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Case Report

Scurvy in Children: A Case Report

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Abstract

Scurvy is all the symptoms associated with a diet deficient in ascorbic acid or vitamin C, found in fresh fruit and vegetables. Scurvy is a pathology resulting from a deep and prolonged vitamin C deficiency, which can manifest itself as a bleeding syndrome, wound healing disorders, rheumatic signs, or gum damage. If left untreated (vitamin C supplementation), the disease can be fatal. This water-soluble vitamin is neither synthesized nor stored in the body. In recent years, there has been a resurgence of this pathology due to unbalanced nutrition. Clinical presentations are deceptive and variable, with orthopedic abnormalities and a diffuse hemorrhagic syndrome secondary to a defect in collagen fiber synthesis in children. The diagnosis of scurvy is confirmed by measuring ascorbemia, which is less than 2 mg/L (5-15 mg/L or 17-94 mmol/L), and treatment is based on vitamin C supplementation at 100 to 300 mg/day until complete recovery. We report the case of a 7-year-old patient with an autistic spectrum disorder associated with epilepsy, who presented with scurvy revealed by bone involvement consisting of bone pain, a bleeding syndrome and gingival hypertrophy in context of altered general condition. The diagnosis was confirmed by a collapsed ascorbic acid level (<3 mmol/l) and progressed favorably on vitamin supplementation.

Keywords

Ascorbic Acid, Bleeding Syndrome, Scurvy, Vitamin C

1. Introduction

Scurvy, or Barlow's disease, is the classic and severe form of major vitamin C deficiency. It was identified in the 15th and 16th centuries as a disease affecting sailors who had no access to fresh food, particularly vegetables or fruit, during their long voyages. Long before the discovery of vitamins, the English navy was in the habit of supplying lemons and other citrus fruits to sailors. Today, scurvy is very rare in industrialized countries, especially in children [1]. We report a case of scurvy in children, in order to recall the clinical and radiological manifestations of this historic and sometimes forgotten disease.

2. Case Report

We report the case of a 7-year-old male child treated for autism spectrum disorder and generalized epilepsy on sodium Valproate. The child presented with a selective eating disorder consisting solely of dairy products, with no intake of fruit or vegetables. Two weeks prior to hospitalization, he presented with a hemorrhagic syndrome consisting of gingivorrhagia, hemorrhagic gingival hypertrophy and petechial purpura associated with bone pain in the lower and upper limbs and follicular keratotic papules, all evolving in a context of altered general condition.

On admission, the child appeared pale, painful and mal-

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nourished. He was prostrate, refusing to move. Joints were stiff and muscle palpation painful, with hemarthrosis of the left knee and left shoulder, enlarged and hemorrhagic gingiva (figure 1), right peri-ocular ecchymosis associated with diffuse petechial purpura, especially on the lower limbs (figure 2).

The blood count showed regenerative normocytic normochromic anemia (hb 5.3 g/dl, VGM 89 fl, TCMH 34 fl), a disturbed hemostasis panel with a low PT of 34% and a prolonged APTT of 50.7 seconds, and a deficiency of vitamin K-dependent factors (Fact II 18%, Fact X 16%).

The myelogram showed hyperplasia of the erythroblastic lineage, with giant metamyelocytes and signs of dysplasia.

Ultrasound of the soft tissue revealed a small effusion in the left knee and superficial subcutaneous soft tissue infiltration, with no evidence of collection in the shoulder.

Given the clinical picture, the absence of primary haemostasis disorders and the eating disorders, scurvy was suggested after ruling out malignant pathology. The vitamin C blood test showed a level of < 3 mmol/L (normal = 17-94 mmol/L). The diagnosis of scurvy was therefore confirmed, and vitamin C supplementation of 1 gr/day was initiated. One week later, the bleeding syndrome had disappeared, gingival hypertrophy had regressed and joint mobilization was less painful.

In fact, this child's diet has always been completely unbalanced, based on UHT milk combined with cookies and starchy foods with no fruit or vegetables.

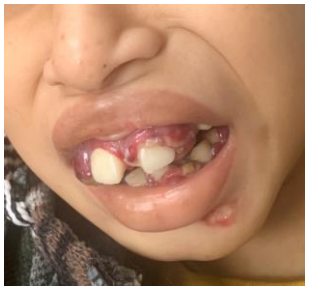

Figure 1. *Gingival hypertrophy.*

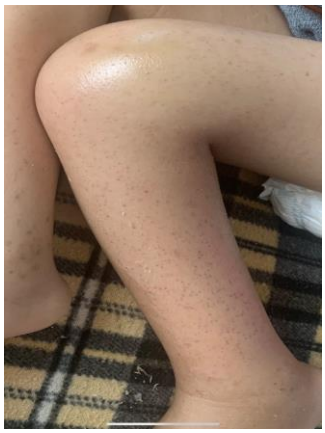

Figure 2. *Purpura and keratosis.*

3. Discussion

This clinical case illustrates the characteristics of scurvy and provides an opportunity to discuss the circumstances in which vitamin C deficiency may occur. Diagnosis is essentially clinical, based on the triad of deficient diet, symptom onset and resolution.

Ascorbic acid is a water-soluble vitamin, an essential metabolic molecule that is neither synthesized nor stored in the body [2], and a cofactor in the assembly of the collagen triple helix. Its deficiency therefore affects tissues that owe their three-dimensional cohesion to functional collagen fibers [3].

Scurvy, or "Barlow's disease", dates back to ancient times, and was first described in the 15th and 16th centuries among seafaring voyagers [4]. Today, scurvy mainly affects isolated or institutionalized elderly people, alcoholics, people suffering from psychiatric disorders and famine-stricken populations [2].

This deficiency only exceptionally affects pediatric populations in developed countries [5]. Scurvy in children has been reported to be associated with health problems (including malabsorption, iron overload, food allergies, dialysis-treated renal failure, and malignancy), neurodevelopmental disorders and psychiatric disorders [6-9].

Anecdotal reports have linked it to eating disorders, autism, and cerebral palsy [5].

As humans are unable to synthesize vitamin C from glucose, daily intakes from fresh fruit and vegetables are necessary. It is a water-soluble vitamin, most of which is absorbed from the ileum [10, 11]. Vitamin C is an essential cofactor for proline and lysine oxidase, which are involved in procollagen biosynthesis. Ascorbic acid deficiency induces alterations in blood vessel walls and connective tissue, with abnormal formation of the basement membrane, responsible for the hemorrhagic manifestations of the disease through extravasation of red blood cells: purpura, digestive hemorrhages, hemarthroses, subungual hemorrhages, perifollicular hemorrhages.... [12]. The latter particularly affect the legs. Hemorrhagic gingivitis and bone pain could suggest acute myeloid leukemia, which was easily ruled out by the blood count.

The first symptoms of scurvy are vague and non-specific [9, 13]. These include lethargy, irritability, joint and muscle pain [9, 14], follicular hyperkeratosis and bruising/bleeding may occur as early as 3 months after inadequate vitamin C intake.

The classic dermatological signs of scurvy are present in our observation: disseminated purpura and follicular keratotic papules. Perifollicular hemorrhages are secondary to the extravasation of red blood cells and their accumulation in weakened connective tissue, with hemosiderin deposits and chronic inflammation. Disturbed collagen synthesis is responsible for keratin abnormalities, leading to hair brittleness. Dilatation of the hair follicle and formation of a keratin plug trap the hair shaft, giving it a "corkscrew" appearance [15].

In Barlow's disease or infantile scurvy, rheumatological

manifestations (linked to subperiosteal haematomas) are very often in the foreground and are early onset, as shown in our case [11].

Anemia is another feature of scurvy and is thought to be multifactorial [15, 16]. Iron deficiency is caused by inadequate oral intake, poor absorption (vitamin C improves systemic iron absorption) and/or increased bleeding may be a more important factor.

Scurvy affects children from 6 to 18 months of age receiving exclusive unsupplemented artificial nutrition. Our patient had no dietary source of vitamin C and no medication. His case is a good illustration of vitamin C deficiency. The diagnosis of scurvy was confirmed by ascorbemia, which was less than 2 mg/L (5-15 mg/L or 17-94 mmol/L) [11]. There is no consensus on the doses of vitamin C to be administered to treat scurvy. Some authors recommend 100-300 mg/d for children and 500-1000 mg/d for adults until complete recovery [4, 12].

Improvement in patients after vitamin C supplementation is rapid and dramatic [17, 18], with oral and systemic symptoms improving within a few days [13, 14]. Bone abnormalities and bruising may take several weeks to disappear. Complete healing takes 2 to 3 months.

The diagnosis of scurvy relies on the integration of nutritional history and clinical features with laboratory and radiographic findings [19]. Due to the rarity of the disease, lack of experience and nonspecific clinical signs, physicians may not immediately suspect scurvy [20].

Clinical improvement in response to vitamin C treatment may help to confirm the clinical diagnosis while awaiting laboratory tests.

4. Conclusions

Although scurvy is nowadays considered by most to be a historical pathology, we have been able, through this observation, to show that the disease has not disappeared. It affects well-defined populations. Scurvy is found in poor, isolated populations where the diet is not varied, but also in people with eating disorders. The diagnosis of scurvy should be made in the presence of the following symptoms.

Author Contributions

Siham Satlane: Conceptualization, Resources
Imane Chahid: Supervision, Validation, Methodology
Fatima Harim: Resources
Meriem Atrassi: Resources
Dalal Bensabbahia: Resources
Abdelhak Abkari: Project administration

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Data Availability Statement

The data supporting the outcome of this research work has been reported in this manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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