Fruits and Vegetable Intake, Any Relation with Purpuric Lesions?

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Abstract

We are reporting the case of a 72 year old male patient with no previous relevant medical history presenting with a one week history of non-palpable purpuric rash with normal platelet count. All investigations came back negative except for vitamin C. The resolution of the clinical picture was achieved after vitamin C supplementation was added to prednisone.

Keywords: Male patient; Purpuric rash; Platelet count

Case Report

A 72 year old man was admitted to the hospital due to the recent onset of a rash in upper extremities. The lesions appeared the week prior to admission and gradually spread the abdomen and lower limbs. He did not report any fevers, appetite loss or weight loss. He is a nonsmoker and drinks a glass of wine with dinner 2-3 nights a week. He is retired. Her medical history is free of chronic illness, and his family history is of no interest. No exposure to the physical examination revealed a healthy appearing obese patient. Heart auscultation was normal and lung sounds were clear. He had no axillary, cervical or inguinal adenopathy. Abdominal examination was negative with no tenderness or mass.

Figure 1: Not palpable purpuric lesions in both legs.

Non palpable purpuric lesions (Figure 1) were observed in the four extremities and abdomen. The rash was not excoriated. The rest of the physical exam was irrelevant. A complete blood cell count showed normal hemoglobin and hematocrit levels, platelet count as well as normal white blood cell count with no blast on peripheral smear. Prothrombin time was 80%. Metabolic panel findings were normal including creatinine levels, liver and thyroid function tests. Ferritine, vitamin B12 and folates levels were all with the normal ranges. The immunologic panel came back negative for antinuclear antibody titer, rheumatoid factor; complement C3 and C4 and anti-neutrophil cytoplasmic antibodies. Cryoglobulines tests were negative. Protein electrophoresis was normal. B2 microglobulin was within the normal range. Serological tests for EBV, CMV, HBV, HCV, HIV and parvovirus returned negative. Vitamin C was 9 umol/L (normal range between 26.1 and 84). Cardiomegaly was described on the chest X-rays and abdominal ultrasound was normal. The differential diagnosis of a non-palpable non thrombocytopenic purpuric rash is wide. In this respect, the absence of blasts on the peripheral smear, normal coagulation tests, negative immunology panel and normal abdominal ultrasound allowed us to rule vascular collagen diseases, cryoglobulinemia and hematologic malignant disorders. Prednisone 1 mg/kg/d were started with no relevant clinical improvement at day 5, and then vitamin C 1 g/d during 3 days was added. After the initiation of this combination the skin lesions gradually improved.

Discussion

The vitamin C deficiency is rare condition nowadays but it is still presents even in industrialized countries. Clinicians may often encounter this condition but it is most of the time misdiagnosed. Differently from other vitamins, vitamin C is not synthesized by humans so an adequate intake of fruits and vegetables is needed [1]. Fruits and vegetables are considered the main source of vitamin C in western countries. There is no clear consensus about the daily intake of vitamin C required for a single individual, in this regard, there have been suggested values ranging from 30 to 200 mg. However, according to British essays, the minimal daily requirement to prevent scurvy is 10 mg or less [2,3].
Vitamin C deficiency may be seen at any age but specially in elderly patients, those with special diets or patients with chronic conditions such us renal chronic disease or cancer. All these situations may lead to a poor intake of vitamin C or a loss of it as in the case of patients requiring hemodialysis or suffering from malabsorption syndromes. In the case presented, the patient referred poor vegetable or fruits intake. On the other hand, alcohol abuse can also lead to scurvy as alcohol beverages contain no vitamin C and alcoholic patients are generally malnourished [4].

The diagnosis of scurvy is generally based on clinical features, dietary history, and the rapid resolution of the signs and symptoms after vitamin C supplementation. In our case, typical signs of vitamin C deficiency such as gingival hypertrophy or bleeding were absent. Purpuric lesions in the setting of vitamin C deficiency may result from blood vessel fragility. Vitamin C plays an important role in the in collagen synthesis and this element explains most of the symptoms in scurvy. Mature collagen is composed of three polypeptide molecules in a triple helix. The polypeptides lysyl and prolyl use vitamin C as a cofactor for hydroxylation. The absence of hydroxylysyl and hydroxyprolyl residues renders the polypeptide unstable and unable to self-assemble into rigid triple helices [1].

As a consequence of blood vessel fragility, patients mostly present with bruising and pedal edema. As previously mentioned, gingival hypertrophy and bleedings are the most common signs and symptoms in scurvy but the clinical spectrum is wide. Clinical features may vary from lassitude and fatigue to hemorrhage, bone disease due to subperiosteal bleeding. Follicular hyperkeratosis and fever [1,5-7]. Seizures, cerebral haemorrhage or haemopericardium have also been described [6,7].

**Conclusion**

Vitamin C deficiency is a rare and forgotten condition in industrialized countries. Diagnosis is based on clinical features and response to treatment.

**References**