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Case Report and Review of the Literature

Polyuria after Renal Transplantation: A Case Report and Review of Literature

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ABSTRACT

Polyuria is defined as a urine output (UOP) of more than 3 litres per day in adults or 2 l/m²/day in children. Polyuria is common following live donor kidney transplantation (LDKT). This case report and review describes a 32-year-old male with chronic kidney disease who underwent LDKT. The donor was his brother. He had polyuria in the postoperative period with the maximum urine flow rate of 3700 ml/hr and the first 24-hour urine output of 42 litres. He was managed with intravenous crystalloid solutions guided by the central venous pressure and the mean arterial pressure. Electrolytes were replaced with potassium chloride, calcium gluconate and magnesium sulfate. He made an uneventful recovery. The Polyuria improved without any pharmacological interventions. Therefore, guided fluid and electrolyte administration is the key to the successful management of post-transplant polyuria.

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Introduction

Polyuria is defined as a urine output (UOP) of more than 3 litres per day in adults or 2 l/m²/day in children [1]. Polyuria is common following live donor kidney transplantation (LDKT). Studies have suggested that early post-transplant polyuria is associated with good short-term and long-term renal transplantation outcome [2, 3]. In earlier studies early mean UOP documented following LDKT were 10.06 +/- 5.89, 5.45 +/- 3.05 l at first 24 and 48 hours [4]. In the early post LDKT, polyuria indicates good perfusion and no vascular and ureteric anastomosis complications. However excessive urine output may result in fluid and electrolyte imbalances [2, 3].

Physiology

Both Kidneys receive 25% of cardiac output (1.2 - 1.3L/min in a 70-kg adult male). Urine production begins by plasma getting filtered at the glomerulus (glomerular filtration). Usually about 125ml of plasma is filtered per minute (Glomerular Filtration Rate - GFR). Therefore, in a day about 180 l of Plasma is filtered. Most of this filtered fluid is reabsorbed. Therefore, a normal person passes only about 2000ml to 2500ml of urine a day (urine production rate of 1ml/min). This means only about 1.1-1.4% of GFR volume is passed as urine under normal circumstances. But under abnormal circumstances this can increase up

to 23 l per day and the patient passes diluted urine [5]. The Possible causes of post KT polyuria include rapid increase in the GFR in a kidney with abnormally functioning nephrons soon after reperfusion. Some other mechanisms are over administration of intravenous fluids and new onset hyperglycaemia after transplantation [6]. The Renin-Angiotensin-Aldosterone System (RAAS) is the main regulator of GFR, and it regulates this by modifying the afferent and efferent arteriolar resistance. Prolonged ischaemic time results in RAAS activation resulting in reduced urine output and fluid overload results in suppression of RAAS activity resulting in polyuria [7]. Post-transplant polyuria can result in electrolyte and acid base imbalances like hyponatremia, hypokalemia, hypocalcaemia, hypomagnesemia, hypophosphatemia and metabolic acidosis [8]. Post-transplant polyuria usually resolves without major interventions.

Case Summary

A 32-year-old male with chronic kidney disease (CKD) stage V, secondary to membranous glomerulonephritis underwent LDKT on 4th December 2020. The donor was his brother. He was 27-year-old with a dry weight of 64kg and body mass index (BMI) of 23.79. Recipient's pre transplantation native UOP was about 300ml/Day. His baseline preoperative serum creatinine was 10.4mg/dl, potassium was 5.4mmol/l and Haemoglobin was 8.2g/dl. He received Basiliximab, Tacrolimus,

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Mycophenolate Mofetil (MMF) and methylprednisolone as induction immunosuppressive agents. Transplant procedure was uncomplicated. An end to side anastomosis of the donor renal vessels to the recipient external iliac vessels was done. The total ischaemia time was 92 minutes. There was immediate urine production. An ureteroneocystostomy was performed.

His post-operative UOP was as following; 42 l on D1 with a maximum rate of 3700ml/hr. UOP was 24 l on D2, 18 l on D3, and 5 l on D7. His creatinine improved rapidly to 1.5mg/dl (0.73-1.18mg/dl) by post-transplantation D1, 0.98mg/dl on D2 and 0.9mg/dl on D7. His serum potassium was 3.5mmol/l (3.5-5.1mmol/l) on D1, 3.8mmol/l on D2 and 4.5 mmol/l on D5. Serum magnesium was 1.84mg/dl (1.58-2.55mg/dl) on D1 and 1.1mg/dl on D2. His total calcium was 8mg/dl and ionized calcium was 1.09mmol/l (1.12-1.32mmol/l) on D2. Serum sodium was 134mmol/l (135-148mmol/l) on D1 and became 138 mmol/l from D3. Patient was managed with intravenous (IV) 0.9% saline and Ringers Lactate (Hartman's) solutions for volume replacement. IV calcium gluconate, magnesium sulphate (MgSo4) and potassium chloride (KCL) were administered to maintain the electrolyte balance. The early post-operative course was notable for profound polyuria of 42 l in the first 24 hours.

Discussion

Polyuria following renal transplantation is common and is a desirable feature. Because previous studies have shown that initial high UOP is a predictor of immediate graft function. The Possible causes of post KT polyuria include rapid increase in the GFR in a kidney with malfunctioning nephrons due to pre transplantation ischaemia. Some other mechanisms are over administration of intravenous fluids and new onset hyperglycaemia after transplantation [6]. Post-transplant polyuria can result in fluid, electrolyte and acid base imbalances like hyponatremia, hypokalemia, hypocalcaemia, hypomagnesemia and hypophosphatemia and metabolic acidosis [8]. However, administration of excessive fluid for post-transplant polyuria results in persistence of polyuria and complications of fluid overload.

Therefore, accurate assessment of the fluid and electrolyte status and guided administration of intravenous fluids are the key steps in successful management of polyuria. The fluid status of the patient can be assessed clinically, and by measurement of pulse rate, mean arterial pressure (MAP) and Central Venous Pressure (CVP) values. In a patient with a pre-existing renal failure, measurement of systemic blood pressure may not reflect the fluid balance status of the patient. Because most of the patients with renal failure have systemic hypertension. In addition, blood pressure of these patients can also be altered by the cardiac status of the patients. Because in patients with renal failure heart failure can occur due to uraemic cardiomyopathy. Therefore, depending on CVP and MAP, guided fluid replacement is an established practice.

Crystalloids are the first recommended choice of fluid replacement. These are physiological and results in less side effects [9]. The fluid replacement is usually done with ringer lactate solution and 0.9% saline solutions. In patients needing rapid plasma expansion intravenous colloids (starch and albumin solutions) can be given. But randomized

trials have shown that infusion of starch solution is associated with renal impairment [10]. In addition, studies have found that administering plasma expanders like albumin, does not provide additional benefits other than in patients needing volume expansion.

Therefore, we administer intravenous crystalloids routinely in addition to correcting electrolyte imbalances with calcium, magnesium and potassium replacement. In early postoperative period IV potassium chloride (KCL) is administered if the serum K⁺ level is less than 3mmol/l. Our target MAP is 70 to 100 mmHg with a CVP target of 8-12cm H₂O. Our fluid replacement regime is; if hourly UOP is more than 1000ml it is replaced by UOP-100ml/hr rate of fluid. When UOP is reduced to 100ml/hr it is replaced with equal volume to volume replacement. Later when UOP drops to a rate of 50-100ml/hr replacement is done at a rate of 100ml/hr. but If UOP drops to less than 50ml/hr the input is increased to UOP+40ml/hr. In most patients the polyuria settles spontaneously. Very rarely drugs like Vasopressin, Captopril and Indomethacin are used to control polyuria. Therefore, post renal transplant polyuria is common and guided fluid replacement to avoid dehydration or over hydration is the key to successful management.

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