Case Report

SEVERE KWASHIORKOR AND SCURVY SECONDARY TO AVOIDANT/RESTRICTIVE FOOD INTAKE DISORDER (ARFID) IN A CHILD WITH PREVIOUSLY UNDIAGNOSED AUTISM SPECTRUM DISORDER: A CASE REPORT

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Abstract

Background: Avoidant/Restrictive Food Intake Disorder (ARFID) is highly prevalent in children with Autism Spectrum Disorder (ASD). While selective eating is common, Severe Acute Malnutrition (SAM) manifesting as Kwashiorkor in non-famine settings is rare. This report highlights a life-threatening presentation of nutritional deficiency driven solely by behavioral restriction.

Case Presentation: A 6-year-old Thai boy presented with generalized edema, desquamating rash for 2 month. History revealed a 1-year duration of extreme food selectivity, consuming only specific brands of rice crackers and refusal of solids, coinciding with developmental regression. Physical examination revealed anasarca, "flaky paint" dermatosis, and perifollicular hemorrhages. Anthropometry showed severe wasting masked by edema. Laboratory investigations confirmed hypoalbuminemia (2.4 g/dL), anemia, and undetectable Vitamin C levels (<0.10 mg/L). Developmental assessment revealed deficits in social communication and restrictive interests, confirming a diagnosis of ASD. The patient was diagnosed with Kwashiorkor, Scurvy, and ARFID. Management involved a multidisciplinary approach including gradual nutritional rehabilitation to prevent refeeding syndrome, vitamin supplementation, and behavioral therapy initiated with low-dose risperidone. The patient showed significant dermatological and physical recovery within 1 month.

Conclusion: Severe nutritional complications can arise from undiagnosed neurodevelopmental disorders. Clinicians must maintain a high index of suspicion for ASD and ARFID in children presenting with unexplained malnutrition. Early identification and multidisciplinary management are essential to prevent irreversible developmental sequelae. ASEAN Journal of Psychiatry, Vol. 28 (1) January, 2026; 1-5.

Keywords: Autism Spectrum Disorder (ASD); Avoidant/Restrictive Food Intake Disorder (ARFID); Kwashiorkor; Scurvy; Malnutrition; Flaky Paint Dermatosis

Introduction

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition characterized by social communication deficits and restricted, repetitive behaviors [1]. Up to 80% of children with ASD exhibit atypical feeding behaviors, ranging from food refusal to severe selectivity based on texture or brand [2,3]. In 2013, the DSM-5 introduced Avoidant/Restrictive Food Intake Disorder (ARFID) to describe restrictive eating that leads to significant nutritional deficiency or weight loss, independent of body image concerns [4].

While failure to thrive is a known complication of ARFID, the development of Kwashiorkor (edematous malnutrition) is exceptionally rare in middle-income countries like Thailand, where food availability is not the primary constraint [5]. Furthermore, specific micronutrient deficiencies like Scurvy (Vitamin C deficiency) are often overlooked in the modern era [6]. This case

report describes a 6-year-old boy who presented with severe Kwashiorkor and Scurvy secondary to ARFID, leading to the first diagnosis of his underlying autism spectrum disorder.

Case Presentation

Patient information

A 6-year-old Thai boy from Kamphaeng Phet province presented with a 2 months history of progressively decreasing food intake and recent onset of generalized swelling and skin lesions.

The patient had been developmentally delayed in language part and lack of social skill. He had no history of other chronic medical conditions. At age of 5 years, he stopped attending school since the COVID-19 pandemics.

The parents reported that for the past year, the child refused all solid meals (rice, meat) and milk. His diet consisted exclusively of specific brands of crispy rice crackers and corn snacks (estimated <500 kcal/day). He accepted only a small amount (50 ml twice daily) of blended vegetable soup containing potato, carrot, pumpkin, tomato and broccoli. He exhibited significant weight loss (approx. 3 kg/month), social withdrawal and loss of speech (speaking only monosyllables). He also suffered from severe insomnia, sleeping only from 20:00 to 22:00 (2 hours). Two months prior to admission, he developed generalized edema and extensive skin peeling. The father brought him to the hospital after observing decreased snack consumption and the progression of the skin rash.

Physical examination

Vital signs were stable but tachycardic (HR 122 bpm). Anthropometry revealed a body weight of 13.5 kg, height of 102 cm and BMI of 13 kg/m^2. Head circumference was 42 cm (<3rd percentile), indicating microcephaly. His weight and height were below 3rd percentile of age and sex specific Thai reference standards, which z-score of weight for age was -3.38, z-score of height for age was -2.92 and z-score of BMI for age was -2.07, indicated severe underweight, severe stunning, and thinness.

Generalized physical examination revealed pale conjunctivae, puffy eyelids, and generalized pitting edema. Classic "flaky paint dermatosis" characterized by hyperpigmented, desquamating patches on the back, trunk and extremities with poor skin tugor (Figure 1 and Figure 2). Perifollicular hemorrhages were noted on the limbs. Conscious but non-verbal; muscle strength was grade 4 in all extremities.

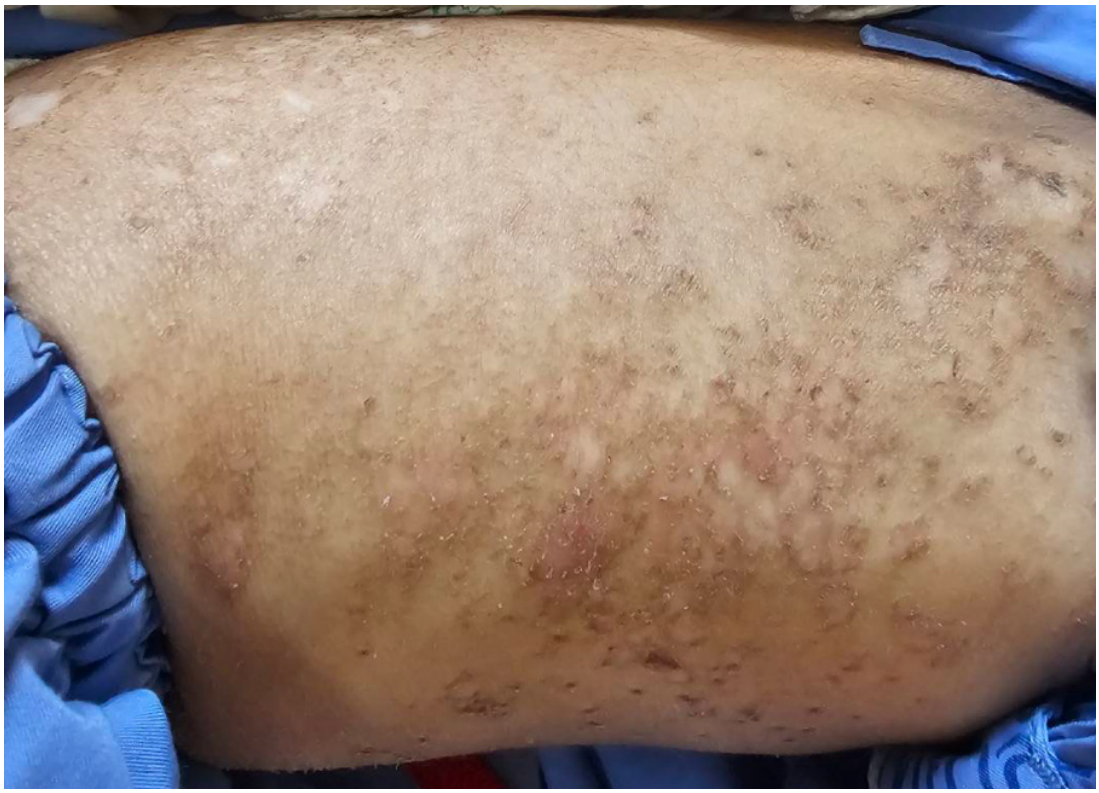

Figure 1. Flaky paint dermatosis at back and trunk (left side).

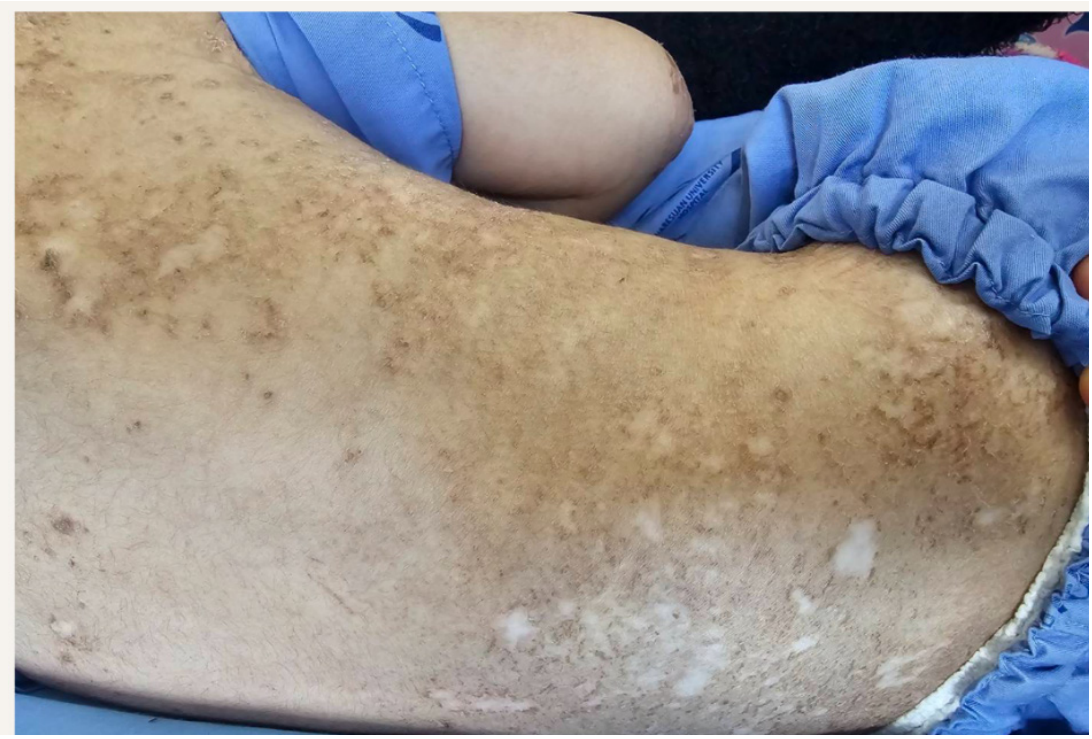

Figure 2. Flaky paint dermatosis at back and trunk (right side).

Laboratory investigations

Hematological profiling indicated microcytic anemia (Hb 9.4 g/dL, Hct 25.9%, MCV 70.8 fL, RDW 17.6%) with significant eosinophilia (12.7%) and normal platelet count. Biochemical investigations revealed severe hypoalbuminemia (2.4 g/dL) and hypoproteinemia (Total Protein 4.3 g/dL, Globulin 1.9 g/dL). Liver function tests demonstrated conjugated hyperbilirubinemia (Total Bilirubin 1.84 mg/dL, Direct 1.73 mg/dL) and elevated liver enzymes (AST 49 U/L, ALT 39 U/L, ALP 156 U/L), while coagulation profiles were within normal limits (INR 1.14). Renal function was preserved (BUN 2.5 mg/dL, Creatinine 0.31 mg/dL). Electrolytes showed hypocalcemia (7.8 mg/dL) and progressive hypokalemia, declining from 3.7 mmol/L to 2.1 mmol/L. Crucially, micronutrient assessment confirmed multiple deficiencies, notably low Vitamin A (0.22 µmol/L) and undetectable Vitamin C levels (<0.10 mg/L), confirming a diagnosis of Scurvy, whereas Vitamin D levels were sufficient (46.8 ng/mL). Additionally, neuroimaging *via* CT brain revealed diffuse mild cerebral atrophy with prominent ventricular systems, a finding secondary to severe chronic malnutrition.

Diagnostic assessment

Developmental screening using the M-CHAT indicated a high risk for autism spectrum disorder (failed 12 items). Subsequent clinical observation confirmed the diagnosis, evidenced by deficits in social reciprocity and restrictive interests (e.g., fixation on specific snack packaging).

The patient was diagnosed with Kwashiorkor, vitamin A deficiency, Scurvy and Autism Spectrum Disorder (ASD) with Avoidant/Restrictive Food Intake Disorder (ARFID). Based on the M-CHAT screening, the patient failed 12 items, indicating a high risk for ASD. Further evaluation confirmed that he met the DSM-5 diagnostic criteria for Autism Spectrum Disorder (ASD). Specifically, he demonstrated persistent deficits in social communication and interaction (Criterion A), evidenced by a lack of social initiation, preference for solitary play, avoidance of eye contact, and absence of peer relationships. He also exhibited restricted, repetitive patterns of behavior (Criterion B), manifesting as repetitive play with specific toys and extreme food selectivity (consuming only puffed rice snacks and vegetable soup). Additionally, the patient met the criteria for Avoidant/Restrictive Food Intake Disorder (ARFID). He presented with significant nutritional deficiency (severe acute malnutrition) resulting from a persistent eating disturbance. This restriction was not attributable to a lack of available food or cultural practices, nor was it associated with the body weight or shape concerns

typical of Anorexia Nervosa or Bulimia Nervosa. Consequently, the patient was diagnosed with ASD co-occurring with ARFID.

The patient was admitted for nutritional rehabilitation adhering to the Clinical Practice Guidelines for the Care of Children with Severe Acute Malnutrition [7], with strict monitoring to prevent refeeding syndrome. Throughout the admission, the patient did not exhibit any clinical features of refeeding syndrome. During the stabilization phase (Days 1-7), Due to severe oral aversion, enteral feeding was initiated *via* a Nasogastric (NG) tube using a lactose-free formula. Concurrent management included the correction of electrolyte imbalances and the administration of empirical antibiotics (ceftriaxone 75 mg per kg per day, totally 7 days) according to the guideline. Micronutrient deficiencies were aggressively treated with Thiamin (100 mg per day, orally), Vitamin A (200,000 IU at day 1, 2 and 14, orally), Zinc (20 mg per day, orally), and high-dose Vitamin C (100 mg three times daily). Upon transitioning to the rehabilitation phase, caloric intake gradually titrated to 1,300 kcal/day. To address behavioral rigidity and facilitate food acceptance, low-dose risperidone (0.1 ml HS) was initiated, alongside behavioral therapy implemented by a multidisciplinary team comprising a pediatrician, psychiatrist, nutritionist, and occupational therapist.

By week 2, the edema had resolved, and the "flaky paint" rash had significantly improved with re-epithelialization. The patient began to accept small amounts of soft rice with pork orally, though NG tube feeding continued to ensure adequate intake. After 19 days of hospitalization, he was discharged with a body weight of 14.9 kg. At the 15-month follow-up, the patient's weight had increased markedly to 27.5 kg. This rapid weight gain suggests an aggressive rebound effect, necessitating monitoring for metabolic complications. Despite this weight gain, the patient remained dependent on enteral feeding due to exacerbated oral aversion. Nutritional rehabilitation was further hindered by suboptimal caregiver compliance, as the patient was frequently offered snacks instead of adhering to the recommended feeding therapy. Conversely, regarding development, the patient demonstrated marked improvement in social engagement."

Discussion

This case illustrates the "perfect storm" of undiagnosed neurodevelopmental pathology leading to life-threatening physical illness. While picky eating is a common complaint in pediatrics, the severity, in this case, meets the DSM-5 criteria for ARFID: significant weight loss, nutritional deficiency, and dependence on enteral feeding [4].

Children with ASD are at a five-fold increased risk of feeding problems compared to neurotypical peers [2]. The drivers in this patient were likely sensory sensitivity (texture aversion) and rigid insistence on sameness (brand-specific snacks), which are hallmarks of ASD-associated ARFID [8]. The delayed diagnosis of ASD until age 6 contributed to the chronicity of malnutrition, as the parents lacked strategies to manage behavioral rigidity.

The patient presented with flaky paint dermatosis, A pathognomonic sign of Kwashiorkor caused by severe protein deficiency, skin barrier breakdown, and hypopigmentationz. Concurrently, the patient had Scurvy, evidenced by perifollicular hemorrhage and undetectable Vitamin C levels. This highlights that a diet high in carbohydrates (rice crackers) but void of fresh produce can sustain caloric needs temporarily while leading to profound micronutrient depletion [6-10].

Treating ARFID in the context of ASD is complex. Standard refeeding can be met with extreme behavioral resistance. In this case, the use of low-dose antipsychotics (risperidone) combined with behavioral modification (positive reinforcement) and nasogastric feeding was necessary to break the cycle of starvation [9]. The finding of brain atrophy on CT underscores the critical impact of chronic malnutrition on the developing brain, emphasizing the need for early intervention.

Conclusion

Malnutrition in the pediatric population should trigger a developmental assessment when organic causes are absent. This case emphasizes that ARFID in children with ASD can progress to life-threatening Kwashiorkor and Scurvy. A multidisciplinary approach involving pediatricians, nutritionists, and child psychiatrists is vital for successful management. Early screening for ASD in children with "picky eating" may prevent such severe sequelae.

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