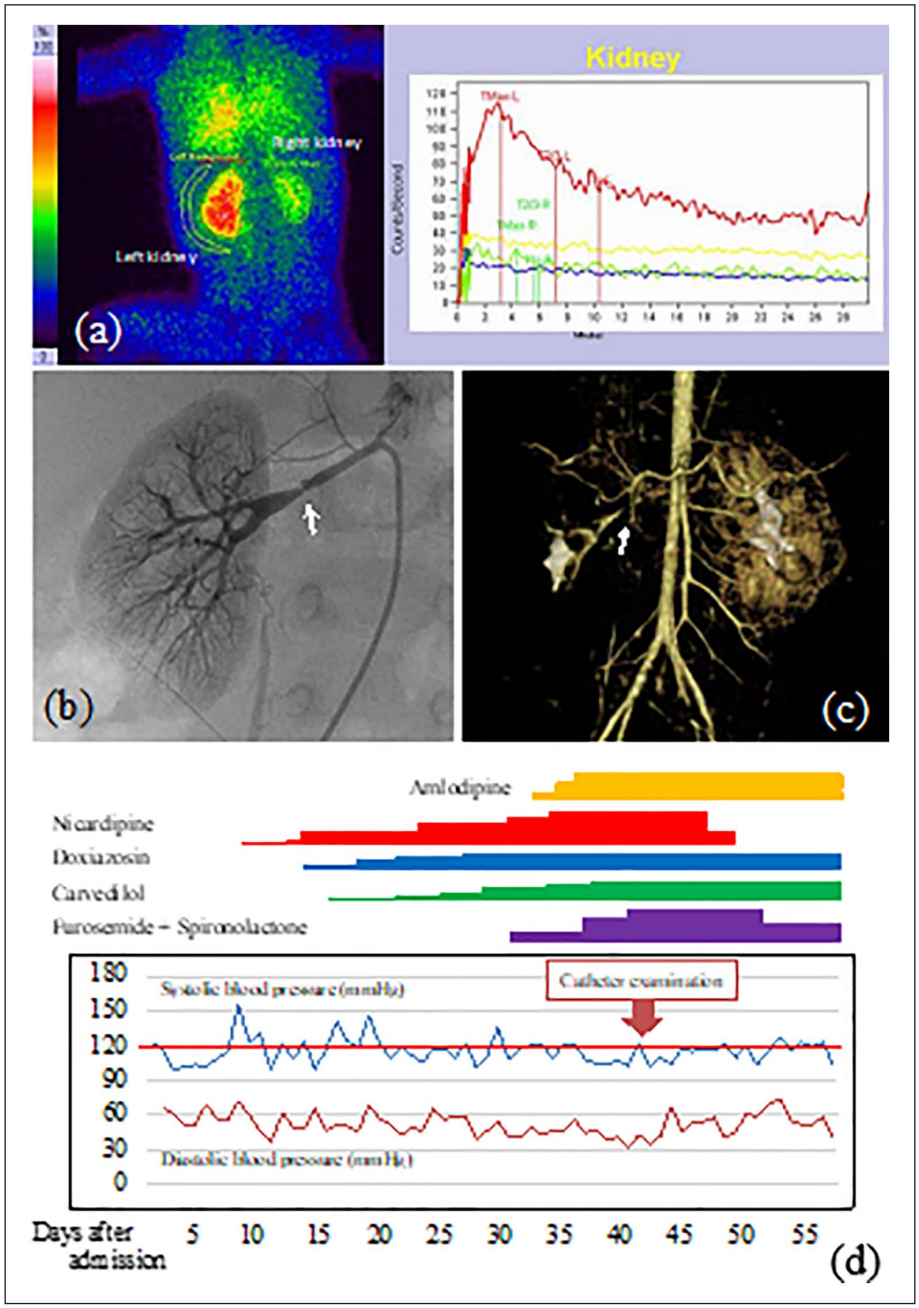


**Figure 1.** The imaging findings of our patient. (a) Diuresis renogram shows a high dysfunction of the right kidney. Red and green lines show function of the left and right kidney, respectively. (b) Three-dimensional computed tomography angiography (3DCTA): right renal artery stenosis. (c) Angiography: the right renal artery showed a stenosis at 2/3 of the site from the aorta to branch (white arrows). (d) The clinical course after admission just before catheter therapy: hypertension appeared at day 9, which lasted after administration of several anti-hypertension drugs. We examined the catheter angiography and found a right renal artery stenosis. He underwent balloon catheterization for the stenosis of the renal artery to another hospital.

suggested chloride-resistant metabolic alkalosis involving increased PRA and hyperaldosteronemia without volume loss. These findings were consistent with HHS, although SIADH was provisionally considered. Symptoms such as vomiting, hypovolemia, and hyponatremia in our patient mimicked gastroenteritis with dehydration, which usually involves metabolic acidosis. However, continuous alkalemia is unlikely in gastroenteritis. Underlying alkalemia induced by HHS might have masked metabolic acidosis. Hence, hyponatremia, hypokalemia, and alkalemia are clues to diagnosing HHS in our patient.

To the best of our knowledge, there are 23 previously reported pediatric patients with HHS under the age of 15 years, excluding causes such as tumor or Takayasu disease (Table 2).[2,4,8–20] Including our patient, the male-to-female ratio was 9:15. The average age of onset was 3 years

**Table 2.** Clinical features of 24 pediatric patients with hyponatremic hypertensive syndrome (HHS).

| Patient | Age | Sex | BP at first visit | The highest BP | Sodium (mmol/L) | Potassium (mmol/L) | pH | Bicarbonate (mmol/L) | Proteinuria | Hematuria | Renal tubular injury | Affected kidney | Treatment | | | | Complications and Prognosis | References |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Systolic/diastolic (mmHg) | Systolic/diastolic (mmHg) | | | | | | | | | Drugs | PTA | Stenting | Surgery | | |
| 1 | 2 years | Female | Not measured | 220/160 | 126 | 3.1 | NA | NA | NA | NA | NA | Left | Labetalol and nitroprusside (div) → Labetalol and nifidepine (po) | | | Nephrectomy | | 10 |
| 2 | 4 years | Female | Not measured | 210/160 | 120 | 3.3 | NA | NA | 1.642 (g/day) | 5–6 (cells/HPF) | NA | Right | Captopril | | | | | 17 |
| 3 | 14 days | Male | 30 (MAP) | >70 (MAP) | 125 | 4.6 | NA | NA | NA | NA | NA | Left | Labetalol hydrochloride | | | Nephrectomy | | 9 |
| 4 | 20 days | Male | 41 (MAP) | 64 (MAP) | 129 | 4.3 | NA | NA | NA | NA | NA | Right | Captopril → Amlodipine and labetalol hydrochloride | | | | | |
| 5 | 1 year | Male | 210/160 | 210/160 | 120 | 2.1 | NA | NA | NA | NA | NA | Left | Labetalol and amlodipine (po) | + | | Surgical correction of triple RAS | | 11 |
| 6 | 2 years | Male | Not mentioned | Not mentioned | 124 | 2.8 | NA | NA | 1.23 (g/day) | NA | NA | Left | Hydralazine and several drugs | | | Reconstration with Goretex patch clamp | | 18 |
| 7 | 2 years | Female | Not mentioned | Not mentioned | 128 | 2.7 | NA | NA | 2.4 (g/day) | NA | NA | Left | Nifedipine, beta blockers and hydralazine | | | Reconstration with Goretex patch clamp | | |
| 8 | 4 years | Male | 220/120 | NA | 123 | 2.8 | NA | NA | 368 (mg/dL) | 3+ | NAG 42 (U/L) | Right | Nicardipine (div) → Nicardipine (div) Enalapril, benidipine hydrochloride and valsartan (po) | + | + | Nephrectomy | | 8 |
| 9 | 4 years | Female | 219/130 | NA | 130 | 3.4 | NA | NA | 18.667 (g/gCr) | 20–29 (cells/HPF) | NAG 18.9 (U/L) | Left | [ Nicardipine (div) Spironolactone (po) → Ca antagonist (po) | | | Nephrectomy | | |
| 10 | 1 year | Female | 190/120 | 190/120 | 122 | 2.4 | 7.45 | 29.5 | 1.8 (g/day) | Negative | NA | Right | Propranolol (po) | + | | | | 2 |
| 11 | 7 years | Male | 210/120 | 210/120 | 114 | 2.4 | NA | NA | Negative | NA | NA | Left | Amlodipine and prazosin | | | Nephrectomy | | 15 |
| 12 | 3 years | Female | 86/42 | 200/140 | 132 | 2.8 | 7.559 | 32.5 | 8.7 (g/gCr) | Negative | β2-MG 675 (ng/L) | Left | [ Nicardipine (div) ACE inhibitor (po) → ACE-I, beta blocker and Ca antagonist (po) | | | | | 20 |
| 13 | 20 days | Male | NA | 104/60 | 101 | 3 | 7.56 | 22.9 | >2.0 (g/dL) | Yes | NA | Right | Dihydralazine (div) | | | | Neurological abnormality | 19 |
| 14 | 2 years | Female | 90/42 | 215/156 | 129 | 3 | NA | 27 | 0.7 (g/day) | NA | NA | Left | Not mentioned | + | | | | 12 |
| 15 | 2 years | Male | 142/92 | 142/92 | 122 | 3.9 | NA | 25.9 | 3.2 (g/day) | NA | NA | Right | Not mentioned | + | | | | |
| 16 | 2 years | Male | 220/150 | 220/150 | 125 | 3.2 | NA | 27.2 | 5.3 (g/day) | NA | NA | Left | Not mentioned | + | | | | |
| 17 | 1 year | Female | 220/140 | 220/140 | 135 | 2.8 | NA | 30 | 2.1 (g/gCr) | NA | NA | Right | Labetalol (div) → Beta blocker, ACE-I and Ca antagonist (po) | | + | | | |
| 18 | 9 years | Male | 156–166/114–123 | 166/120 | 124 | 3.2 | 7.55 | 34 | Negative | Negative | NA | Left | Sodium nitroprusside (div) → Enalapril and amlodipine | | + | | | 4 |
| 19 | 1 year | Male | 218/144 | 248/150 | 128 | 3.2 | NA | 24 | NA | NA | NA | Right | Nicardipine (div) Amlodipine (po) | + | + | | | 16 |
| 20 | 12 days | Male | NA | 86 (MAP) | 122 | 5.6 | 7.176 | NA | 44 (mg/dL) | NA | NA | Right | Nicardipine (div) Amlodipine (po) | | | | | 13 |
| 21 | 5 years | Male | 236/120 | 236/120 | 112 | 3.2 | 7.448 | 33.4 | 6.847 (g/gCr) | Negative | NA | Left | Enalapril, nifedipine and atenolol | + | | | PRES Hypertension[a] Proteinuria | 14 |
| 22 | 8 years | Male | 184/110 | 184/110 | 127 | 3.1 | 7.532 | 31.8 | 3.912 (g/gCr) | Negative | NA | Left | Not mentioned | + | | | PRES | |
| 23 | 12 years | Male | 244/166 | 244/166 | 126 | 3.2 | 7.605 | 32.2 | 4.362 (g/gCr) | Negative | NA | Left | Enalapril | | + | | | |
| Our patient | 1 year | Male | 124/– | 169/94 | 127 | 3.3 | 7.456 | 26.7 | 8.622 (g/gCr) | 2+ | β2-MG 4868 (ng/L) | Right | [ Nicardipine (div) Doxazosin and carvedilol (po) → Amlodipine, doxazosin, carvedilol, furosemide and spironolactone (po) | + | | | | |

ACE: angiotensin-converting enzyme; BP: blood pressure; MAP: mean artery pressure; NA: not available; PTA: percutaneous transluminal angioplasty; RAS: renal artery stenosis; PRES: posterior reversible encephalopathy syndrome; ACE-I: angiotensin-converting enzyme inhibitor; NAG: *N*-acetyl-β-D-glucosaminidase; HPF: high-power field.
[a]Partially improved.

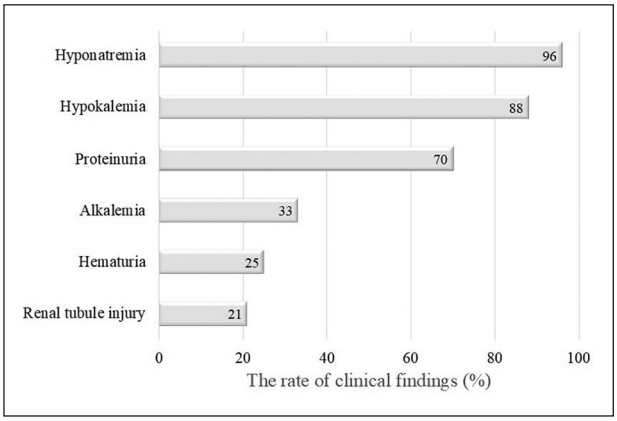


**Figure 2.** The rate of clinical findings of our patient and those in the literature. We defined hyponatremia as serum sodium of <135 mEq/L, whereas hypokalemia was serum potassium of 3.5 mEq/L.

(range: 14 days–12 years). Among these patients, HHS was associated with umbilical arterial catheter insertion in three neonatal patients.[9,13,19] The rates of clinical findings are shown in Figure 2. All patients presented with an electrolyte abnormality, such as hyponatremia or hypokalemia. Alkalemia or alkalosis was indicated in eight patients. Regarding urinalysis, our patient had hematuria, proteinuria, and renal tubular injury. As shown in Table 2, including our patient, proteinuria and hematuria are shown in 17 and 6 patients, respectively. Renal tubular injury was manifested by five patients, including our patient. Hypertension is likely to induce hyperfiltration leading to proteinuria, endothelial injury, and renal tubular injury. Affected kidney was the right in 10 patients and the left in 14. In our patient, it is likely that the right renal artery stenosis led to renovascular hypertension and diuresis of the left kidney. As mentioned previously, HHS patients may have varying clinical features of biochemical values, urinary findings, and/or endocrinological findings.

Our patient transiently showed hypertension on admission. At that time, it was assumed that his crying affected it, because it spontaneously decreased in the next day. Subsequently, he did not show remarkable hypertension. His blood pressure was around 90th percentile until the eighth hospital day, making the diagnosis of HHS difficult. It is unclear that hypertension did not persist in acute phase in present patient, while mydriatic agent used on ninth hospital day to screen ophthalmologic complication probably triggered it. There are only two reports on HHS without remarkable hypertension in acute phase, excluding neonatal patients, in whom HHS was iatrogenically induced.[12,20] All cases showed hypovolemia, weight loss, or hyponatremia, and severe hypertension occurred after fluid replacement. Our patient had fever, vomiting, and weight loss before admission. Acute gastroenteritis and dehydration might have masked his hypertension transiently. After PTA, increased PRA and plasma aldosterone level lasted, whereas proteinuria immediately improved. Hence, doses of antihypertensive drugs were gradually decreased. These findings indicate that he immediately recovered from HHS by PTA, but it took time to improve endocrinological abnormality-associated renovascular disease.

Renal artery hypertension causing HHS is difficult to treat with antihypertensives only. To treat hypertension, 20 patients, including our patient, needed interventions such as PTA, stenting, and/or nephrectomy. Except for one patient, these interventions were successful.

## Conclusion

HHS is curable with proper diagnosis and timely intervention. Therefore, pediatricians should pay attention to the signs and symptoms associated with HHS, such as alkalosis, hyponatremia, and hypertension, even if remarkable hypertension does not persist during initial presentation.

### Authors' note

A preliminary report was presented at the 645th Japan Pediatric Society Tokyo Chapter, 2018 and the 53rd Annual Meeting of the Japanese Society for Pediatric Nephrology, 2018.

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### Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

### Ethical approval

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### Informed consent

Written informed consent was obtained from a legally authorized representative for anonymized patient information to be published in this article.

### ORCID iD

Nodoka Hinokuma https://orcid.org/0000-0002-4658-529X

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