Paediatrica Indonesiana

p-ISSN 0030-9311; e-ISSN 2338-476X; Vol.65, No.3(2025). p.260-7 ; DOI: https://doi.org/10.14238/pi65.3.2025.260-7

Case Report

Difficulty walking as the main manifestation of scurvy in children: two cases from a resource-limited setting

Angelina Angelina[1], Ruthie Aviana[2], Yolanda Ardelia[1], Arfianti Chandra Dewi[3], Ratno Juniarto Marulitua Sidauruk[4]

Scurvy is a disease caused by a prolonged deficiency of vitamin C. It was first reported in Egypt in 1500 before century (BC) and formally described by the writings of Hippocrates as "the mouth feels bad, the gums are detached from the teeth, blood runs from the nostrils, black-colored ulcerations frequently appear on the legs, some of these heal, others not, and the skin is thin."[1,2] Scurvy has become an uncommon disease nowadays, especially in developed countries. However, there have been sporadic case reports of scurvy in children. One of the largest cohorts of scurvy cases in children took place in Thailand in 2003, where 28 children diagnosed with scurvy manifested with a limp or inability to walk.[3] **[Paediatr Indones. 2025;65:260-7; DOI: https://doi.org/10.14238/pi65.3.2025.260-7**].

Keywords: *scurvy; vitamin C; pesudoparalysis; difficulty walking*

The early manifestations of scurvy are nonspecific and associated with various organ systems. It may overlap with other systemic diseases, such as rheumatologic, infectious, or hematologic disorders.[4,5] A thorough anamnesis of pediatric dietary history is crucial to establishing a scurvy diagnosis. Due to its rarity of occurrence and lack of understanding about its pathogenesis and clinical manifestations, the consideration of scurvy as a diagnostic is often overlooked, resulting in delayed diagnosis and fatal complications. Here we report two cases of children with scurvy from our hospital in a resource-limited setting. Both children were unable to walk due to intense pain in both of their legs. Patient 1 had previously been suspected of having malignancy or juvenile idiopathic arthritis. The knowledge we learned from Case 1, which occurred only 2 months earlier, helped identify scurvy in Case 2.

Case 1

A previously healthy 4-year-old girl, 17 kg of body weight (0 < Weight-for-Age Z-score/WAZ < 1 SD, WHO 2006), 102 cm of body height (-2 < Height-for-Age Z-score/HAZ < 0 SD, WHO 2006) presented to our outpatient department with difficulty walking for two months. At first, she was limping, but then she felt so much pain that she was reluctant to walk, sit, or even move her legs. There was no history of trauma, prolonged fever, or pain in other parts of the body. She had multiple cavities and swollen gums with recurrent spontaneous bleeding for 1.5 months. She looked pale, and there were some bruises on both of her feet. On physical examination, she was fully alert, and her vital signs were normal. There were no lower limb deformities, but she maintained

From Child Health Department, Faculty of Medicine, Universitas Pelita Harapan[1], Clinical Pediatrics Department[2] and General Practice Department[3], Balaraja General Hospital, Tangerang, Banten; Pediatric Imaging Consultant, Bunda Jakarta Hospital, Jakarta[4], Indonesia.

Corresponding author: Angelina. Child Health Department, Faculty of Medicine, Universitas Pelita Harapan, Tangerang. Jendral Sudirman 20, Bencongan, Kelapa Dua, Tangerang, Banten. Email: angelina.now@gmail.com.

Submitted March 21, 2023. Accepted June 12, 2025.

flexion contractures over bilateral hips and knees. The muscles were atrophied, and the range of movements was very limited due to severe pain. Oral cavity examination revealed cavities with pulpitis, radix gangrene, gingiva hyperplasia, and bleeding in almost every region (**Figure 1**). The other physical examinations were unremarkable.

Her laboratory examination showed microcytic hypochromic anemia (Hb 6.9 g/dL), normal white blood cell count (5,900/μL), thrombocytosis (562,000/μL), and high ESR (70 mm/hour). The examination of peripheral blood smears revealed hypochromic microcytic and mild anisopoikilocytosis in red blood cells, teardrop cells, pencil cells, target cells, spherocyte cells, and an increase in the number of platelet count. The prothrombin time (PT) and activated partial thromboplastin time (aPTT) were both within normal ranges. The rheumatoid factor was negative. A radiograph of the bilateral knee showed osteopenia, periosteal reaction due to subperiosteal hemorrhage, the white line of Frankel at the metaphysis, the scorbutic zone of Trummerfeld beneath the line of Frankel, and the Wimberger ring sign around the epiphysis (**Figure 2**). The patient was strongly suspected of scurvy based on clinical manifestations and radiologic findings. Vitamin C serum level measurement was unavailable in our surrounding area. From further anamnesis, it was known that she refused to eat fruits and vegetables since she was two years old and frequently drank sweetened condensed milk products one or two times a day. She typically ate rice with similar side dishes like fried chicken and nuggets. Her parents noticed that she hadn't eaten any fruits or vegetables for the previous two years.

She was started on parenteral vitamin C 100 mg/day for a week, paracetamol as an analgetic, and vitamin D 1000 IU/day. A dental expert performed some oro-dental procedures on her, such as root planning, deep scaling, gingivectomy, and debridement. She was able to extend her legs after 4 days of treatment with vitamin C, and after 6 days of treatment, she was able to sit by herself. The patient was also referred to the medical rehabilitation department for physical therapy. She was discharged on day 11 of hospitalization and vitamin C 100 mg/day was continued orally. After two weeks, she returned to our outpatient clinic and was able to

Figure 1. Oral cavities showed pulpitis, gingiva hyperplasia (orange arrow) and hemorrhages (blue star)

Figure 2. Radiographs of the both knees showed White line of Frankel (A), scorbutic zone of Trummerfeld (B), Wimberger ring (C), and periosteal reaction due to subperiosteal haemorrhage (D)

stand. She was able to walk again after another two weeks. We temporarily maintained the vitamin C supplementation because changing her eating habits required long-term monitoring.

Case 2

A 4-year-old boy presented to our emergency department with a loss of appetite for one week. The patient appeared thin and pale. There was no prior history of fever, respiratory, gastrointestinal, or genitourinary problems. He had poor oral hygiene and recurrent spontaneous gum bleeding for a month. A thorough anamnesis revealed that he had also had difficulty walking one month prior to admission. He suffered from episodic pain in both of his legs since a year ago, but the pain was getting worse and more persistent in the last month, especially when he stretched his legs. He refused to walk and kept his legs in flexion most of the time (**Figure 3**). There was no history of trauma before. His nutritional history indicated that he was a highly picky eater. Since he was a year old, his diet has primarily consisted of sweet bread, doughnuts, sausages, and nuggets. He was given chocolate-flavored UHT milk as a supplement and refused to consume all kinds of fruits or vegetables, even fruit-flavored beverages.

On examination, he looked irritable, easily cried when approached, and had generalized muscle wasting. He was slightly tachycardic, but other vital signs were within normal limits. He weighed 10.6 kg (WAZ < -3 SD, WHO 2006), and his height was approximately 103 cm (-2 < HAZ < 0 SD, WHO 2006). The nutritional status was classified as severe malnutrition (WHZ < -3 SD, WHO 2006). Remarkable physical findings were gingival enlargement with hemorrhage, cutaneous manifestations of follicular hyperkeratosis with corkscrew hairs (**Figure 4**), and bilateral knee edema with flexion stiffness. There was no organomegaly or swollen lymph nodes. A complete blood count revealed severe anemia (Hb 3.6 g/dL), normal white blood cell (5,470/μL), and thrombocytopenia (121,000/μL). A peripheral blood smear revealed hypochromic microcytic and poikilocytosis red blood cells with teardrop cells, schistocytes, burr cells, and decreased platelet count with giant platelet. A

Figure 3. Flexion at hip and genu at initial presentation (A) and its improvement after receiving parenteral vitamin C for 7 days (B)

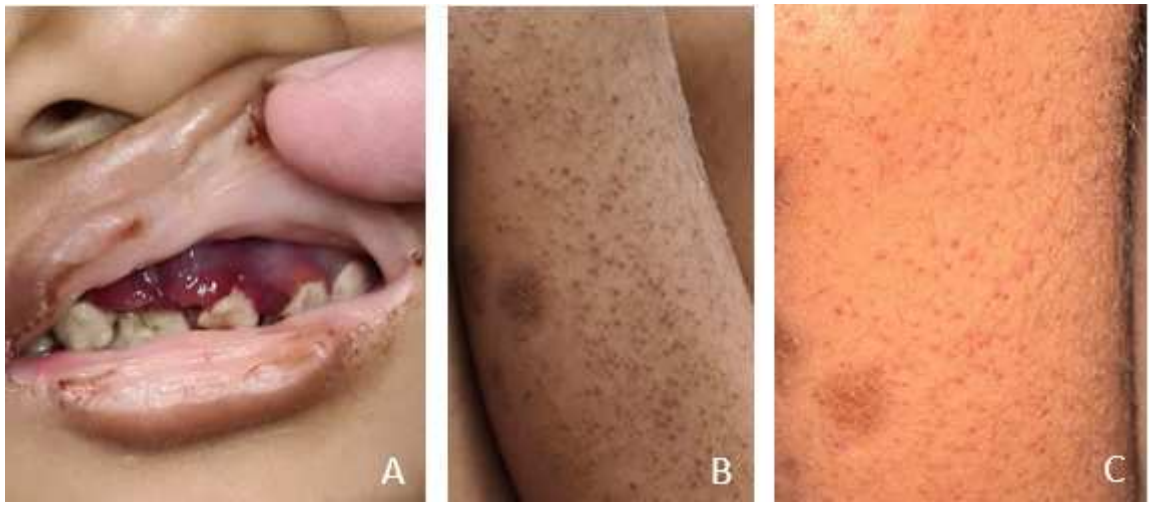

Figure 4. Gingival enlargement with haemorrhage (A) and cutaneous manifestations of follicular hyperkeratosis with corkscrew hairs (B) and its improvement after 3 days course of parenteral vitamin C (C)

lateral view of bilateral knee x-ray showed diffuse osteopenia, metaphyseal sclerotic white line of Frankel most prominent in the distal femur, and the opaque radiologic shadow of Wimberger ring around the epiphysis (**Figure 5**).

The patient was diagnosed with complicated severe malnutrition and scurvy. As mentioned in Case 1, vitamin C serum measurement was not performed due to a lack of availability in our area. Chronic severe infection was strongly considered due to bicytopenia in this patient. Further laboratory testing, which included GeneXpert MTB/RIF and urinalysis, revealed values within the normal ranges. He received a transfusion of packed red blood cells,

Figure 5. Radiographs of the lateral view of both knees showed white line of Frankel (A) and Wimberger ring (B).

the antibiotic cefotaxime, and therapeutic milk for severe malnutrition (formula 75 for the initial phase continued with formula 100). A gingivectomy and tooth extraction were performed, and in addition to routine micronutrient supplementations for severely malnourished children such as vitamin A, folic acid, and zinc, the patient was also given parenteral vitamin C 100 mg/day and vitamin D 1000 IU/day. He was able to extend his legs after receiving parenteral vitamin C for seven days. Vitamin C therapy was then switched to oral, and 3 days later, he could sit independently and stand for around 20 seconds. The patient was discharged on day 14 of hospitalization, vitamin C therapy and medical rehabilitation programs were continued in the outpatient department. When the patient visited the outpatient clinic a week later, he was able to stand up for longer, but he still refused to walk. Medical rehabilitation procedures, vitamin C supplementation, and nutritional management programs for severe malnutrition were all continued. The patient was still under our close monitoring.

Discussion

Vitamin C is an essential micronutrient that acts as a potent antioxidant and cofactor for many enzymes involved in the biosynthesis of collagen, carnitine, and catecholamines. Humans have no ability to synthesize vitamin C and there is no storage of it in human body, so adequate dietary intake is mandatory. Insufficient intake of vitamin C results in the onset of biological deficiency within 3 months and clinical signs within 6 months.[2,3,5] Certain populations of children are more prone to scurvy, such as those with dietary restrictions due to neuropsychiatric or developmental disorders. However, many recent case studies describe scurvy in healthy children without known risk factors who are picky eaters. They eat restrictive and very selective diets that are primarily carbohydrate-based, and no longer receive enriched formula milk. Fresh fruits and vegetables are not consumed in the vast majority of cases.[3,6-8] Both of our patients had this type of eating habit, their diets were not diversified, and mainly rich in carbohydrate sources. UHT milk is not a sufficient source of vitamin C for children because significant vitamin C destruction occurs during processing and storage. [9,10] Sweetened condensed milk is frequently mistaken for a milk beverage, despite the fact that it has a low nutritional content for children and is only recommended as a food topping or accompaniment to other foods.

The scurvy manifestations result primarily from decreased synthesis of collagen and lead to the weakening of collagenous structure in the skeleton and vasculature.[4,5] Early manifestations are nonspecific, such as irritability, appetite loss, asthenia, low-grade fever, and dermatological symptoms

like petechiae, ecchymoses, hyperkeratosis, and corkscrew hairs. Gingival lesions with inflammation, hypertrophy, and bleeding on slight pressure are more common in patients with poor dental health. Periodontal lysis is a late feature responsible for tooth loosening and potential loss of teeth. Later stages of scurvy are musculoskeletal disorders characterized by osteoporosis, bone growth deformity, and subperiosteal or intraosseous hemorrhagic lesions that mimic inflammatory diseases. The advanced stage disorders affect the general condition including psychological changes, poor wound healing, and death.[3,6]

Musculoskeletal manifestations are present in 80% of scurvy patients and are the most common reason a child seeks medical attention. Leg swelling, severe bone and joint pains, and hemarthrosis are the most common manifestations.[2,6] The children frequently appear to have difficulty walking and pseudoparalysis, with their hips and knees fixed in flexion, as our patients.[6,11] The most typical radiographic changes of scurvy occur at the distal ends of long bones, commonly at knees and ankles. Vitamin C deficiency causes osteoblasts to form a defective osteoid matrix and increases bone resorption, causing the bones to become fragile and easily fractured. Therefore, the most frequently found radiographic finding, although non-specific, is osteopenia. The x-ray revealed an appearance of "ground glass" indicates a deficient osteoid matrix and loss of trabecular projection. When compared to the medullary region, the bone cortex is thin and sharply contrasted. Bone mineralization is generally unaffected, resulting in an extensively calcified provisional zone of calcified cartilage. An irregular but thickened white line appears at the metaphysis (White line of Fraenkel), representing well-calcified cartilage. The more definite but late specific radiologic feature is a zone of rarefaction beneath Frankelline in the metaphysis, known as the Trummerfeld (German word for “field of rubble”) zone. Another finding associated with healing fractures of the Trummerfeld zone is the “beaks”, also known as Pelkan spurs, found at the periphery of the zone of metaphyseal calcification. A circular, opaque shadow in the growth center is often encircled by a white line surrounding the epiphysis, known as the Wimberger ring sign.[6]

Both of our patients had already manifested the classic scurvy clinical spectrum. However, we did not consider scurvy as a differential diagnosis in case 1 until the typical finding of bone radiography. Difficulties in diagnostic assessment and differential diagnosis of scurvy have been reported in many studies. Some studies reported a time interval of one week to two years between clinical onset and scurvy diagnosis, with one to several misdiagnoses made before reaching the correct diagnosis. The common suspected diagnoses of scurvy were leukemia, osteomyelitis/septic arthritis, and bone neoplasia.[7] Hematologic abnormalities are commonly found in scurvy, especially anemia, which may be hypochromic, normochromic, or macrocytic. The cause of anemia in scurvy could be multifactorial, resulting from bleeding, concomitant micronutrient deficiency, and decreased iron absorption.[2,4] Thrombocytopenia can be seen in some cases, like our 2nd case, and immune thrombocytopenic purpura is sometimes considered one of differential diagnosis.[6,12]

The gold standard of scurvy diagnosis is serum vitamin C measurement, which is considered deficient below 200 mcg/dL. However, the serum level does not always reflect tissue levels of vitamin C. It may be expected with recent vitamin C intake, and during the inflammatory response, vitamin C is transferred from the serum to leucocytes, resulting in a decrease in serum level.[2,5,7] Micronutrient serum level measurement, including vitamin C, is rarely available in resource-limited areas, and it is also a costly examination. Thus, the most practical and best confirmatory method for scurvy diagnosis is the resolution of signs and symptoms after vitamin C supplementation. Spontaneous bleeding, oral symptoms, and constitutional symptoms begin to improve within days, and overall improvement may be noticeable within 2 weeks. Bony changes and ecchymoses may take several weeks to resolve, and complete resolution of other clinical findings, including hematologic disorders, usually occurs within 3 months.[2,4-7] Both our patients' pseudoparalysis dramatically improved within days of their hospitalization. This quick clinical response may be considered as an ex-juvantibus diagnosis criteria.[13]

There are no standardized treatment regimens for scurvy. Doses for children range from 100-300 mg to 1000 mg daily for one month or until full recovery. The usual treatment, based on many case studies, is 1 g/day for 2 weeks or 100-200 mg/day for a longer

period to achieve complete recovery. When intake exceeds 100 mg/day, the intestinal absorption and renal excretion mechanisms become saturated, suggesting the use of divided doses spread throughout the day. Parenteral administration is required in patients with malabsorption.[2,4,5,7] We gave our patients 100 mg of parenteral vitamin C per day because they were losing appetite and the oral manifestations made feeding more difficult. As the clinical symptoms improved, the treatment was changed to oral administration.

Other associated micronutrient deficiencies may concomitantly occur in scurvy. The most frequently observed was vitamin D deficiency (60%), followed by vitamin B1, B6, and A (9%, each one).[5,7,11] The prevalence of vitamin D deficiency is considered high throughout the world, even in sun-rich countries. We did not measure our patients' vitamin D serum levels due to unavailability in our hospital, but based on their dietary history and radiological findings of osteopenia, we assumed that they were at high risk of vitamin D deficiency. Thus, we also gave routinely recommended doses of 1000 IU/day vitamin D to our patients.[14] In spite of the clinical improvement of our patients after vitamin C therapy, they still need long-term management to change their dietary patterns, as prevention of underlying conditions is necessary to prevent the recurrence of scurvy.[4,15]

Several limitations should be considered in our study. The unavailability of vitamin C level measurement is a major limitation. An unknown serum level of vitamin C may prolong the unnecessarily high dose and long duration of parenteral vitamin C therapy. Intravenous vitamin C should be limited to those only who cannot tolerate oral vitamin C such as in patient with malabsorption and it should be change to oral vitamin C as soon as possible.The behavior modification therapy for dietary changes is a principal management of scurvy and needs long-term monitoring to prevent disease recurrence. Therefore, prevention education about scurvy and good feeding practices for infants, toddlers, and young children should be provided in high-risk population areas. Exploration of how the restricted diet evolved to scurvy should be taken in every feeding history anamnesis. The micronutrient problems should be considered as important as macronutrients.

In conclusion, scurvy should be considered as a differential diagnosis in children with difficulty walking. Anamnesis of inappropriate eating habits, knowledge of other organ system manifestations of scurvy (dermatological, oro-gingival, and hematological), and typical bone radiography findings are important clues to establish the diagnosis in a resource-limited setting. The rapid remission of clinical manifestations following vitamin C supplementation is used to confirm the diagnosis.

Conflict of interest

None declared.

Acknowledgements

We would like to thank our orthopedic surgeons, Henrico Marindian and Oktavian Irawadi Purba, as well as our dental specialist, Devi Oktora, for their assistance in caring for our scurvy patients.

References

1. Magiorkinis E, Beloukas A, Diamantis A. Scurvy: past, present and future. Eur J Intern Med. 2011;22:147-52. DOI: https://doi.org/10.1016/j.ejim.2010.10.006
2. Fain O. Musculoskeletal manifestations of scurvy. Joint Bone Spine. 2005;72:124-8. DOI: https://doi.org/10.1016/j.jbspin.2004.01.007
3. Chalouhi C, Nicolas N, Vegas N, Matczak S, Jurdi H, Boddaert N, *et al.* Scurvy: a new old cause of skeletal pain in young children. Front Pediatr. 2020;8:1-5. DOI: 10.3389/fped.2020.00008
4. Alqanatish JT, Alqahtani F, Alsewairi WM. Childhood scurvy: an unusual cause of refusal to walk in a child. Pediatric Rheumatology. 2015;13:23. DOI: https://doi.org/10.1186/s12969-015-0020-1
5. Kitcharoensakkul M, Schulz CG, Kassel R, Khanna G, Liang S, Ngwube A, *et al*. Scurvy revealed by difficulty walking: Three cases in young children. Journal of Clinical Rheumatology. 2014;20:224-8. DOI: https://doi.org/10.1097/RHU.0000000000000101
6. Agarwal A, Shaharyar A, Kumar A, Bhat MS, Mishra M. Scurvy in pediatric age group - A disease often forgotten? J Clin Orthop Trauma. 2015;6:101-7. DOI: https://doi.org/10.1016/j.jcot.2014.12.003

7. Trapani S, Rubino C, Indolfi G, Lionetti P. A Narrative review on pediatric scurvy: the last twenty years. Nutrients. 2022;14:684. DOI: https://doi.org/10.3390/nu14030684
8. Noble JMC, Mandel A, Patterson MC. Scurvy and rickets masked by chronic neurologic illness: Revisiting "psychologic malnutrition"? Pediatrics. 2007;119:783-90. DOI: https://doi.org/10.1542/peds.2006-1071
9. Goldsmith SJ, Eitenmiller RR, Toledo RR, Barnhart Hm. Effects of processing and storage on the water-soluble vitamin content of human milk. J Food Sci. 1983;48:994-5. DOI: https://doi.org/10.1111/j.1365-2621.1983.tb14951.x
10. Holmes Ad. Stability Of Ascorbic Acid In Milk At Different Stages Of Processing. J Pediatr. 1952;40:91-5. DOI: https://doi.org/10.1016/S0022-3476(52)80238-0
11. Ceglie G, Macchiarulo G, Marchili MR, Marchesi A, Aufiero LR, Camillo C, *et al*. Scurvy: still a threat in the well-fed first world? Arch Dis Child. 2019;104:381-3. DOI: https://doi.org/10.1136/archdischild-2018-315496
12. Perry M, Page N, Manthey D, Zavitz J. Scurvy: dietary discretion in a developed country. Clin Pract Cases Emerg Med. 2018;2:147-50. DOI: https://doi.org/10.5811/cpcem.2018.1.36860
13. Brambilla A, Pizza C, Lasagni D, Lachina L, Resti M, Trapani S. Pediatric scurvy: When contemporary eating habits bring back the past. Front Pediatr. 2018;6:126. DOI: https://doi.org/10.3389/fped.2018.00126
14. Ikatan Dokter Anak Indonesia. Panduan Praktik Klinik IDAI Vitamin D. Jakarta: BP IDAI: 2018.
15. Hahn T, Adams W, Williams K. Correction: Is vitamin C enough? A case report of scurvy in a five-year-old girl and review of the literature. BMC Pediatr. 2019;19:1-6. DOI: https://doi.org/10.1186/s12887-019-1437-3