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Intradialysis Cardiac Arrest in a 40 year old Woman with Diabetic Nephropathy Precipitated by Hyperkalemia Induced by Type 4 Renal Tubular Acidosis.

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Abstract

Purpose: We present a rare case of intra-dialytic cardiac arrest secondary to renal tubular acidosis precipitated by severe hyperkalemia. Renal tubular acidosis (RTA) type 4 results from defective distal tubular (DT) response to aldosterone and could lead to hyperkalemia and metabolic acidosis, conditions which in their severe forms, can precipitate cardiac arrest.

Methodology: Here, we present the management of intra-dialysis cardiac arrest precipitated by severe hyperkalemia.

Results: Patient was an 18 year old female, diabetic who had 3 episodes of intra-dialysis cardiac arrest requiring cardiac defibrillation. She was dyspneic, drowsy, pale, had flapping tremor and a tunneled internal jugular catheter insitu. Her pulse was 124/min, blood pressure was 166/98 mmHg and the third heart sounds was heard. Urinalysis showed 3 (+++) of protein. Arterial blood gases (ABGs) revealed hyperkalemia (7.0mmol/l), hypobicarbonatemia (12mmol/l), blood PH (7.2) and blood glucose (249mmol). Serum creatinine was 947umol/l and tall tented T waves were seen on electrocardiogram.

Unique contribution to theory, practice and policy: With a carefully prescribed dialysis, meal and drug regimen, intra-dialysis cardiac arrest can be prevented, and well managed when it occurs.

Keywords: *hyperkalemia, type 4 renal tubular acidosis, metabolic acidosis, intradialysis cardiac arrest, diabetic nephropathy*.

Background

Renal tubular acidosis (RTA) is a common condition that is often associated with defective proximal tubular bicarbonate reabsorption and distal tubular hydrogen secretion. The commonest type of RTA (Type 4) often results from hyporeninemic hypoaldosteronism and leads to

hyperchloremic metabolic acidosis and hyperkalemia [1]. It is commonly seen from the fifth decade of life and is commoner in females. Hypoaldosteronism could results from reduced synthesis, secretion or loss of target organ response to aldosterone. The hyperkalemic form of RTA (Type 4) is conditions associated with tubulointerstitial nephritis without significant diminution of the glomerular filtration. Diabetic nephropathy, particularly Type 1, could be complicated by hyperkalemia, which in its severe form could lead to intra-dialytic events like cardiac arrest and death [2]. A high index of suspicion is needed to detect some of these challenges, which can be managed optimally to avert catastrophic consequences.

Problem statement: Intra-dialysis cardiac arrest is not uncommon and oftentimes can result to death. Prevention and prompt treatment is needed to avert death. We highlight the management of this condition and reviewed the literature.

Case Review

A 40 year old woman with end-stage kidney disease (ESKD) secondary to diabetic nephropathy, was referred to the Nephrology Division of Babcock University Teaching Hospital, Ilishan-Remo, from a peripheral dialysis center on account of three daily episodes of daily intra-dialytic cardiac arrest, requiring cardiopulmonary resuscitation and defibrillation. She was diagnosed type 1 diabetic at 18 years for which she used insulin. She progressed to chronic kidney disease at 26 years and was commenced on weekly maintenance hemodialysis (MHD) at 36 years. Dialysis frequency was increased to twice-weekly, a year prior to presentation in our facility. During each of her last three session prior to presentation, she had an episode of intra-dialytic cardiac arrest occurring within the first two hours of dialysis. Her routine medications were insulin, losartan, erythropoietin and hematinics.

She was dyspneic, drowsy, had a tunneled internal jugular catheter insitu.and pedal edema. Her pulse rate and blood pressure were 124 /min and 166/98mmHg respectively. The third heart sounds was heard, liver was enlarged and she had ascites and asterixis.

Urinalysis showed: protein (3+), glucose (3+) while arterial blood gases (ABGs) showed hyperkalemia (7.0mmol/l), hypobicarbonatemia (12mmol/l), a blood PH of 7.2, and blood glucose of 249mmol. Electrocardiogram findings included tall tented T wave while laboratory tests showed sodium (131mmol/l), potassium (6.9mmol/l), bicarbonate (15mmol/l) and creatinine (947umol/l) (Table 1). Full blood count showed: leucocytes (12.6 x 106), neutrophil (79%), and viral serologic markers were negative.

She was resuscitated, losartan was discontinued and was commenced on intravenous 10% Calcium gluconate 10 ml, which was given over 5 minutes, and was repeated every 3 hours till dialysis. She received 2 doses of nebulized salbutamol 5mg and a glucose-insulin infusion (5%, 10IU, 500ml) prior to dialysis. On account of her low oxygen saturation, she received intranasal oxygen at

6L/min. Diuretic therapy was with IV Tosemide 40mg daily and her antibiotic regimen consisted of IV Augmentin 1.2g twice daily, and IV Metronidazole 500mg 8-hourly.

Table 1: Kidney function test on admission

Variables	Sodium	Potassium	SBC	Chloride	Creatinine	Urea	URR
	mmol/l	mmol/l	mmol/l	mmol/l	µmol/l	mmol/l	%
	135-145	3.5-5.5	22-30	97-107	40-110	3-7	>65
Day 1 Pre-dialysis	131.0	7.0	15.0	97.0	947.0	22.0	
Day 1 Post-dialysis	133.0	4.8	19.0	100.0	558.0	13.0	40.9
Day 2 Pre-dialysis	132.0	5.9	18.0	99.0	646.0	14.0	
Day 2 Post-dialysis	136	4.1	21.0	104.0	318.0	6.8	51.4
Day 3 Pre-dialysis	134.0	5.3	19.	100.0	499.0	9.2	
Day 3 Post-dialysis	138.0	3.8	23.0	104.0	203.0	3.4	63.0

SBC-serum bicarbonate, URR-urea reduction ratio

The Endocrinologist was called in to review her following which her insulin regimen was changed to Sub cut Insulin 4 IU 6hourly with pre-insulin blood glucose check. Her glycated hemoglobin (HbA1c) was 8.6%. Chest X-ray showed cardiomegaly, aortic unfolding and peri-hilar haziness. Renal ultrasound showed reduced kidney sizes. (Table 2).

Table 2: Renal ultrasound scan of index patient

Variables	Length	Breadth	Width	Cortical thickness	Echogenicity
	10=14cm	4-6cm	2-3cm	>7cm	0-4
Right kidney	9.1	3.9	2.8	6.9	3
Left kidney	9.4	4.0	3.0	6.9	3

CMD-corticomedullary differentiation, Echo- 0(normal); 1(=liver, preserved CMD); 2(>liver, preserved CMD); 3(>liver, partial obliteration of CMD); 4(>liver with complete loss of CMD)

She had the first hemodialysis session in our center 3 hours after presentation with a prescribed blood flow rate (BFR) of 200ml/min, (that was increased by 50 ml/min half-hourly up to a maximum of 350 ml/min), duration of 3 hours, and ultrafiltration volume of 2000ml. The dialysate fluid had a bicarbonate and potassium concentration of 34mmol/l and 2.0mmol/l respectively. Dialysis was to be terminated whenever the heart rate rose up to 120 beats/min, BP was less than 90/60 mmHg (after administering IV Normal saline 500ml and, if necessary IV Hydrocortisone 100mg stat).

First session was uneventful and urea reduction ratio (URR) was 36.1%. Urine output was 1150ml for the first day. She was commenced on thrice-weekly dialysis the following day. The URR for the second and third sessions were 52% and 57% respectively. She was reviewed by the dietician who recommended a moderate carbohydrate, low protein and low fat diet, with liberal intake of recommended fruits and carefully parboiled vegetables.

She was discharged on oral medications that included Torsemide 20mg twice daily; Losartan 50mg daily, Amlodipine 5mg daily; Ferrous sulphate 400mg twice daily, Calcium carbonate 600mg twice daily with meals, Calcitriol 0.125ug daily, Sodium bicarbonate 600mg twice daily and Rosuvastatin 10mg nocte. Her “take home” drugs also included, IV Iron sucrose 200mg weekly, Sub Cut beta Erythropoietin 4000 IU twice weekly and she continue the insulin regimen regimen prescribed by the Endocrinologist.

When she was reviewed a week later, she had no new complaint and she had stable hemodynamics, She continued her maintenance dialysis sessions which have been uneventful. She is being worked up for a possible combined kidney-and-pancreas transplant.

Discussion

Hyperkalemic renal tubular acidosis (RTA Type 4) is characterized by defective distal tubular (DT) response to aldosterone, a steroidal hormone, produced by the adrenal zona glomerulus that

mediates sodium and water absorption, and potassium and acid secretion at the distal tubules and the collecting ducts (CD). Deficiency states lead to hyperkalemia and metabolic acidosis (MA) [2]. In RTA type 4, serum bicarbonate concentration (SBC) is typically not less than 17mmol/l [3]. The SBC of 15mmol/l in the index patient could be attributed to the additional renal insults from the cardiac arrest she suffered prior to presentation.

Impaired renin angiotensin-aldosterone system (RAAS) activity is commonly seen in conditions associated with preferential distal tubular affectation like diabetes and chronic interstitial nephropathies. The differential damage to the juxta-glomerular apparatus (JGA) from hyalinosis, (commonly seen in diabetic nephropathy) leads to urinary tract obstruction [4].

RTA type 4 is common between 50 and 70 years and in women (as the index patient) [5]. Its risk of occurrence is increased in renal graft recipients, from nephrotoxic immunosuppressive agents.

Ammonia tubular generation and hydrogen secretion by collecting ducts are needed for ammonium excretion in the urine [6]. Bicarbonate reabsorption by apical Na^+/H^+ in place of the $Na^+/K^+/2Cl^+$ co-transporter and K^+/H^+ antiport systems reduces the ammonia needed to buffer hydrogen ion thereby reducing urinary acid loss (incomplete form of RTA). However, retained intercalated cell hydrogen ions secretion acidifies the urine. Likewise, principal cell function is preserved in RTA type 4 [7].

The findings of chronic hyperkalemia, low-normal serum bicarbonate and urine PH <5.5 is very suggestive of RTA type 4 as the tonic effect of cortisol makes hyponatremia rare. Moreover, the absence of thirst prevents antidiuretic hormone (ADH) stimulation [8]. Withdrawal of precipitants is often enough in managing these patients. In the index patient with severe hyperkalemia (and ECG changes), it was imperative to stabilize the cardiac myocytes with calcium gluconate 10%, and correct hyperkalemia with beta adrenergic agonist and insulin-containing glucose solutions, in addition to the dialysis treatment.

Compliance with prescribed food regimen is also highly essential in RTA type 4 and it was re-emphasized at every clinic and dialysis visit by the index patient. The non-usage of fludrocortisone (one of the drugs used in treating RTA type) in the index patient was to avoid the risk of excessive fluid retention on account of kidney disease. Hemodialysis is indicated in non-resolving and potentially fatal hyperkalemia as was done for the index patient. Prognosis depends on etiology and is poor with ECG features of life-threatening disease, and in the presence of kidney disease [9].

Recommendations: In all patients on maintenance hemodialysis, dyselectrolytemia (particularly hyperkalemia which can precipitate cardiac events and case fatalities).

Proactive measures are needed to prevent its occurrence and to ensure promptly treatment.

Unique contributions based on the analysis conducted: A progressively optimized dialysis prescription can prevent and treat a hyperkalemia-induced cardiac arrest.

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