

Case Report

An Unexpected Case of Scurvy in a Peritoneal Dialysis Patient

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Keywords

Scurvy · Ascorbic acid · Vitamin C · Peritoneal dialysis · Dialysis vitamin · Nutrition

Abstract

This case describes an obese adult male peritoneal dialysis patient who presented with a pruritic follicular rash. Nutrient deficiency was not suspected initially in this case because there was no history of protein-calorie malnutrition, but the patient reported a diet devoid of fruits and vegetables and had not been taking his dialysis vitamin as prescribed. Skin biopsy showed follicular hyperkeratosis with fragmented hair shafts and corkscrew hairs consistent with scurvy. After supplementation with ascorbic acid 500 mg twice daily for 2 weeks, the rash resolved completely. Dialysis patients are at increased risk for vitamin C deficiency due to indiscriminant clearance of the nutrient with dialysis, but scurvy is rarely seen.

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Introduction

Scurvy, a disease resulting from vitamin C deficiency, is a disease that has afflicted humans throughout history [1]. In modern times, scurvy is rare but has been reported in patients who are deficient in vitamin C intake or absorption [2]. The kidneys normally reabsorb vitamin C and appear to excrete it in the urine only when it exceeds a threshold serum level. Dialysis patients are at risk for deficiency because of the indiscriminant clearance of vitamin C via dialytic losses [3]. Coupling poor dietary intake of vitamin C with dialysis increases the risk of deficiency. Recently, a case of scurvy was reported in a 6-year-old peritoneal dialysis (PD) patient with developmental delay and poor intake of dietary sources of vitamin C [4]. In this report, we present a case of scurvy in an obese adult PD patient who consumed a Western diet heavy in animal protein and starches and scant in fresh fruits and vegetables.

Case Report

A 65-year-old Caucasian American man with end-stage renal disease due to diabetic nephropathy treated with PD presented with a 1-day history of an intensely pruritic rash. It initially erupted over his trunk then spread to his arms and legs, sparing the palms and soles. No new topical exposures or medications had been introduced. He tried taking diphenhydramine and ranitidine for the itching but presented to the hospital after finding no relief. The past medical history included insulin-dependent diabetes mellitus, coronary atherosclerotic disease, congestive heart failure, liver disease thought due to chronic passive congestion, hypertension, history of renal cell carcinoma treated with unilateral nephrectomy, recurrent lower extremity deep venous thrombosis, and obesity. Medications included aspirin, venlafaxine, isosorbide mononitrate, humulin R insulin, amlodipine, carvedilol, ramipril, calcium acetate phosphate binders, and warfarin.

The peritoneal dialysis prescription was five 2.5-liter exchanges of dextrose-containing solution (Dianeal, Baxter) over 9 h of cycling peritoneal dialysis at night and a long dwell of 2 L of icodextrin-containing solution (Extraneal, Baxter) during the day. Weekly Kt/V urea kinetics were: PD: 1.64, kidney: 0.43, total: 2.07. The patient had been on peritoneal dialysis for approximately 1 year prior to presentation. There was no history of protein-calorie malnutrition or gastrointestinal disease and no history of alcoholism. Serum albumin measurements were 3.7 g/dL prior to starting peritoneal dialysis and had ranged between 2.8–4.1 g/dL (mean 3.47 g/dL) since dialysis initiation. Nutritional history revealed that the patient consumed a diet rich in animal protein and starches and did not eat fruits or leafy vegetables because of personal preference. His weight had remained stable over the past year. He had been prescribed a dialysis vitamin that contained vitamin B complex and vitamin C but had not been taking it.

Physical examination showed no fever or hemodynamic instability. The patient's height was 1.80 m, weight was 124.67 kg, and body mass index was 38.4. He appeared uncomfortable due to pruritus. Central adiposity was evident and there was no sign of muscle wasting or atrophy. There were widespread perifollicular keratotic erythematous papules on abdomen, chest, arms, and legs (Fig. 1). There were two dark red purpuric gingival lesions. No other

signs of bleeding were noted. Serum laboratory studies showed hemoglobin: 10.0 g/dL (14.0–18.0 g/dL) and albumin: 2.9 g/dL (3.2–5.2 g/dL) (Table 1). Peritoneal fluid laboratory studies showed white blood cells: 4/mm³ and red blood cells: 1/mm³.

Skin biopsy revealed a perivascular lymphohistiocytic infiltrate with interstitial eosinophils and mast cells. There were extravasated red blood cells both perifollicularly and interstitially and follicular hyperkeratosis with fragmented hair shafts and corkscrew hairs consistent with scurvy (Fig. 2). The patient was treated with ascorbic acid 500 mg by mouth twice daily for 14 days. The rash improved after 2 days of treatment and was completely absent after 2 weeks. The patient was transitioned to a daily dialysis vitamin that contained 60 mg of vitamin C per tablet.

Discussion

Vitamin C (ascorbic acid) is an essential nutrient with many roles in the human body, including cofactor in multiple enzymatic reactions, co-substrate, antioxidant, and reducing agent [5]. It is required for the formation of collagen, normal immune function, and the generation of corticosteroids and catecholamines [5–8]. Scurvy is largely due to impaired collagen synthesis that occurs from lack of adequate vitamin C. The signs and symptoms of scurvy, which can occur as early as 3 months into a vitamin C-deficient diet, include petechiae or frank ecchymoses, gingival bleeding, coiled hairs and hyperkeratosis, malaise, edema, joint swelling, neuropathy, vasomotor instability, and impaired wound healing [2, 9]. More subtle symptoms of fatigue and irritability without other features of clinical scurvy have been described in the non-dialysis population with vitamin C levels approaching 2 mg/L [10]. The patient in this case had predominantly skin findings and some mild gingival bleeding.

Daily vitamin C requirements to prevent scurvy in the non-dialysis population are estimated at 75 mg for women and 90 mg for men. Scurvy can occur in healthy volunteers when the total body pool of vitamin C falls below approximately 300 mg, which correlates with a serum ascorbate level of less than 2–3 mg/L [9]. Vitamin C deficiency is defined as <2 mg/L and insufficiency as 2–4 mg/L [2].

The normally functioning kidneys reabsorb vitamin C and excrete it in the urine when the serum level exceeds 60 μM (10.5 mg/L) [11]. Vitamin C is removed by both PD and hemodialysis, putting dialysis patients at risk for deficiency. Removal of ascorbate by PD is proportional to the peritoneal creatinine clearance, with losses estimated at 0.095 mL/s for a 2.5% dextrose exchange and 0.084 mL/s for a 1.5% dextrose exchange [3]. A study by Singer et al. [2] demonstrated loss of 31 mg of ascorbate per 6-hour exchange and found 33% of their PD cohort to have ascorbate deficiency.

Most dialysis vitamins contain some amount of vitamin C to help replace dialytic losses, commonly in doses ranging from 50 to 60 mg per day. A study of continuous ambulatory peritoneal dialysis patients demonstrated that doses of 80–140 mg/day of vitamin C safely maintained serum ascorbate levels within normal limits [12].

Dialysis patients are at increased risk for vitamin C deficiency and insufficiency. Herein, we report a case of scurvy in a peritoneal dialysis patient. The diagnosis was unexpected because the patient did not appear to have protein-calorie malnutrition, so nutrient deficiency was not suspected. The patient did have a low serum albumin; however, this finding was

attributed to liver disease and portal hypertension increasing albumin losses in the peritoneal dialysate instead of protein malnutrition.

Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

The authors declare that they have no conflicts.

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Fig. 1. The patient had a widespread follicular hyperkeratotic pruritic rash.



Fig. 2. Histologic examination of skin biopsy revealed follicular hyperkeratosis (arrow) with multiple corkscrew hair shafts (black circles) and perifollicular and interstitial infiltrates composed of lymphocytes and extravasated red blood cells (box). Hematoxylin-eosin stained digital scanned images.

Table 1. Laboratory studies

	Result	Reference range
Hematology		
White blood cell count, ×1,000	2,800	4.8–10.8
Hemoglobin, g/dL	10.0	14.0–18.0
Hematocrit, %	29.3	42.0–52.0
Mean corpuscular volume, fL	96.4	80.0–94.0
Platelet count, ×1,000	70	160–360
Chemistry		
Sodium, mmol/L	131	132–146
Potassium, mmol/L	4.3	3.5–5.3
CO ₂ , mmol/L	23	21–33
Blood urea nitrogen, mg/dL	62	8.0–24.0
Creatinine, mg/dL	8.92	0.5–1.5
Phosphorus, mg/dL	4.2	2.5–4.5
Calcium, mg/dL	8.2	8.5–10.5
Magnesium, mg/dL	2.1	1.8–2.4
Intact parathyroid hormone, pg/mL	536	12–72
Albumin, g/dL	3.4	3.2–5.0
Ferritin, ng/mL	215	30–300
Transferrin saturation, %	52	20–50
Alkaline phosphatase, U/L	51	20–125
Aspartate aminotransferase, U/L	20	5–50
Hemoglobin A1c, %	6.7	4.5–6.4