A Multi-Disciplinary Approach to Managing End-Stage Renal Disease in Anorexia Nervosa: A Case Report

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ABSTRACT

BACKGROUND: Renal dysfunction and electrolyte abnormalities are common complications of anorexia nervosa (AN), particularly in the binge-eating/purging type (AN-BP). Hypokalemic, or kaliopenic, nephropathy is an established clinical entity and a leading cause of end-stage renal disease (ESRD) in AN. Here, we present a case which demonstrates the difficulties of managing refeeding and nutrition in a psychiatrically and medically complex patient with severe AN-BP and ESRD most likely secondary to hypokalemic nephropathy.

CASE REPORT: A 54-year-old female with AN-BP that has resulted in chronic hypokalemia, with newly diagnosed ESRD on hemodialysis, presented to an eating disorder medical stabilization unit for weight restoration and treatment of the medical complications associated with her severe malnutrition and ESRD. She was admitted with a body mass index (BMI) of 15 kg/m^2, serum potassium of 2.8 mmol/L and serum creatinine of 6.91 mg/dL. She had failed to gain weight in the outpatient setting while on hemodialysis. She initially denied having an eating disorder, but ultimately a history of excessive laxative abuse for many years, without primary physician follow up, was revealed. While she did not undergo a renal biopsy to confirm the etiology of her ESRD, given her history of long-standing hypokalemia and lack of other risk factors, her ESRD was presumed to be secondary to hypokalemic nephropathy. She required significant oversight from a multidisciplinary eating disorder treatment team to restore weight while also managing her ESRD.

CONCLUSION: This case report highlights the complexity of managing ESRD in patients with AN who require weight restoration. A multidisciplinary team was vital to ensure this patient's adherence to treatment. With this case, we hope to raise awareness of the deleterious effect on the kidneys from prolonged hypokalemia, the elevated risk of poor renal outcomes in patients with AN-BP, and the danger of easy accessibility to over-the-counter stimulant laxatives.

KEYWORDS: Anorexia nervosa, binge-purge subtype, kaliopenic/hypokalemic nephropathy, end-stage renal disease, hypokalemia, refeeding/weight restoration

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Background

Anorexia nervosa (AN) is a complex and highly lethal psychiatric disorder that results in medical complications impacting almost every organ system. AN has 2 subtypes, restricting type (AN-R), where significant weight loss is primarily achieved through dieting, fasting, and/or exercising; and binge-eating/purging type (AN-BP), which involves recurrent episodes of binge eating and/or purging behaviors, such as self-induced vomiting or abuse of laxatives or diuretics, in addition to caloric restriction. Both subtypes have intense fear of weight gain, disturbance of body image, and symptom duration of at least 3 months.[1]

AN has the second highest mortality rate of all mental health disorders, with a standardized mortality ratio of 5.9% per decade, surpassed only by opioid use disorder.[2] Studies also show that relative to patients with AN-R, patients with AN-BP have a worse prognosis, more treatment-resistance, increased impulsivity, higher rates of comorbid substance use disorder, increased risk for self-harm and suicidality, more cognitive impairments, and worse renal outcomes.[3–14]

Studies report that more than 70% of patients with AN will have renal and electrolyte abnormalities in their lifetime,[15,16] and up to 5% of patients with AN will progress to end-stage renal disease (ESRD) after 2 decades of illness.[17] Compared to patients with AN-R, patients with AN-BP had twice the incidence of hypokalemia.[18] Hypokalemia remains one of the strongest risk factors for progressing to ESRD in AN,[19] for which the term "Kaliopenic" or Hypokalemic Nephropathy was proposed in 1956.[20] Hypokalemic nephropathy can result from any cause of chronic hypokalemia, including GI infections, inflammatory bowel disease, or renal wasting of potassium from hyperaldosterone states. However, one study predicted that 30% to 40% of patients with hypokalemic nephropathy have an underlying eating disorder.[21] We present a case of a patient with long-standing AN-BP who developed ESRD likely from hypokalemic nephropathy secondary to chronic hypokalemia from over-the-counter laxative abuse.

Table 1. Patient's laboratory workup.

LAB	PATIENT VALUE	REFERENCE RANGE
Sodium	133 (L)	135-143 mmol/L
Potassium	2.8 (L)	3.6-5.1 mmol/L
Chloride	92 (L)	99-110 mmol/L
Carbon dioxide	30 (H)	18-27 mmol/L
Blood urea nitrogen	76 (H)	6-22 mg/dL
Creatinine	6.91 (H)	0.50-1.39 mg/dL
Magnesium	2.3 (H)	1.3-22 mEq/L
Phosphorus	6.4 (H)	2.7-4.8 mg/dL
Albumin	4.1	3.2-5.5 g/dL
WBC	11.8 (H)	4.5-10 k/uL
Hemoglobin	8.1 (L)	12.0-16.0 g/dL
Hematocrit	25 (L)	37.0-47.0%
Platelets	311	150-400 k/uL
Prealbumin	19.6 (L)	20.0-52.0 mg/dL
Thyroid stimulating hormone	0.17 (L)	0.34-6.00 uIU/mL
Free T4	0.83	0.50-1.90 ng/dL
Vitamin D	49	30-90 ng/mL

Abbreviations: *H* = high value *L* = low value.

Case Report

Medical

A 54-year-old female with severe malnutrition and newly diagnosed ESRD was admitted for medical stabilization. Two months prior, she had presented to another hospital with an elevated serum creatinine of 9.56 mg/dL (normal range 0.50-1.39 mg/dL), necessitating initiation of dialysis, and hypokalemia with serum potassium level of 2.7 mmol/L (normal range 3.6-5.1 mmol/L). Her baseline creatinine was unknown. She had never required dialysis prior to that admission.

She was admitted to this medical stabilization unit, for patients with severe forms of eating disorders, after failure to gain weight in the outpatient setting and was still reliant on dialysis. Her past medical history was significant for chronic (of more than 10 years) hypokalemia, AN, hyponatremia, psychogenic polydipsia, osteoporosis, and hypophosphatemia. She reported being "excruciatingly itchy" for 2 years prior to her ESRD diagnosis, and had been diagnosed with food allergies by a dermatologist. Her itching resolved with dialysis and was ultimately attributed to uremic pruritis from her undiagnosed chronic kidney failure. She reported having been in denial of having an eating disorder until her ESRD diagnosis.

On the day of admission, her physical exam showed cachexia, with a height of 170 cm and weight of 44.2 kg (body mass index [BMI] of 15.2 kg/m^2 and percent ideal body weight [IBW] of 71.8%). Her temperature was 36.5°C, blood pressure 110/66 mmHg, heart rate 71 beats per minute, respiratory rate 15 breaths per minute, and oxygen saturation was 98% on room air. Admission orthostatic vital sign were within normal limits. Her mucus membranes were dry and her conjunctival capillaries were pale. Cardiopulmonary, abdominal, and neurological exam were benign. She had a tunneled dialysis catheter in her right chest, and complained of edema in her legs. Laboratory workup on admission (see Table 1) was significant for hyponatremia, hypokalemia, low prealbumin, low thyroid stimulating hormone, and multiple lab abnormalities congruent with kidney failure (such as elevated creatinine, phosphorus, BUN, and magnesium). See Table 2 for list of her home medications. Chest x-ray on admission confirmed appropriate location of the dialysis catheter.

Urinalysis results from the other hospital were not available, however, the admitting physician there wrote that her urinalysis showed moderate blood and protein. Kidney ultrasound performed at the other hospital revealed small kidneys with increased echogenicity of the cortex and cortical thinning, consistent with chronic kidney disease. No hydronephrosis, nephrolithiasis, renal mass or cysts were present. Renal biopsy had not been performed.

There was concern of ongoing laxative abuse given her hypokalemia on admission (see Table 1). Patient however

Table 2. Patient's home medications.

MEDICATION	DOSAGE	ADMINISTRATION
Levothyroxine	100 mcg	Daily
Calcium/Vitamin D	650 mg-1000 unit	Daily
Cinacalcet	30 mg	3×/week non-dialysis days
Epoetin alfa	10 000 units	3×/week on dialysis days
Sevelamer	2400 mg	TID with meals
Sevelamer	800 mg	TID with snacks
Vitamin B12	2500 mg	BID
Docusate	100 mg	BID
Hydroxyzine	25 mg	Every 6 h for anxiety
Mirtazepine	15 mg	HS for mood, anxiety, insomnia

denied using laxatives (or diuretics) other than docusate as prescribed (see Table 2). Her hypokalemia resolved with one oral dose of 20 mEq potassium chloride. She maintained normal potassium until week 2 of her hospitalization, when she developed hyperkalemia. She was then managed with dietary reduction of potassium along with dialysis. The complications of her ESRD were managed with nephrology consult, thrice weekly dialysis, a dialysis diet which was geared toward weight restoration, and routine medical care for ESRD—including treatment with phosphate binders, calcitriol analog cinacalcet, and a weekly darbepoetin alfa injection for anemia.

Given that her thyroid stimulating hormone (TSH) was suppressed on admission, with normal free T4, there was concern for excessive dosing of levothyroxine. Her thyroid medication was reduced, and then discontinued, when her TSH remained suppressed on recheck. This patient also experienced nearly daily episodes of hypoglycemia on fingerstick glucose checks, usually after waking up or immediately after dialysis. She also frequently complained of edema and would become upset when she felt not enough fluid was removed at dialysis, though edema was not observed on physical exam. Her hyponatremia on admission also resolved quickly with nutrition and dialysis. This patient arrived on a 1 L fluid restriction, and required extensive education to allow liberalization of fluids to facilitate her weight restoration. She also frequently complained of constipation, which was not always supported by objective evidence from her abdominal x-ray, and fullness, and had difficulty completing all of her prescribed meals and snacks; however, she declined medications to manage her suspected gastroparesis. Despite struggling to tolerate, both psychologically and physically, the weight restoration process while hospitalized, she reported a desire to return home as she felt confident she would be able to weight restore on her own.

Given the ego-syntonic nature of her AN, her minimization of the impact of her behaviors on her medical instability, and her hesitancy to continue with ongoing weight restoration, she required support from a multidisciplinary team, including a psychologist, psychiatrist, and registered dietician.

Psychology

This patient met with a clinical psychologist 4 days per week. At the beginning of her hospitalization, this patient presented as hyperverbal with pressured speech and tangential thought process. This was assessed to be due to anxiety, as well as having difficulty concentrating and focusing, largely as a result of malnutrition and uremia. During her intake session, she disclosed her history of AN-BP, explaining that it began in her late 30s secondary to stress. This patient only admitted to laxative abuse with bisacodyl when directly questioned about this behavior, and connected it to a need to alleviate constipation. She demonstrated extremely limited insight into the connection between her severe laxative abuse and renal dysfunction. Additionally, she remained guarded about the extent of her bisacodyl abuse, and she endorsed reluctance to gain weight due to her eating disorder cognitions. Through building trust with her treatment team, she was able to acknowledge a degree of connection between her stimulant laxative abuse and her ESRD, although required continued and frequent provider education regarding this fact.

Psychiatry

This patient met with a consulting psychiatrist approximately twice a week. The patient noted a recent history of depression and anxiety and admitted to the unit on mirtazapine 15 mg QHS for mood, anxiety and insomnia, along with hydroxyzine 25 mg every 6 hours as needed for anxiety or sleep. She was able to provide only limited clarity on her symptoms of depression other than feeling sad at times, and she denied hopelessness, suicidal ideation or a history of suicide attempts. Her anxiety centered around stressors, and identified her profession and

recent diagnosis of kidney failure as her main current stressors. Given the lack of a clear history of a mood and anxiety disorder predating her eating disorder, and the known impacts of malnutrition on mood, the patient was diagnosed with unspecified mood disorder and unspecified anxiety disorder, with the expectation that once the overlay of malnutrition resolved, a more definitive diagnosis could be given. She denied a family history of psychiatric illness. The patient was given extensive education about levels of available eating disorder care and the effects of malnutrition on the brain and body, along with the need for appropriate nutrition to optimally benefit from any psychopharmacologic or therapeutic interventions and to avoid dietary indiscretion in the setting of her ESRD. Her family provided collateral information regarding the severity of her eating disorder and they were also given education and support during her stay.

Nutrition

Five days per week, the patient met with a registered dietician (RD) who assisted the patient in making appropriate food choices that complied with the dietary restrictions of ESRD but also promoted weight restoration. Upon admission, the patient was put on a 1400 kcal diet with a 1 iter (L) all fluid restriction, 2 g sodium restriction, 2 g potassium restriction, and 1 g phosphorus restriction. After conferring with the renal team, the patient's fluid restriction was liberalized to and maxed out at 2 L, as the 1 L all fluid restriction was difficult to maintain given the need for meal plan increases. The patient's dialysis sessions caused difficulty with trending weights and managing meal plan increases, as the patient's weights would be falsely elevated prior to dialysis, and then much lower after dialysis. The patient's meal plan advanced by 400 kcals every 3 days until she was ultimately on a 3400 kcal/day meal plan.

This patient struggled with the quantity of food, and she was also very limited in her dietary choices due to her ESRD-induced restrictions of phosphorus, potassium, and sodium. Furthermore, her eating disorder thoughts encouraged her to choose foods that she deemed "healthy" or foods that would promote "muscle gain" versus "fat gain." The RD assisted the patient in incorporating Nepro Carb Steady (a nutrition supplement for patients on dialysis) into her meal plan so it could be included in her fluid restriction while helping reduce the volume of the food she needed to eat. Upon discharge, she was completing her meal plan of 3400 kcal and slowly restoring weight.

With support from her family and the multidisciplinary care team, this patient ultimately opted to continue on to a residential level of eating disorder care for increased focus on the psychological aspects of her illness and ongoing weight restoration, while still receiving dialysis 3 times per week.

Discussion

Mortality is markedly elevated in eating disorders.[22] This patient met the DSM-5 diagnostic criteria for severe AN-BP given her excessive restriction of calories leading to a low BMI of 15 kg/m^2, fear of weight gain, disturbance in body image with frequent complaint of edema and constipation, with laxative abuse of 15 years.[1] While data do not specifically show that AN-BP subtype is more fatal than AN-R subtype, many studies show that patients with AN-BP have poorer outcomes from a medical and psychiatric perspective.[9,11,12,14]

The medical complications associated with chronic laxative abuse are serious and deadly and include multiple electrolyte abnormalities, resultant QTc prolongation, kidney stones, cathartic colon and rhabdomyolyses, among others.[23] Stimulant laxatives, such as senna and bisacodyl, are easily accessible over the counter, and are typically fast acting, thus prone to abuse. This ready accessibility of over-the-counter stimulant laxatives likely contributed to this patient's ability to hide her laxative abuse. Currently, there is no restriction or oversight in the United States over the sale of stimulant laxatives to adolescents or adults. In 2020, the United Kingdom introduced restrictions on over-the-counter accessibility to stimulant laxative; large pack sizes are no longer available for purchase except under supervision of a physician or pharmacist, and sale to children under 12 is prohibited except when prescribed.[24] Having oversight and restriction on laxatives access at the consumer level might have prevented this patient from progressing to ESRD, particularly given this patient's lack of healthcare follow up.

Due to this patient's denial of having an eating disorder and the shame that patients often experience surrounding laxative abuse, she did not seek routine medical care or targeted treatment for her eating disorder, which lead to undiagnosed chronic kidney disease and ultimately ESRD. There is a 1 in 20 chance that a patient with a chronic eating disorder will progress to ESRD.[17] More research is needed to determine the incidence of ESRD in the AN-R versus AN-BP subtype. While this patient did not undergo a renal biopsy to confirm the etiology of her ESRD, given her history of chronic hypokalemia of over 10 years, concurrent history of laxative abuse, years of uremic pruritis preceding ESRD, and lack of other risk factors (such as diabetes, hypertension, or ingestion of nephrotoxic agents), her ESRD was presumed secondary to hypokalemic nephropathy.

Hypokalemic or kaliopenic nephropathy was first identified as a clinical entity in 1956 with vacuolization of renal tubular epithelial cells.[20] This abnormality generally developed after 1 month of persistent hypokalemia and reversed with potassium repletion. Chronic hypokalemia however led to more severe and irreversible changes, including tubulointerstitial nephritis and fibrosis, tubular atrophy, and cyst formation. These histopathological findings have been further corroborated in more recent case reports and cohort studies.[25-27] Recent studies also show that chronic hypokalemia impairs renal angiogenesis, which further leads to fibrosis from loss of peritubular capillaries.[28] Potassium repletion helped reverse some renal cysts, however, the tubulointerstitial changes and the associated renal insufficiency were often irreversible.[20,29-31] It is also likely

that, along with chronic hypokalemia, renal ischemia from dehydration also hastened the decline in renal function in this patient. Chronic tubulointerstitial nephritis from chronic hypokalemia along with acute tubular injury from dehydration may explain the moderate blood and protein seen in the urinalysis of this patient.

Differential diagnosis for this patient's ESRD also includes chronic kidney infections (though less likely, given she had no past medical history of it), and glomerular disease. Given the blood and protein in her urinalysis, glomerular disease cannot be definitely ruled out. Further studies, such as a 24-hour urine potassium collection (to distinguish between extrarenal vs intrarenal source of potassium loss), a 24-hour urine protein collection, and a renal biopsy would have aided in establishing a definitive diagnosis for her ESRD. Because these studies were not performed, we can only presume a diagnosis of hypokalemic nephropathy.

Levothyroxine likely also interfered with this patient's weight restoration. She was diagnosed with hypothyroidism while in acute kidney injury at the other hospital. Hypothalamic-pituitary axis changes are commonly seen with the severe malnutrition of AN, and with uremic syndrome.[32,33] Thyroid supplementation is generally not indicated in these scenarios. Not commonly seen in dialysis patients, this patient's hypoglycemia was attributed to her severe malnutrition and intermittent caloric restriction throughout her hospitalization.

In addition to the numerous medical complications, there is research to suggest that laxative abuse in patients with AN-BP is associated with greater severity of eating disorder psychopathology.[34-37] It is difficult to understand what leads individuals to abuse laxatives and how much their psychological distress contributes to their experience of constipation and other gastrointestinal symptoms. Laxative abuse has been framed as a form of self-punishment, self-harm, or as an anxiolytic.[38] One study suggested that laxative use may be a way of ritualistically and compulsively relieving tension produced by obsessional thoughts about fears about having food in the body.[39] It has also been noted that individuals who struggle with binge-eating and purging behaviors tend to be harm avoidant and reward dependent with high novelty seeking.[40,41] This in part may explain the higher rate of comorbid substance use disorder and increased risk of self-harm and suicidality seen in patients with AN-BP relative to patients with AN-R.[9,11]

Conclusion

We highlight the complexity of managing refeeding and weight restoration in a psychiatrically and medically complex patient with severe AN-BP and ESRD most likely secondary to hypokalemic nephropathy. We advocate a multidisciplinary team approach in the care of patients with extreme forms of eating disorders who require medical stabilization. With this case, we hope to raise awareness of the risks of long-standing hypokalemia associated with purging behaviors, the elevated risk of renal complications in patients with AN-BP, and the dangers of easily accessible over-the-counter stimulant laxatives.

Author Contributions

V.K., writing—original draft preparation abstract, background, case report-medical, discussion, conclusion, writing—review and editing of entire manuscript, project administration; M.B., writing—original draft preparation case report-psychiatry, discussion-psychiatry, writing—review and editing of entire manuscript; M.F., writing—original draft preparation case report-nutrition, writing—review and editing of entire manuscript; C.L., writing—original draft preparation case report-psychology, writing—review and editing of entire manuscript; P.M., conceptualization of entire manuscript, writing—review and editing entire manuscript, supervision of entire manuscript. All authors have read and agreed to the published version of the manuscript.

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