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Massive Post-Obstructive Diuresis

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Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Male, 8
Final Diagnosis: Post obstructive diuresis
Symptoms: Nausea • vomiting
Medication: —
Clinical Procedure: Surgical release of obstructed ureter
Specialty: Nephrology

Objective: Unusual clinical course





Background: The purpose of presenting this case is to demonstrate the degree to which the kidney is capable of selectively excreting a massive load of sodium and water when challenged with both of these, without altering the plasma levels of other ions.

Case Report: An 8-year-old boy was admitted in severe renal failure. Workup demonstrated a high grade obstruction of a single kidney. Following dialysis, the patient underwent surgery to correct the obstruction and he developed post-obstructive diuresis. Within one week he was receiving 34 liters of essentially 1/2 Na by IV and by mouth and was excreting 70% of his filtered load of water and 50% of his filtered load of sodium. As soon as the administered fluids and Na were cut back, the kidney responded appropriately.

Conclusions: While post-obstructive diuresis is a real phenomenon, very frequently it is magnified by forcing diuresis with the administration of too much water. These patients are best treated by administering fluids to equal output for two to three days and then gradually cutting back on fluid intake. If the kidney responds appropriately, then fluids can be given as the patient requests.

MeSH Keywords: Acute Kidney Injury • Natriuresis • Nephrology

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/906238>

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Background

The kidney is a unique organ capable of facing multiple challenges while maintaining perfect chemical balance. Bricker et al. [1], using various models of chronic renal disease, have demonstrated the ability of the impaired kidney to handle large loads of sodium, potassium, and water, as well as other electrolytes (the magnification phenomenon). As each nephron is lost, each remaining nephron must increase its excretion of sodium and water. In various models of chronic renal failure in animals, each remaining functioning nephron has been shown to excrete as much as 32% of its filtered load of sodium and water. In humans, previous cases of post-obstructive diuresis have been reported with urine outputs of 58.0 liters and 88.4 liters per day in adults, but there were no reported measurements of glomerular filtration rates or urine sodium in the reported cases [2,3].

Some years ago, a young child with massive diuresis following relief of ureteral obstruction of a single kidney was treated at Loma Linda University Medical Center. The sodium loss by this single kidney far exceeded previously reported maximum excretion rates.

Case Report

The patient was an 8-year-old boy who was well until six weeks prior to admission when he was seen by his physician for vague abdominal pain which was not investigated. Two weeks prior to admission, he became tired and irritable. One week prior to admission, he complained of a headache and was noted to have puffiness of the face, and periorbital edema. Three days prior to admission, he developed nausea and vomiting resulting in his hospital admission. There had been no urinary complaints and his urine volume was reportedly normal. His past history and family history were noncontributory.

On physical examination, he was noted to be well nourished and well developed. He was lethargic, but in no acute distress. Blood pressure (BP) was 150/100 mm Hg, pulse 100 bpm, and respirations were normal. The skin was a pale brown color. A 10 cm by 15 cm mass was noted in the right upper quadrant of the abdomen and he had 1+ edema of his ankles. The remainder of the physical was normal.

His initial laboratory data were as follows: hemoglobin 9.0 g/dL, serum sodium 128 mEq/L, serum potassium 8.3 mEq/L, serum creatinine 13.8 mg/dL, and blood sugar 70 mg/dL. An intravenous pyelogram showed no renal excretion on x-ray contrast.

The patient was transferred to Loma Linda University Medical Center and immediately started on peritoneal dialysis with an

excellent response. Following peritoneal dialysis, a retrograde pyelogram demonstrated a right ureteropelvic junction obstruction with massive hydronephrosis, and agenesis of the left kidney and ureter. He was taken to surgery the same day and underwent a pyeloureteroplasty and nephrostomy. Following surgery, the patient began passing large volumes of urine consistent with post-obstructive diuresis. Over the next seven days (June 17 to June 23) he was given increasing amounts of 5% dextrose in 1/2 normal saline or in normal saline. Fluid intakes progressively increased from 11 liters to 33 liters per day, while urinary output increased from 12 liters to 34 liters per day. Edema of his legs persisted and his blood pressure remained elevated at 160/100 mm Hg. Therefore, because of fluid overload and a progressive reduction of the BUN to 17 mg/dL and reduction of the serum creatinine to 1.1 mg/dL, his fluid intake was progressively reduced resulting in progressive reduction in urinary output to 2 liters per day over the next six days (Table 1). His blood pressure was normalized at 120/80 mm Hg and he was edema free.

At the peak of his diuresis (on June 23), the 8-year-old boy with one kidney was excreting over 34 liters of fluid and over 3,074 mEq of sodium per day. This amounted to a phenomenal 71.9% of the filtered load of water and 50% of the filtered load of sodium.

Discussion

The normal kidney contributes in a remarkable fashion to body fluid homeostasis. Human intake of fluid and solute varies from day to day. For example, on one day the intake of salt may be 6 grams, 10 grams the next day, and 4 grams the following day. At the end of each 24 hours, sodium balance is restored. The same is true for other solutes. Free water intake can vary from 1 liter per day to 16–20 liters per day, and again water balance is restored at the end of each 24-hour cycle.

The control of sodium secretion by the kidney is multifactorial and includes the effective arterial volume [4], which in turn effects renal blood flow, glomerular filtration rate (GFR), angiotensin [5] and aldosterone [5,6], peritubular flow and oncotic pressure [4], the release of vasopressin [7,8] and natriuretic hormone [9], action of the sympathetic nervous system [10,11], increases in blood pressure (pressure natriuresis) [12], atrial natriuretic peptides [13], and the action of the prostaglandins [14]. Water excretion is dependent primarily on the presence or absence of vasopressin and the solute load.

It is well known that as the nephron population diminishes in chronic renal disease. Assuming a relatively constant intake of sodium and water, sodium and water excretion per nephron must double with every 50% loss of nephrons [15,16].

Table 1. Serum urine.

Date	Na mEq/L	K mEq/L	Cl mEq/L	CO2 mEq/L	BUN mg/dL	Creatinine mg/dL	GFRmL/ min	Na mEq/L	K mEq/L	Creatinine mg/dL	I	O
6/13	128	8.3	104	25	165	13.8		60		44	?	180
6/14	Dialysis											
	131	4.8	105	15	138	13.8						
6/15	140	4.3	105	21	100	12.9		93	12	24		
6/16	Surgery											
	129	4.2	91	18	67	7.5		91	26			
6/17	132	4.3	102	15	70	4.2		61	18			
6/18	126	4.3	105	15	33	2.2		72	13			
6/19	133	3.9	104	16				63	9			
6/20	133	4.6	108	18	20	1.4	32.75			660 mg	11060	11940
6/21	128	4.8	129					88	14		14345	14645
6/22	124	6.0	104		17	1.1		85	10		22505	23205
6/23	130	5.5						90	6		32940	34155
6/24	120	5.6	100	18				80	6		23065	25855
6/25	128	5.1	105					77	4		14855	20800
6/26	130	5.7									8465	9945
6/27	138	6.4	103	24							6000	7380
6/28	133	5.5									2700	2685
6/29	139	5.3		29	29	1.0					2385	2130
6/30	135	5.0					33.3			500 mg	1310	1380

The normal kidney excretes about 0.5% of the filtered load of both sodium and water. Both increase to 1% of the filtered load when 50% of the nephrons are lost. When 95% of the nephrons are lost and the GFR is 10 mL/minute each nephron must excrete 10% of its filtered load of both sodium and water (see Table 1). When patients develop severe chronic renal failure with a GFR of 5 mL/minute, they must excrete 20% of the filtered load of both sodium and water in order to maintain sodium and water balance on a normal intake. Thus, for each mEq of sodium ingested, residual nephrons must excrete 20 to 40 times as much sodium as renal failure progresses on the same sodium intake. When progressive renal failure develops, the kidney may be capable of excreting up to as much as 30% of its filtered load of sodium and water but it begins to lose its capacity to excrete sodium and water when the GFR drops below 4–5 mL/minute and patients must have their sodium and water intake restricted [1]. As can be seen in Table 1, our patient not only far exceeded the levels of sodium excreted in far advanced renal failure, but was able to do it at a GFR of 33.3 mL/minute when the usual sodium excretion rate should have been about 1–2% of the filtered load.

Most of the research evaluating the kidneys' capability to excrete large loads of sodium and water has been done in animals by decreasing the nephron population and measuring their ability to excrete a sodium and water load. To do this type of research in humans would be unethical. Therefore, this case is of particular interest because it occurred in a patient during the course of treatment for severe post-obstructive diuresis.

In order for this patient's kidney to excrete 50% of the filtered load of sodium and 72% of the filtered load of water, almost all segments of the nephron must have been involved. The patient was obviously fluid overloaded with the development of hypertension and edema, which cleared when the fluid and sodium intake were progressively curtailed. This increase in the effective arterial volume should cause all of the following events to occur: suppression of vasopressin, renin, angiotensin and aldosterone, and probably also suppression of the sympathetic nervous system and endothelin. There would be an increased release of natriuretic peptides and natriuretic hormone and possibly prostaglandins accompanied by an increase in GFR and renal blood flow and a decrease in the oncotic pressure in the post-glomerular vasa recta. Due to the high rate of urine flow there would also be a marked

washout of the osmolality of the interstitium. All of these factors worked together to produce a massive natriuresis and diuresis. With a gradual reduction in the patient's sodium and water intake over a one-week period his BP returned to normal, the edema cleared and he returned to normal levels of sodium and water excretion.

Of significant interest is the apparent lack of tubuloglomerular feedback (TGF) [17,18]. All urine specimens had a sodium concentration between 60 and 93 mEq/L. Theoretically, this level of sodium and chloride concentration passing the macula densa should produce afferent renal artery constriction with a decrease in GFR and an increased Na reabsorption in the proximal convoluted tubule. Obviously this did not occur and in this case it would have been very inappropriate. Therefore, under states of sodium and water expansion, TGF appears to be impaired. It has been postulated that the lack of TGF is due to the high flow rate through the glomerulus producing minimal or no concentration of the serum albumin at the efferent arteriole, and therefore, a very low oncotic pressure in the peritubular capillaries. This in turn would decrease the reabsorption of Na and water in the nephron [19].

This patient case demonstrated the remarkable ability of the kidney to compensate for over vigorous fluid and sodium replacement in the setting of post-obstructive diuresis.

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Conclusions

This is a case report of an 8-year-old patient with post-obstructive diuresis that was markedly accentuated by giving the patient more sodium and fluids than he was excreting. This forced his impaired kidney to excrete 70% of the filtered load of water and 50% of the filtered load of sodium in order to maintain salt and water balance. When treating a diuretic state it is necessary every two to three days to cut back on fluid administration by 500-600 mL below the urine output for one to two days. If the kidney follows, and cuts back its urine output, the patient is past the diuretic phase and can manage his or her own fluids. If the kidney continues to diuresis more than is administered, then the fluids should be increased to intake equal output.

This case demonstrates the ability of the kidney to balance massive loads of sodium and water when it is challenged.

Conflict of interest

None.

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