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Rare Presentation of Uremic Frost in a Young Adult with Advanced Renal Failure

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ABSTRACT
Uremic frost is an uncommon occurrence that can arise as a result of chronic renal failure. Here, we offer a case of a 32-year-old male who presented for the first time with the lesions. The most prominent physical observation was the presence of a white powdery frost covering the entire body, particularly concentrated on the face and limbs. Frosting can occur with less severe forms of uremia, but typically occurs when the blood urea nitrogen concentration exceeds 200 mg/dL. The impairment of the small blood vessels and the structures responsible for hair follicles and oil glands, observed in chronic renal illness, may explain the accumulation of elevated quantities of urea on the skin surface. The primary objective of uremic frost treatment is to rectify the root cause of uremia and other critical medical conditions. Uremic frost is a rare manifestation of chronic kidney disease. Here, we present a case of a 32-year-old boy who presented for the first time with the lesions. The most striking physical finding was whitish powdery frost all over his body, especially the face and limbs. Uremic frost generally occurs at blood urea nitrogen of more than 200 mg/dL, although it may arise with less severe uremia. Damage of cutaneous microvasculature and pilosebaceous units, as seen in chronic kidney disease, could account for the high levels of urea deposited outside the skin. Treatment of uremic frost is mainly aimed at correcting the underlying cause of uremia and other life-threatening conditions.

INTRODUCTION

Uremic frost is an uncommon manifestation of azotemia. This disease is currently uncommon as a result of prompt dialysis therapy. This disorder, known as uremic frost, arises when significant quantities of urea and other nitrogenous waste substances are present in perspiration and solidify on the skin following evaporation. This condition is referred to as dyshidrosis or dyshidrosis. This is indicative of renal failure and indicative of suboptimal physical condition. The participation of the urea transporter may have a significant impact on its development. Urea accumulation in the skin can occur due to damage to the capillaries, eccrine glands and sebaceous glands. To ascertain if the crystalline substance is urea or nitrogen-containing trash, the scraped sample can be diluted with physiological saline and examined for an increase in urea nitrogen concentration. There is no alternative treatment other than the treatment mentioned below[1,2].

Chronic kidney disease (CKD) refers to a range of physiological processes that are linked to poor kidney function, frequently resulting in a gradual decrease in glomerular filtration rate or GFR (Fig. 1). The likelihood of deteriorating CKD is intricately associated with both the GFR and the level of albuminuria. The image presents a classification of CKD based on estimations for the likelihood of continued progressive deterioration in GFR using these two factors[2]. End-stage renal disease is a stage of CKD characterized by the buildup of toxins, fluid and electrolytes that are typically eliminated by the kidneys. If these toxins are not cleared, it might result in death.

Case report: A 32-year-old male patient presents with complaints of scrapping of skin for the last 1 month, increased sleepiness since 20 days and edema on lower limbs for the last 3 days. **History** the patient had h/o viral meningoencephalitis 6 years back. No h/o of chronic illness. The patient had a h/o of. The patient's vital signs on presentation were a temperature of 37.20C, heart rate of 102 beats/min, respiratory rate of 28 breaths/min, blood pressure of 100/70 mmHg and oxygen saturation of 97% on room air. On examination, the patient had pallor present, icterus absent, cyanosis absent and edema in lower limbs present. On local examination, the skin was On physical examination, he had uremic fetor, the most striking physical finding was whitish powdery frost, which was present all over his body, especially over the face and limbs (Fig. 2,3). On CNS examination, the patient had altered consciousness, was not responding to commands and was drowsy.

Based on the laboratory findings, the patient was confirmed with chronic kidney disease (e.g.,-9 ml/min/1.73m2). The patient's treatment was started with an empirical antibiotic and a blood culture and urine culture were sent. On ECHO, mild pericardial effusion, patient attendees were convinced about the need for dialysis. Consent for central venous line insertion was taken and was inserted. The patient was put on hemodialysis with a double-lumen internal jugular catheter under local anesthesia. Hemodialysis for 21/2 hours was done. OBF:- 120 ml/min OAF:- 300 ml/min. ultrafiltration :- 1L .

RESULTS AND DISCUSSIONS

Chronic kidney disease (CKD) embodies a spectrum of kidney dysfunctions marked by a.

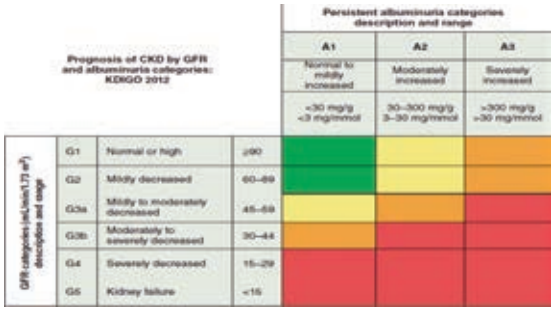

Fig. 1: This figure illustrates the classification of CKD based on GFR and albuminuria categories according to the KDIGO guidelines.

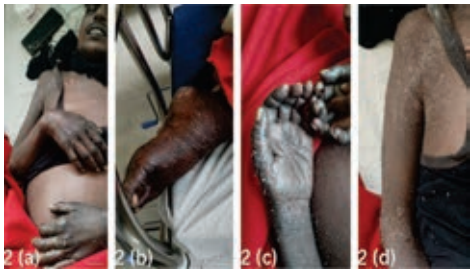

Fig. 2: Manifestations of Uremic Frost in Advanced Renal Failure (a) Thoracic and abdominal extensive uremic frost, (b) Uremic Frost on Left foot, (c) Palmar view of uremic frost, (d) Ventral view of right arm.

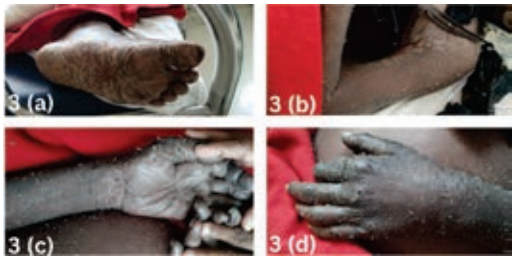

Fig. 3: Manifestations of Uremic Frost in Advanced Renal Failure. (a) Plantar view of the left foot (b) Left upper extremity (c) Palmar aspect showing dense frost across the left palm. (d) Dorsal view of left palm

Table 1 : Laboratory Finding

Laboratory parameters		At the time of admission
Cbc	Hb	4.7
	Wbc	27.56
	Rbc	1.83
	Platelet	313
Renal function test	Urea	218 mg/dL
	Creatinine	9.4
Urine analysis	Pus cells	30-35
	Urine sugar	Absent
Liver function test	Total protein	6.4
	Albumin	2.8
	Globulin	2.1
	Sgpt	19.0
	Sgpt	15.4
	Alp	142.6
Serum electrolytes	Sodium	<120
	Potassium	5.1
Radiological Findings		
USG Abdomen	Bilaterally small Kidneys	

progressive decline in glomerular filtration rate (GFR The progression of CKD is closely linked to GFR levels and the extent of albuminuria. The disease's pathophysiology is multifaceted, with initial damage mechanisms varying based on the root cause. For instance, specific types of glomerulonephritis may involve inflammatory processes and the deposit of immune complexes, while renal tubular and interstitial diseases might stem from exposure to harmful substances[1]. Additionally, CKD's evolution involves general mechanisms like the compensatory hyperfiltration and enlargement of the remaining healthy nephrons. This response is governed by various hormones and growth factors that react to the nephron deficit[2]. Initially, these adaptations may help maintain kidney function, however, they can eventually lead to deleterious outcomes. Increased pressure and flow within the nephrons may distort the structure of the glomeruli, impair podocyte functionality and compromise the integrity of the filtration barrier, culminating in sclerosis and the loss of nephrons. The renin-angiotensin system (RAS), with its augmented activity within the kidneys, contributes to both the early compensatory phase and the subsequent damaging processes[2]. This understanding clarifies how a reduction in renal mass due to an isolated incident can progressively impair renal function over time and underscores the potential benefits of pharmacological interventions that target these pathways.

High blood urea levels can lead to its accumulation in the dermis and subsequent release through the sweat glands, a phenomenon known as "uridrosis" or "urine sweat". The formation of uremic frost crystals results from the evaporation of the water component of this secretion[3]. While the precise pathophysiological mechanisms behind uremic frost remain unexplored in vitro, recent studies have suggested potential explanations for urea leakage. Even in healthy individuals, urea is naturally secreted through sweat, indicating the existence of physiological mechanisms for urea excretion via eccrine sweat glands[4]. The hypothesis that uremia exacerbates atherosclerosis through endothelial damage is supported by emerging evidence indicating that kidney disease may also increase Micro vascular permeability[5]. This could contribute to transporting urea to the skin's surface. The examination of common skin issues in renal disease, such as uremic pruritus and xerosis, might provide insights into uremic frost's pathogenesis. Despite no increase in transdermal water loss in renal disease[6], xerosis remains prevalent in dialysis patients due to eccrine sweat and sebaceous gland dysfunction[7]. Interestingly, the application of emollients has significantly alleviated pruritus for these individuals[6]. The topical use of urea for various dermatological conditions hints at its regulatory role in skin function. The presence of urea transporters like UT-A1 suggests a specific function for urea in the skin[8], with facilitated diffusion likely contributing to the external deposition of urea in cases of uremic frost. Immunohistochemical studies indicating an upregulation of UT-A1 and UT-B1 transporters in the skin of severely uremic patients further support this notion[9].

Although skin scrapings to measure urea content can confirm a diagnosis[9,10], they are generally unnecessary unless distinguishing from conditions with similar presentations, such as cystic fibrosis. Moreover, the presence of uremic frost is often associated with prolonged renal failure. Differential diagnosis includes atopic dermatitis and other conditions that produce skin scales, but these are typically distinct from the crystalline appearance of uremic frost. While uremic frostbite and uremic pruritus can co-occur, it's crucial to consider that white flake formation could be attributed to uremia or an alternate dermatological condition. Distinguishing uremic frost from other conditions, especially in hair-covered areas where it may resemble seborrheic dermatitis, is important. Lastly, although crystal deposition within the dermis is noted in diseases like gout and oxalosis, it has not been observed to precipitate externally like uremic frost[11-14].

CONCLUSION

The case shows a patient with a lesion of uremic frost due to an increase in urea levels in the body, which was accumulated due to CKD. The patient was started on hemodialysis with the insertion of a jugular catheter and the patient improved in consciousness as well as in the lesions present over the body.

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