CASE REPORT

Diagnostic dilemma of flaky paint dermatitis versus acquired acrodermatitis enteropathica in a person presenting with generalized dermatitis, diarrhoea, and reduced level of consciousness

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Abstract

Differentiating Flaky Paint Dermatitis (FPD) in Protein Energy Malnutrition (PEM) from skin manifestations of Acquired Acrodermatitis Enteropathica (AAE) imposes a challenge due to overlapping clinical manifestations. This case study is on a 76-year-old presenting with reduced level of consciousness, generalized body oedema, and desquamating dermatitis with areas of hypopigmentation. His intake has reduced due to difficulty in swallowing, immobility, dependency for activities of daily living, and poor family support. This has led to severe malnutrition complicated with electrolyte imbalances, dehydration, and aspiration pneumonia. The hypothetico-deductive approach led to two main differential diagnoses in the forms of PEM and AAE. Following stabilization with correction of dehydration and imaging to exclude other causes, the nutritional plan was gradually implemented. Energy and protein delivery were increased under thiamine cover due to the high risk of refeeding while correcting the electrolyte imbalances. One recommended daily allowance (RDA) of vitamins and minerals was administered through a multivitamin and mineral syrup. Supportive measures were also given, preventing hypothermia, hypoglycemia, and application of aqueous cream, betamethasone with soframycin for skin lesions.

Even though the oedema settled with time, skin manifestations did not respond as expected with PEM treatment. There was rapid clinical improvement with the addition of a higher dose of zinc therapy (in the form of $ZnSO_4$ 1mg/kg), suggesting underlying zinc deficiency. This case study highlights the diagnostic challenge between AAE and FPD in PEM, particularly in resource-limited settings where clinicians should maintain a high index of suspicion for multiple nutritional deficiencies not only in children, but also in adults presenting with severe dermatitis and malnutrition.

Keywords: flaky paint dermatitis; kwashiorkor; acrodermatitis enteropathica

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Introduction

Protein-energy malnutrition (PEM) is a multifactorial condition that is commonly seen in children. There are several entities of this, ranging from marasmus and kwashiorkor. In kwashiorkor, usually there is adequate carbohydrate consumption and decreased protein intake, leading to decreased synthesis of visceral proteins. The resulting hypoalbuminemia contributes to extravascular fluid accumulation. Impaired hepatic synthesis of B-lipoprotein produces a fatty liver. Low zinc levels cause skin ulceration appearing as typical flaky paint dermatitis (FPD). This is difficult to differentiate from the dermatitis in acrodermatitis enteropathica. Generally, PEM is not common in adults, and the presentation can vary from body weakness to body oedema, and may also be reflected with skin manifestations.

Acquired acrodermatitis enteropathica (AAE) is a form of zinc deficiency caused by external factors rather than inherited defects in zinc absorption. Characteristic features of acrodermatitis are acral/periorificial dermatitis (with sharp margins, vesicular and pustular lesions), alopecia, and diarrhoea.[1] Zinc deficiency may coexist in severe protein-energy malnutrition. Diagnosis of this condition needs to be context-specific in a resource-limited setting like Sri Lanka. The present case had overlapping features of both conditions, such as skin manifestations, alopecia, and diarrhoea. Furthermore, dermatological features did not respond to standard PEM treatment.

Case Presentation

A 76-year-old male patient had a background history of a chronic medical condition, which led to immobility. He presented with a reduced level of consciousness and generalized body oedema. His level of consciousness had deteriorated during the past few weeks, and intake had been reduced as well. Loss of appetite and loss of weight could not be identified. However, there was generalized skin peeling with itching. There were no other signs of infections apart from the respiratory signs, which were possibly secondary to aspiration pneumonia. He was having loose stools for a few days, which were watery in consistency, with a frequency of 4-5 times /day. There was a considerable reduction in food intake, and energy delivery was less than 10kcal/kg/day with a very low amount of protein intake (less than 0.4g/kg/day). There were no good-quality proteins in his diet. His intake was reduced due to difficulty in swallowing, immobility, dependency for activities of daily living, and poor family support.

Examination revealed features of dehydration and low GCS ranging from 10 to 11 /15, generalized dry skin with thick scales, erythematous and hypopigmented areas with deep fissures. Even though this was generalized, it was worse in the hands, lower limbs, and face. Hair was dry, sparse, and easily pluckable. There was generalized body oedema, mainly involving both upper and lower limbs (Figure 1).

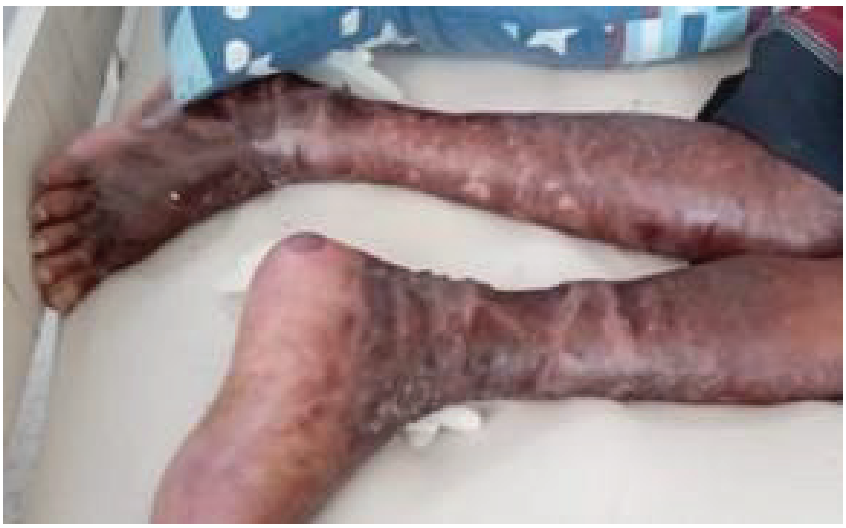


Figure 1. Bilateral lower limb oedema at admission.

Table 1. Laboratory investigations on admission. Reference ranges are shown in the right column

Investigation	Findings	Normal range
Haemoglobin (g/dL)	6.6	12-16.5
MCV (fL)	93.2	80–100
RDW %	15.4	11-16
Platelet (X10 ^{3}/uL)	64	150-400
White cell count (X10 ^{3}/uL)	9.84	4-11
Sodium (mmol/L)	133	135-145
Potassium (mmol/L)	2.8	3.5-5.1
S. Creatinine (mg/dL)	1.1	0.72-1.25
eGFR (ml/min /1.73m^2)	72.67	> 90
AST (u/L)	76	5-34
ALT (u/L)	68	0-55
Serum albumin (g/dL)	1.8	3.5- 5
CRP (mg/L)	220	0-5
Calcium (mg/dL)	6.1	8.6-10.3
Magnesium (mg/dL)	1.1	1.6-2.6
Phosphorus (mg/dL)	2.7	2.3-4.7

Weight (as bed-bound) and the mid-upper arm circumference (MUAC) (as oedematous) could not be measured, but there were features of muscle wasting and fat loss. Investigations revealed electrolyte abnormalities, low calcium, potassium, and magnesium levels, and a low haemoglobin level of 6.6 g/dL. His liver enzymes were marginally elevated. The albumin level was low (Table 1). On admission, the blood sugar level was 78 mg/dL. The blood picture showed RBC- normocytic normochromic blood cells, suggesting possible bacterial infection/sepsis.

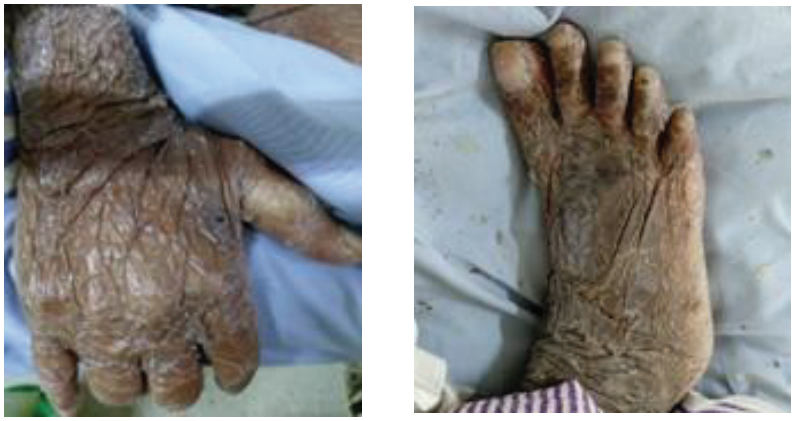
Diagnosis and Management: The main differential diagnoses in this case presentation were FPD in PEM and AAE. Certain features were in favour of niacin deficiency, but the skin manifestations were not typical of pellagra.[2] However, there were no facilities to check the micronutrient levels, such as serum niacin levels and serum/red blood cell or hair zinc levels, in our setting.

Initial management was to stabilize the patient. Dehydration was corrected using intravenous

fluids. Imaging was arranged to rule out the acute medical conditions that might have needed urgent care. CT scan of the brain revealed the chronic subdural haemorrhage, which did not indicate surgical management. Electrolyte derangements were corrected using intravenous KCl, $MgSO_4$, and calcium supplements. One unit of packed red blood cells was transfused. Gradually, a nutritional plan was implemented, considering the very high risk of refeeding under intravenous thiamine cover. Since the patient's level of consciousness was reduced, feeds were given through a nasogastric tube, taking precautions to prevent aspiration. Energy delivery initiated with 10 kcal/kg /day with protein 0.8g/kg/day. Energy intake and protein delivery were slowly increased to (40 kcal/kg of energy and 1.5g/kg of protein) while monitoring for deterioration of electrolyte imbalances and correcting any. One RDA of all vitamins and minerals was given in the form of a multivitamin and mineral syrup. Respiratory tract infection was treated with intravenous antibiotics and aqueous cream, soframycin + betamethasone creams were applied following dermatology opinion. Supportive measures were also given to prevent hypothermia and hypoglycemia.

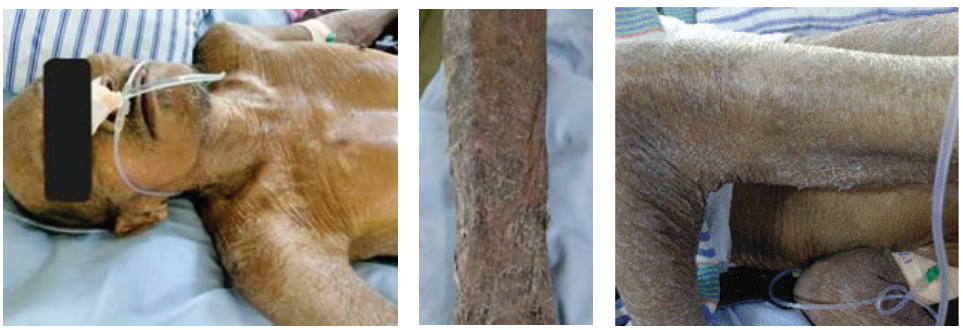
The body oedema improved gradually with an increase in energy and protein delivery (Figure 2A-B). Oedema completely disappeared after 2 weeks of adequate protein and energy.

However, the skin manifestations did not respond as expected. Therefore, zinc sulphate was started to treat skin manifestations (zinc sulphate 20 mg tds, providing 1 mg/kg/day), keeping in mind the possibility of AAE.[3,4] Multivitamin and mineral delivery increased to a level of double the RDA. There was a notable improvement in skin manifestations within two days of $ZnSO_4$ therapy (Figure 3A-C).



A B

Figure 2. Resolution of oedema after nutritional therapy.
(A) Appearance of the hand following the settlement of oedema.
(B) Appearance of the foot following the settlement of oedema.



A B C

Figure 3. Skin manifestations two days after initiation of zinc therapy. (A) Body.
(B) Legs. (C) Upper limbs.

Since there was an increased risk of aspiration with oral feeds (impaired swallowing), feeds and medications were given through a nasogastric tube. PEG tube insertion was planned for long-term enteral nutrition.

Discussion

This is a case that highlights the diagnostic dilemma of flaky paint dermatitis secondary to severe PEM and acquired acrodermatitis enteropathica, both of which potentially present with overlapping skin manifestations (Table 2). The index patient presented with generalized desquamating dermatitis, periorificial and acral involvement, reduced level of consciousness, and body oedema, making the differentiation between micronutrient deficiency and severe macronutrient malnutrition challenging.

Acrodermatitis enteropathica is classically characterized by periorificial and acral dermatitis, alopecia, and diarrhea resulting from zinc deficiency.[1] However, several studies have reported that acquired zinc deficiency can occur in the setting of PEM, particularly kwashiorkor, due to reduced zinc-binding proteins such as albumin and impaired intestinal absorption.[2,3] Rapid tissue breakdown during acute illness may further worsen serum zinc levels. FPD, a hallmark of kwashiorkor, presents with hyperpigmented patches, desquamation, and

Table 2. Comparison of flaky paint dermatitis, acrodermatitis enteropathica, and pellagra

	Flaky paint dermatitis	Acrodermatitis enteropathica	Pellagra
Cause	PEM	Zinc deficiency	Niacin deficiency
Typical skin manifestations	Dry scaly dermatitis with peeling and hypopigmented areas	Erythematous, vesicular or eczematous dermatitis	Hyperpigmented scaly dermatitis
Distribution	Generalized, commonly over limbs and pressure areas	Acral and periorificial areas	Symmetrical, mainly sun-exposed areas
Other mucocutaneous features	Glossitis, Angular stomatitis, Alopecia	Nail changes – brittle nails, ridges, Alopecia	Glossitis, chelitis, angular chelitis, mouth ulcers, Alopecia
Systemic features	Body swelling	Diarrhoea	Diarrhoea Dementia Neuropsychiatric manifestations
Diagnostic investigations	Clinical diagnosis, serum albumin/total protein levels	Serum/ blood cell or hair zinc levels	Blood or tissue NAD levels
Management*	Adequate energy and protein with micronutrients	Acquired- Elemental Zn 0.5-1mg/kg/day for 3-4 months Congenital – life-long 3mg/kg/d of zinc therapy	Nicotinic acid 15-20mg/kg/d + nicotinamide 300mg /d

*** See reference 4**

peeling skin, which may mimic zinc deficiency – related dermatitis. This potential overlap creates a diagnostic challenge, especially in resource-limited settings where zinc level measurements may not be readily available.

The rapid dermatological improvement following high-dose zinc supplementation supports the possibility of overlapping deficiencies rather than a single isolated etiology. Similar observations have been reported in the literature, emphasizing that children with severe malnutrition may frequently get multiple micronutrient deficiencies, which can be seen in adults as well. Pellagra was considered in the differential diagnosis because of diarrhoea and dermatitis; however, the absence of classical photosensitive dermatitis and the rapid response to zinc therapy made this diagnosis less likely.

This case provides important clinical teaching points. First, clinicians should consider combined macronutrient and micronutrient deficiencies when evaluating a case with severe dermatitis and malnutrition. Secondly, reliance solely on dermatological appearance may be misleading, as both conditions can share overlapping features. The third point is that early empirical zinc supplementation (usually with one RDA, but with higher doses in suspected zinc deficiency), along with standard management of severe acute malnutrition, may be justified in suspected cases, particularly in settings where laboratory confirmation is not readily available. Early recognition and prompt management are crucial, as untreated zinc deficiency and severe PEM are associated with increased morbidity and mortality.

This case study has several limitations. Serum zinc levels were not measured, which limited definitive differentiation between isolated zinc deficiency and kwashiorkor-associated dermatitis. Additionally, a dermatological biopsy was not performed, since it was thought to be of limited value. The short follow-up period also limited the assessment of long-term outcomes and recurrence. Despite these limitations, the clinical response to therapy and overall clinical picture strongly suggest overlapping nutritional deficiencies.

In conclusion, this case highlights the diagnostic challenge between AAE and FPD in PEM. Awareness of this overlap is essential, particularly in resource-limited settings, and clinicians should maintain a high index of suspicion for multiple nutritional deficiencies not only in children but also in adults presenting with severe dermatitis and malnutrition.

Ethics statement

Written informed proxy consent was obtained from the patient's caregiver.

Author contributions

Both authors contributed to conceptualization, data collection, and manuscript preparation.

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