

African Journal of Internal Medicine ISSN 2326-7283 Vol. 5 (5), pp. 428-432, May, 2017. Available online at www.internationalscholarsjournals.org © International Scholars Journals

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Case Report

Vitamin C deficiency and purpuric hyperkeratotic skin lesions in the elderly: What relationship? A clinical case report

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Accepted 22 March, 2017

Gingival bleeding and dental heaving during scurvy are the most reported in the literature. However nonspecific isolated purely cutaneous manifestations due to hypovitaminosis C are not uncommon. Their incidence increases with age related decrease in microcirculation of the skin causing trophic disorders observed in the skin extremities in the elderly. We report the case of an elderly vascular poly 73 years whose examination of the extremities showed purpuric hyperkeratotic lesions. The various complementary examinations for different diagnostic hypotheses have helped to spread a disease of cholesterol emboli, systemic vasculitis and retain low vitamin C after dosing incidentally the serum ascorbic acid. The disappearance of these skin lesions after two weeks of supplementation with vitamin C orally confirms the diagnosis. The appearance of mucocutaneous lesions in geriatric subject to high risk of atherosclerosis and living in unfavorable socio-economic conditions must take the clinician to look for low vitamin C and titrate the serum ascorbic acid.

Keywords: Hypovitaminosis C. Serum ascorbic acid, purpuric hyperkeratotic lesions, elderly atherosclerosis.

INTRODUCTION

Vitamin C is supplied exogenously by diet in humans, primates and some animals. Vitamin C level decrease with age, especially in the epidermis (Leveque et al., 2002; Leveque et al. 2003) is at the origin of diseases and the best known is scurvy which occurs when the value of ascorbic acid level is less than 6µmol/l. However, there are isolated specific manifestations purely Cutaneous in Hypovitaminosis C that are rarely described in the literature. They are a kind of follicular hyperkeratosis, pigment icthyosis, delays and or healing difficulties even venous ulcers (Fain et al., 2003). In addition, during human aging there is a reflex decrease in skin vasodilatation (Kellogg et al.,1995; Kenney, 1988) then of the microcirculation of the skin causing

trophic disorders observed in the skin extremities in the elderly. Furthermore vitamin C supplementation improves skin blood flow (Jennifer, 2011). We report a case of vitamin C deficiency in a septuagenarian revealed by purpuric hyperkeratotic lesions that were treated in fifteen days by vitamin C supplementation by oral route.

OBSERVATION

A 73-year-old-man, Guadeloupean of origin, bachelor and retired is addressed in internal medicine for the fortuitous discovery of a monoclonal peak with light kappa chain associated with acrorhigosis with paresthesia of the feet that has been developing for a year in a clinical picture of chronic frostbite. Personal history is hypertension known and treated since 1998 on

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quadritherapy (ACEBUTOLOL-200MG AMLOR® 5MG 3-CoAprovel® 300 / 5MG). There was a tobacco intoxication weaned in 1967. Dyslipidemia treated since 2002 by Crestor, ischemic stroke that occurred in 2002 from which it does not remain sequelae, if not a Babinski on the left. On admission, we note a blood pressure to 135 / 80mmHg and a heart rate to 76/min, pedal pulses that are frankly abolished on the left, very reduced on the right as well as tibioposterior pulses. Both femoral pulses are well perceived. There is no breath on auscultation of arterial axes but on the other hand the local state of the foot shows a very poor venous condition of the lower limbs and especially distal ischemic lesions in all areas of support points, toes and the sole of the foot associated with acrocyanosis and painful stasis with distal ulcerations. The rest of the physical examination was unremarkable.

Biologically, there was a normal blood count, normal blood and urine electrolytes with creatinine clearance decreased to 44 ml/minute.

Serologically, HIV serology, HBsAg, HCV-Ab, Syphilis, Epstein-Barr virus, HTLV-1 and HTLV2 viruses were negative, but HBS-Ab, HCV-Ab serologies are positive, which is an evidence of an old infection.

Immunologically, anti-nuclear antibodies, native anti DNA antibodies, anti Beta2 GP1 antibodies are negative and presence of anti Cardiolipin antibodies with IgM positive to 54 units.

Additional tests carried out show the existence of cataract in the right eye fundus and a clear glass with the slit lamp without cholesterol embolus but on the other hand, there is a retro-fovea serous retinal detachment of the left eye, without cholesterol embolus.

A skin biopsy of the base of the left first metatarsal, which shows a normal thickness of the epidermis, a discrete vascular dilation without vasculitis or thrombosis. The direct Immuno-Fluorescence examination is negative.

Capillaroscopy showed the absence of abnormality, organic microangiopathy.

The other morphological examinations are unremarkable except the echocardiography that has objectified a concentric LV cardiomyopathy with an ejection fraction to 65%.

Fortuitously a determination of vitamin C was required. Hypovitaminosis in ascorbic acid was found, estimated at 8.18 µmol/l (technique used: HPLC electrochemistry, laboratory standard: 45- 95 m/dl).

Supplementation with vitamin C, for a two-week period made disappear skin lesions that have been developing for about 1 year.

DISCUSSION

The degree of vitamin C deficiency should be defined. A rate higher than 26µmol/l is considered normal, a rate between 6µmol/l and 26µmol/l corresponds to

Hypovitaminosis C, which is known as severe if the rate is less than 13µmol/l. A rate lower than 6µmol/l defines a deficiency. And scurvy is defined when the rate is less than 6µmol/l associated with clinical signs. Our patient had ascorbemia to 8.18µmol/l therefore severe Hypovitaminosis C. The prevalence of Hypovitaminosis C predominates in men and increases with age. Our patient is one example.

A study of 1108 outpatients in the region of Paris (Hercberg et al., 1994) highlighted an ascorbemia less than 2 mg/l in 5% of women and in 12% of men, percentage reaching 15% of women and 20% of men after 65 years; in the united kingdom in 1970, the studies showed that 50% of elderly people living at home had ascorbemia less than 2 mg / I (Kenney, 1988).

A number of vitamin C deficiency risk factors have been identified in the study of Fain et al. in hospital: the elderly, male gender, being retired or unemployed, having an infectious disease and excessive consumption of alcohol and tobacco (Fain et al., 2003). In our case, the patient is in his seventies, single, retired and former smoker. These risk factors are factors limiting the absorption of vitamin C in the body. Moreover, the body stores of vitamin C are low (1500 mg) (Lazareth et al., 2007) so that the clinical picture of scurvy appears in one to three months of absolute deficiency of ascorbic acid when the total pool of the organism is less than 300 mg and the ascorbic acid level falls below 2 (Johnstan et al., 1998) to 2.5 mg/l (Fain et al., 2003). The patient had no scurvy but Hypovitaminosis C with severe acrocyanosis of the extremities of the lower limbs. These skin lesions of type acrorhigosis of chronic evolution (fig 1 and 2) on a constitutional susceptibility of preexisting vascular pathologies of type hypertension, ischemic stroke, Dyslipidemia, moderate renal impairment and former smoker in our patient allowed us to discuss the disease of cholesterol embolism in the presence of a hyper eosinophilia to 572 cells/mm³; but the negative search of micro-crystals of cholesterol in the urine, in the eye fundus and skin biopsy are not in favor of this diagnosis.

The search for ANCA vasculitis, connective and cryoglobulinaemia proves negative. Finally, vitamin C deficiency with ascorbemia equal to 8.18µmol/l revealed by a clinical picture of acrorhigosis with paresthesia of the feet that has been developing for a year has been accepted. Indeed Several studies show that the presence of clinical signs of deficiency leading to a determination of ascorbemia. highlights **Hypovitaminosis** systematically (Blateau, 2005; Le Bris, 2012; Malmauret et al., 2002) up to 100% in the population of Oguike (Oguike, 2014) and 93% in Sentenac (Sentenac, 2016) although it is known that skin symptoms during vitamin C deficiency is extremely polymorphic and non-specific (Fain et al., 2003). Purpuric hyperkeratotic lesions of extremities of limbs are rarely described unlike leg ulcers. Vitamin C is involved in the synthesis of collagen as an



Figure 1: view of the sole of the foot before treatment



Figure 2: view of the dorsum of the foot before treatment

essential cofactor for proline and lysine oxidase responsible for the formation of stable collagen helices. Vitamin C deficiency induces an alteration of the structure of collagen (Carr et al.,1999). There are 3 types of collagen including type 3 that is present in the skin and blood vessels. However the amount of vitamin C is low in the elderly skin, particularly in the epidermis (Leveque et al., 2002; Leveque et al., 2003) all the more as the patient is at risk for atherosclerosis as in our patient. But in patients with atherosclerosis, there is biochemical evidence that indicates the increase of oxidative stress resulting from a change in the balance of pro- and endogenous antioxidants (Kellogg et al.,1995). This oxidative stress is associated with increased consumption of vitamin C.

In short, the deficiency of vitamin C in the epidermis of the elderly associated with atherosclerosis and inadequate vitamin C intake are factors that can combine to cause such skin lesions observed in our patient.

The oxidative attack of lens proteins was also considered as a risk factor for cataract.

In the literature there is no consensus on the correlation between vitamin C status and the frequency of cataracts. Some studies evoking a relationship between poor intakes of vitamin C and cataract, and others (Vitale et al., 1993) not finding any correlation. Cataract found in our patient, however, can be related to severe low vitamin C.

The favorable outcome characterized by the disappearance of purpuric hyperkeratotic lesions (fig 3) by supplementing 1 g/day of ascorbic acid in our patient orally for two weeks retrospectively demonstrates the validity of the diagnosis.

CONCLUSION

The appearance of mucocutaneous lesions in geriatric subject with high risk of atherosclerosis and living in



Figure 3: view of the sole of the foot after treatment

unfavorable socio-economic conditions must lead the clinician to look for Hypovitaminosis C and determine the ascorbic acid level.

The determination of vitamin C is expensive and is not easy so prevention of Hypovitaminosis C by daily supplementation of the diet of the Geriatric subject by the consumption of fruits and vegetables should be advocated.

REFERENCY

- Blateau S (2005). Scorbut et déficit en vitamine C: étude rétrospective à propos de 164 cas (Thèse d'exercice). Université de Reims Champagne-Ardenne.
- Carr Ac, Frei B (1999). Toward a new recommended dietary allowance for vitamin C based on antioxydant and health effects in humans. Am J Clin Nutr; 69: 1086-1107.
- Fain O, Pariés J, Jacquart B, Le Moël G (2003). Hypovitaminosis C in hospitalized patients. Eur J Intern Med. Nov; 14(7):419-425.
- Guillaume Sentenac (2016). Le scorbut au XXIème siècle, une nouvelle maladie ? [Thèse d'exercice]. Université Rouen : UFR de médecine.
- Hercberg S, Preziosi P, Galan P, Deheeger M (1994). Vitamin status of a healthy french population: dietary intakes and biochemical markers. Int J Vitamin nutr. Res: 64:220-223.

- Jennifer J, DuPont, William B, Farquhar, Raymond R (2011). Townsend. Ascorbic acid or l-arginine improves cutaneous microvascular function in chronic kidney disease. J Appl Physiol (1985). Dec; 111(6): 1561– 1567.
- Johnstan CS, Thompson LL (1998). Vitamin C status of an outpatient population. J Am Coll Nutr;17:366-370.
- Kellogg DL Jr, Pergola PE, Piest KL, Kosiba WA, Crandall CG, Grossmann M, and Johnson JM (1995).
- Cutaneous active vasodilation in humans is mediated by cholinergic nerve cotransmission. Circ Res. 77: 1222–1228.
- Kenney WL (1988). Control of heat-induced cutaneous vasodilatation in relation to age. Eur J Appl Physiol Occup Physiol. 57: 120–125.
- Lazareth I, Hubert S, Michon-Pasturel U, Priollet P.J(2007) . Vitamin C deficiency and leg ulcers. A case control study. Mal Vasc. Apr; 32(2):96-99.
- Le Bris T(2012). Quel est l'intérêt de l'ascorbémie dans le diagnostic et le traitement
- du scorbut ? [Thèse d'exercice]. Université de Caen.UFR de médecine.
- Leveque N, Muret P, Mary S, Makki S, Kantelip JP, Rougier A, Humbert P (2002). Decrease in skin ascorbic acid concentration with age. European .J Dermatol.12: 21–22.
- Leveque N, Robin S, Makki S, Muret P, Rougier A, Humbert P (2003). Iron and ascorbic acid

- concentrations in human dermis with regard to age and body sites. Gerontology.49:117–122.
- Malmauret L, Leblanc J, Cuvelier I, Verger P (2002). Dietary intakes and vitamin status of a sample of homeless people in Paris. Eur J Clin Nutr. Avr; 56(4):313-320.
- Oguike M (2014). Scorbut et précarité, prévalence d'une maladie oubliée en médecine générale [Thèse
- d'exercice]. Université de Montpellier I. Faculté de médecine.
- Vitale S, West S, Hallfrisch J, Alston C, Wang F, Moorman C et al (1993). Plasma antioxidants and risk of cortical and nuclear cataract. Epidemiology ;4: 195-203.