CASE REPORT

Management of a pregnant woman with hypouricemia: a case report

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Abstract
Renal hypouricemia is associated with urinary calculi and severe acute renal failure after exercise. The epidemiology of renal hypouricemia is not yet sufficiently understood, and there is no report of it occurring during pregnancy. We report the case of a pregnant woman with renal hypouricemia. At her first pregnancy, she developed preeclampsia at the 34th week of gestation. After parturition, she developed acute renal failure and was diagnosed with renal hypouricemia. During the second pregnancy, when she was referred to our hospital, care was taken to ensure adequate hydration by infusion of liquids and water at the time of labour. Consequently, she did not have onset of renal hypouricemia. We suggest that acute renal failure may be avoided in pregnant women with renal hypouricemia by preventing dehydration via drinking enough during pregnancy postpartum period and by infusion during labour.

INTRODUCTION
Renal hypouricemia is characterized by impaired uric acid reabsorption at the apical membrane of proximal renal tubular cells. Generally, patients may be asymptomatic. However, it is accompanied by nephrolithiasis and exercise-induced acute renal failure in about 10% of patients [1]. The typical symptoms of acute renal failure are sudden lower back pain, nausea and vomiting for several hours after exercise. Patients with acute renal failure often have improved renal function in 2–4 weeks, but dialysis may be required in certain cases [1]. Acute renal failure recurs in about 20% of the patients. Symptoms of urolithiasis include back pain and haematuria. Renal hypouricemia is classified into type 1, type 2 and others, based on causative genes [2, 3].

There are several reports on the association between high uric acid level and hypertensive disorder of pregnancy. However, papers on the effects of low uric acid levels are few even in non-pregnancy cases, and epidemiological data are also limited. When the cut-off of hypouricemia is set at 2.0 mg/dL, the incidence is very low at around 0.2% [4]. Herein, we present the case of a pregnant woman who had preeclampsia at the first pregnancy. After parturition, she developed acute renal failure and was diagnosed with renal hypouricemia. Her second pregnancy was managed carefully at our hospital.
A 35-year-old G1P0 woman was diagnosed with preeclampsia with severe features at 34 weeks of gestation and delivered a 1900 g infant. Her renal function worsened after parturition (Cre: 0.78mg/dL, eGFR: 68.4 ml/min), and she was diagnosed with renal hypouricemia, with 2.0 mg/dL of serum uric acid and elevated clearance of uric acid (UA) (17.0 ml/min) along with the exclusion of other disease in which uric acid excretion is accelerated, such as Fanconi syndrome, syndrome of inappropriate secretion of antidiuretic hormone (SIADH), DM, etc. Serum uric acid of her child was also below 2.0 mg/dL. However, she did not desire genetic testing for renal hypouricemia. At her second pregnancy, she was referred to our hospital at 19 weeks of gestation because of her previous pregnancy history. As renal hypouricemia causes acute renal failure due to severe exercise and dehydration, we advised her to drink sufficient water and avoid activities that cause fatigue. We advised her to urinate 5 times/day, excreting >1 litre of urine. We instructed her to measure home blood pressure so that she could respond early to the onset of preeclampsia.

Blood pressure during pregnancy was around 110/60 mmHg. The blood urea nitrogen (BUN)/creatinine (Cre) ratio was not affected during pregnancy and postpartum (Table 1). At the first visit, clinical measurements were taken after she had adequate water. Thereafter, dehydration did not worsen, as estimated from the serum BUN/creatinine ratio.

At 38 weeks and 4 days' gestation, rupture of membranes occurred, and delivery was uneventful (3240 g female infant, with Apgars score 8/9). We had considered administering epidural anaesthesia to reduce the stress during labour but could not implement it because of rapid delivery that happened in about one-and-a-half hours from onset of the active phase.

Her blood pressure during delivery was on the order of 120/70 mmHg, with an upper limit of 138/88 mmHg in the second term of labour. She was given sufficient infusions during labour. Considering dehydration via lactation after parturition, drinking of water after delivery was also encouraged and she was advised to urinate about five times/day. She was well conditioned and was discharged on the fourth day after delivery. Laboratory results at the outpatient clinic did not reveal abnormal findings, including kidney function parameters, 11 days after parturition.

### DISCUSSION

Herein, we report the case of a pregnant woman with renal hypouricemia and previous history of preeclampsia. Renal hypouricemia is diagnosed as serum urate level less than 2.0 mg/dL along with increased fractional excretion of urate (normal range: 5.5–11.1%) and elevated clearance of UA(normal range: 7.3–14.7%). In addition, other diseases were excluded [5].

Her second pregnancy was managed without recurrence of acute renal failure, which she had experienced during her first pregnancy. Management of pregnant women with hypouricemia is required to ensure that intense exercise, especially anaerobic exercise, and dehydration are avoided. A severe complication of renal hypouricemia is acute renal failure, and adequate hydration may prevent the onset of acute renal failure. In addition, deterioration of kidney function during pregnancy is one of the risk factors for preeclampsia.

Labour usually is accompanied by anoxic breathing. As she was multiparous, there was lesser bearing down effort. With regard to exercise stress, painless delivery was also considered via epidural anaesthesia; however, delivery often takes prolonged time with epidural anaesthesia. If labour and uterine contractions are stressful, a shorter delivery time may be more desirable. In the current instance, she was paragravida with a short delivery time and with less stress, and these factors may have led to the prevention of the onset of acute renal failure.

In her previous pregnancy, there was a complication of preeclampsia, leading to intravascular dehydration, and the stress of delivery burdened her. In addition, dehydration due to lactation after birth also impacted the risk of acute renal failure.

In her second pregnancy, it was considered that stress to the kidneys can be reduced by appropriately infusing fluids during delivery and thereby avoiding dehydration. Consequently, it can be said that in this instance, dehydration was successfully prevented and delivery was appropriately managed based on the symptoms and the BUN/creatinine data (Table 1).

Since there is a high risk of dehydration due to lactation after delivery, we encouraged her to drink enough fluids after delivery. We instructed her to drink enough fluids such that the frequency of urination was five or more times/day, for an estimated urine volume of >1 l/day.

Considering that dehydration is a risk factor for urolithiasis and acute renal failure, early stage of pregnancy with morning sickness and emesis may also be a period of high risk for complication of urolithiasis and acute renal failure. Although her first visit to our hospital was at 19 weeks of gestation, she did not have hypouricemic complications. Management with infusions is necessary so that pregnant women diagnosed with renal hypouricemia do not become dehydrated due to hyperemesis.

Although there are no reports on the association between pregnancy and hypouricemia, encouraging the patient to drink water and liquids enabled safe management of delivery, without causing acute renal failure. Moreover, it may also be suggested that prevention of preeclampsia by aspirin, can prevent intravascular dehydration and prevent the onset of acute renal failure [6].

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### CONFLICT OF INTEREST

All of the members have no conflict of interest.

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### ETHICAL APPROVAL

No approval is required.
CONSENT
The patient agreed in writing that we report her case. Consent is held in our treating institution according to locally approved procedures.

GUARANTOR
Keiichi Kumasawa

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