Case report

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작은 요로 결석에 의한 급성 신후성 신부전 및 폐쇄 후 이뇨 1례

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정의석 · 양은미 · 김찬종

Acute Kidney Injury and Postobstructive Diuresis Caused by a 4 mm Urinary Calculus

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Urinary obstructions from ureteral calculi are one of the causes of postrenal acute kidney injury (AKI). Here we present a case of AKI caused by a 4 mm ureteral calculus with postobstructive diuresis following the spontaneous passage of the calculus. A 13-year-old girl who underwent nephrectomy for the removal of a neuroblastoma eight years previously, visited our institution because anuria had developed over the preceding five days. The serum creatinine level was elevated at 13.4 mg/dL. Radiological examinations showed the right solitary kidney with moderate hydronephrosis and a 4 mm calculus in the upper right ureter. The patient immediately underwent hemodialysis. After the ureteral calculus was passed spontaneously on day 2 of hospitalization, urinary output increased to more than 5,200 mL per day. Intravenous fluid replacement with careful monitoring of weight, intake, output, and serum and urine electrolytes was performed. On day 5 of hospitalization, the patient's condition stabilized.

Key words: Calculi, Obstruction, Acute kidney injury, Polyuria, Anuria

Introduction

Postrenal acute kidney injury (AKI) occurs when there is bilateral (or unilateral in the case of a solitary kidney) obstruction of the urinary tract. However, obstruction of the urinary tract is a relatively uncommon cause of AKI. Several studies have reported the incidence of postrenal AKI at 5 % to 10% of all cases of AKI [1, 2]. The causes of postrenal AKI include tumors, urinary calculi, clots, or fungal balls. Ureteral calculi are one of the most common reasons for postrenal AKI [3]. The risk factors for developing AKI in ureterolithiasis patients are bigger stones, ureteral stones in patients with only one functioning kidney or pre-existing kidney

disease, and bilateral ureteral stones [4]. Postobstructive diuresis (POD) is defined as a syndrome in which a potentially life-threatening polyuria and inappropriate excretion of electrolytes follows the release of an obstruction in the urinary tract. We experienced a case of AKI and POD caused by a 4 mm ureteral stone in a 13-year-old girl with a solitary kidney. In a brief discussion of the literature, we describe the clinical features and methods for management of AKI and POD caused by a tiny urinary calculus.

Case report

A 13-year-old girl was admitted to our hospital presenting with general weakness, headache, anuria, and edema. She was diagnosed with a neuroblastoma eight years ago in the left adrenal gland and then treated with chemotherapy, radiotherapy, and left radical nephrectomy. The patient noted that oliguria had started seven days ago and anuria had developed five days ago. A physical examination revealed blood pressure of 160/100 mmHg and peripheral edema. The initial laboratory evaluation disclosed the following: serum creatinine 13.4 mg/dL, blood urea nitrogen 138.3 mg/dL, uric acid 16.5 mg/dL, glucose 142 mg/dL, sodium 136 mEq/l, potassium 6.0 mEq/l, chloride 93 mEq/L, phosphorus 6.3 mg/dL, calcium 8.4 mg/dL, and serum osmolality 329 mOsm/kg. Glomerular filtration rate measured by the Cockroft-Gault formula was 6 ml/min/1.73m^2. The patient was anuric, and had no urine flow after the insertion of a urinary catheter into the bladder. An ultrasonography and computed tomography showed moderate hydronephrosis (SFU grade 3) in the right solitary kidney and a 4 mm calculus in the right upper ureter (Fig. 1A, 1B). A computed tomographic (CT) scan was performed three months ago for follow-up of neuroblastoma but showed no hydronephrosis and no visible urinary calculi.

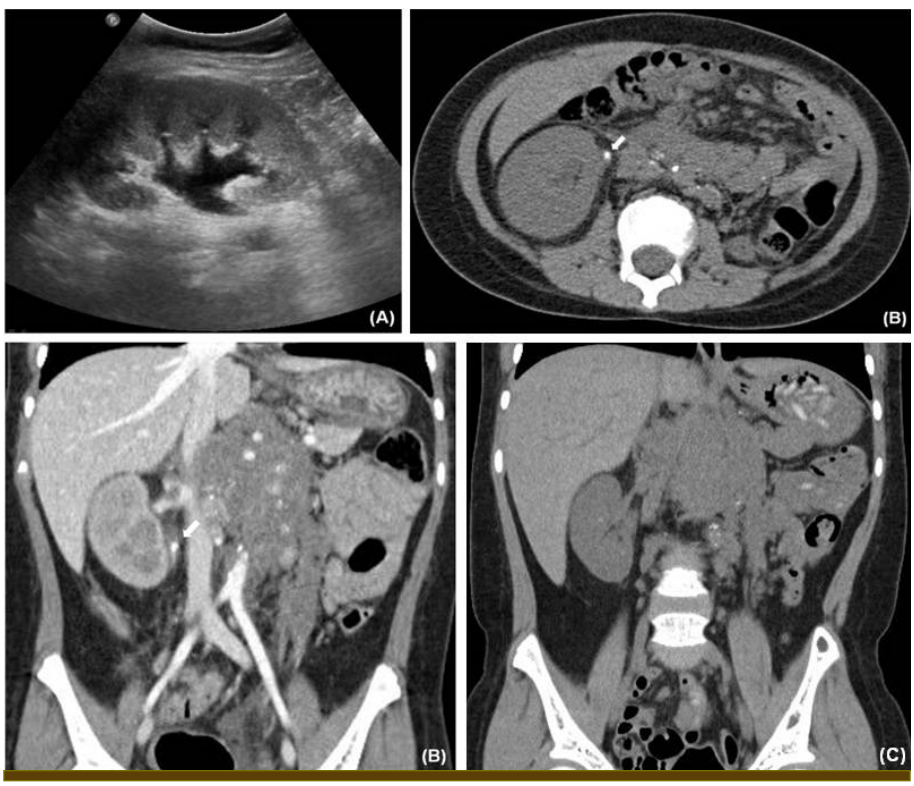

Fig. 1. Radiologic examinations. (A) Ultrasonography on the first day in hospital: moderate hydronephrosis in right kidney. (B) Computed tomographic scan on the first day in hospital: right solitary kidney with moderate hydronephrosis and a 4 mm calculus (white arrow) in upper right ureter (C) Computed tomographic scan on the sixth day in hospital: marked regression of the hydronephrosis, and no visible ureteral calculi ipsilaterally.

The patient received antihypertensive medication and continuous renal replacement therapy (CRRT) immediately. On the second day in hospital, we planned to perform a percutaneous nephrostomy for urinary tract obstruction. However, the patient unexpectedly presented with gross hematuria, and the urinary output had increased abruptly. The patient did not complain irritative urinary symptoms such as dysuria. Although this situation suggested the spontaneous passage of a ureteral calculus, we could not find it. Urinalysis showed trace protein, 5 to 9 red cells and 10 to 19 white cells per high-power field, and negative dysmorphic RBC. The 24hr urine was analysed spectrophotometrically for oxalate, calcium, magnesium, citrate, uric acid, phosphate and creatinine. A 24-hour urine collection contained 64.6 mg/day of oxalate which is considered as hyperoxaluria. The urinary output, which was 4,500 mL on the second day in hospital, increased progressively and reached a peak of 5,200 mL on the third day in hospital. As the patient's urinary output had increased remarkably, her blood pressure was reduced to normal levels, and there appeared to be no need for a replacement therapy. It was then decided to stop the CRRT on the second day in hospital.

To replace the fluid loss caused by polyuria, alkaline isotonic saline was administered through a large-bore intravenous access. During diuresis phase, we checked urine output hourly. The urine output of the past hour was replaced intravenously with 0.9% saline and 10% dextrose in 0.3% NaCl solution. After 48 hours of persistent diuresis, total fluid volume was reduced to 70-80% of the infusion rate before 4 hours depending on urine output and clinical situations (Table 1) (Fig. 2). The patient was hemodynamically stable, had no signs of hypovolemia and electrolyte imbalance. The urinary output, which was at its maximum on the third day, had declined gradually during the following week. A serum creatinine level had decreased from 13.4 mg/dL to 1.3 mg/

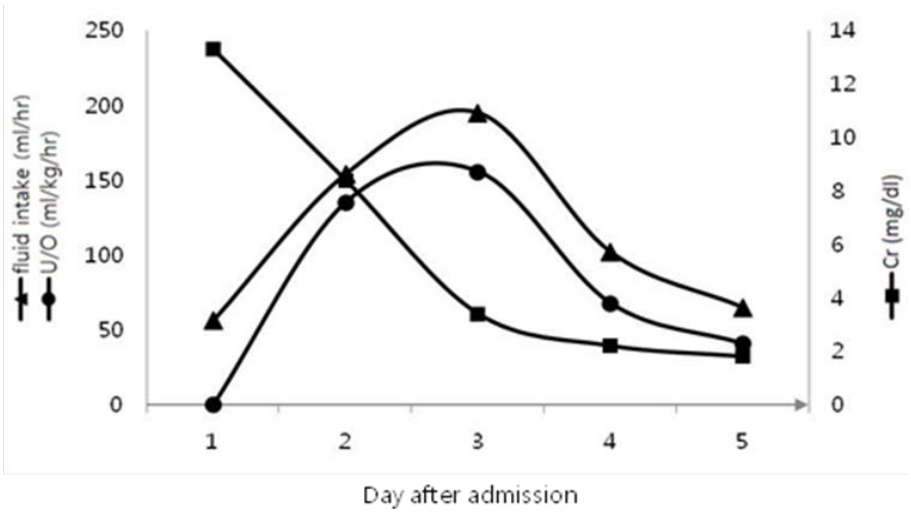

Fig. 2. Urinary output, fluid intake, and serum creatinine levels (Vertical axis) during hospitalization (Horizontal axis).

Table 1. Daily fluid, Fluid Volume, Urine Output and Creatinine

	Hospital Day 1	Hospital Day 2	Hospital Day 3	Hospital Day 4	Hospital Day 5
Input					
Fluid volume (ml/day)					
10% D/W in 0.3% NaCl	600	2,200	3,600	1,700	500
Normal saline	0	1,200	1,000	0	0
Oral feeding (ml/day)	0	300	300	250	800
Urine output (ml/day)	0	4,480	5,170	2,550	1,360
Creatinine (mg/dL)	13.3	8.3	3.4	2.2	1.8

dL within the week. At discharge, serum Creatinine level was 1.3 mg/dL with a glomerular filtration rate of 35 ml/min/1.73m^2 (Fig. 2). A follow-up CT scan was performed on the sixth day in hospital and showed a marked regression of the hydronephrosis and no visible ureteral calculi ipsilaterally (Fig. 1C). The patient was discharged on the ninth day in hospital. At follow-up after three months, the serum creatinine level was normal, and a follow-up CT scan showed no hydronephrosis in the right kidney.

Discussion

The majority of studies of pediatric stone series with data on the etiology are either limited or have incomplete data, which may be explained by the lower prevalence of pediatric urolithiasis. Its prevalence has been reported to be more common in western countries and parts of Asia than in East and West Africa [5]. The overall incidence of pediatric urolithiasis is estimated at 2 to 3% of the general population and is increasing [5-7].

However, urolithiasis in children can cause considerable morbidity, such as urinary tract infection, sepsis, and obstructive uropathies. In particular, postrenal AKI caused by an obstruction in the urinary tract requires emergency treatment. Wang et al. [4] reported that the risk factors for developing AKI in adults with ureterolithiasis were bigger stones, ureteral stones in patients with only one functioning kidney or pre-existing kidney disease, and bilateral ureteral stones. The mean size of a single ureteral calculus with AKI in adults was 1.35±0.38 cm. On the other hand, Coll et al. [8] reported the relation of the spontaneous passage of ureteral calculi to stone size in adults. They found that stones measuring 4 mm or smaller usually pass spontaneously (frequency of spontaneous passage=78%). Stones measuring 5-7 mm frequently passed spontaneously (frequency of spontaneous passage=60%); and stones measuring 8 mm or larger usually do not pass spontaneously (frequency of spontaneous passage=39%). In our case, a ureteral calculus that induced life-threatening AKI and POD was only 4 mm.

The early diagnosis and appropriate treatment are important in preserving kidney function. A high index of suspicion is necessary for an early diagnosis. The decrease in urinary output may be a clue for post-renal AKI [9]. The gold standard method for the detection of AKI is inulin clearance or nuclear isotope clearance [10], but this method is not convenient in clinical practice. Although measuring the serum creatinine level is a practical approach for discovering short-term alteration in renal function, the level of serum creatinine may be affected by several factors, such as age, sex, race, muscle mass, and medications [11, 12]. In patients with suspected postrenal AKI, radiological examinations to evaluate hydronephrosis and causes of the obstruction should be performed. Ultrasonography or non-enhanced computed tomography is available to find out causes of postrenal AKI such as a urinary stone.

The pathogenesis of POD is not clear. POD has been the subject of numerous pathophysiological studies [13-18]. Physiological factors developing potentially POD include (a) excess sodium and water retention; (b) accumulation of urea and other non-reabsorbable solutes resulting in an osmotic diuresis. Pathological factors may be associated with (a) decreased tubular reabsorption of sodium secondary to altered expression of proximal and distal sodium transporters; (b) inability to concentrate urine maximally, secondary to a decreased medullary concentrating gradient, leading to decreased response to ADH; (c) increased tubular transit flow time, reducing the equilibration time for absorption of sodium and water; (d) increased production of prostaglandins immediately following the release of the obstruction.

Patients who develop POD need meticulous management and close monitoring. To replace the fluid loss caused by polyuria, treatments should ensure that the replacement volume does not exceed two-thirds of the daily urinary output to avoid iatrogenic extracellular volume expansion. The intravenous fluid replacement with careful monitoring of weight, intake, output, and serum and urine electrolytes are necessary until the situation stabilizes. To avoid hypothermia, fluids should be warmed to body temperature before infusion [19].

Urinary output usually returns to normal when extracellular volume and composition return to normal. This condition is usually self-limiting and resolves over several days to a week. The recovery of renal function after the release of an obstruction depends on the duration and degree of the obstruction, whether the obstruction is unilateral or bilateral, and whether there is a concomitant infection [20].

In conclusion, postrenal AKI could be caused by a tiny urinary calculus in patients with a solitary kidney. The release of a urinary obstruction caused by a tiny urinary calculus can result in POD, which needs careful monitoring and the appropriate replacement of fluid and electrolytes, as in the case of our patient.

한글요약

결석으로 인한 요관 폐쇄는 신후성 신부전의 주요한 원인으로 즉각적인 치료가 필요하다. 폐색 후 이뇨는 폐쇄성 요로 질환의 막힘 제거될 때 흔히 나타날 수 있는 증상으로 특별한 치료 없이 회복되는 경우가 많으나 저혈압이나 전해질 이상 등의 소견이 나타날 경우에는 수액 요법을 통한 치료가 필요하다. 단일신 환아에서 4 mm 크기의 작은 결석으로 인한 신후성 신부전이 발생하였으며 결석이 배출되고 발생한 폐쇄 후 이뇨는 보존적 치료로 회복되었다. 대부분의 4 mm 미만의 작은 결석은 저절로 배출 된다고 알려져 있으나 저자들은 4 mm 크기의 결석으로 생긴 신후성 신부전 및 폐쇄 후 이뇨가 발생한 예를 경험하였기에 보고하는 바이다.

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