Phrynoderma: a forgotten entity in a developed country

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ABSTRACT

Phrynoderma is a type of follicular hyperkeratosis attributed to various nutritional deficiencies, most notably vitamin A. We report a case of a 31-year-old mentally-deficient man who was a resident of a voluntary welfare nursing home. He presented with characteristic hyperkeratotic follicular papules on his trunk in a setting of low serum level of vitamin A and malnutrition. Although commonly seen in South and East Asia, it is rarely reported in Singapore. However, there is still a population at risk here—patients with malabsorption and eating disorders, and institutionalised persons. Phrynoderma should, therefore, be considered in the differential diagnosis in patients with hyperkeratotic folliculitis, especially when malnutrition is also present.

Keywords: follicular hyperkeratosis, hypovitaminosis A, malabsorption, nutritional deficiencies, phrynoderma

Singapore Med | 2008; 49(6): e | 60-e | 62

INTRODUCTION

In a developed country like Singapore, malnutrition is seldom encountered. On the contrary, obesity and related disorders have become major public health issues. We present a case of phrynoderma associated with nutritional deficiency. Although rare in developed countries, there is still a population at risk in Singapore.

CASE REPORT

A 31-year-old mentally-deficient man was referred to the dermatology department with keratotic follicular papules on his right anterior chest wall. He was admitted to the medical ward for investigation of increasing lethargy with loss of appetite and weight. This was later found to be secondary to hyponatraemia, which was caused by his antipsychotic medications. The patient was a resident of a voluntary welfare nursing home. Premorbidly, he was uncommunicative and mainly chairbound, and had a history of behavioural problems. Four months prior to this admission, he was investigated for poor appetite and malnutrition, and found to have oesophagitis from gastroscopy.



Fig. I Clinical photograph of the patient. He was cachexic, severely malnourished and poorly hydrated.



Fig. 2 Close-up clinical photograph of the patient's chest shows reddish-brown papules with prominent keratotic follicular plugging

On examination, the patient was alert but uncommunicative. He appeared unkempt and his general hygiene poor. He was cachexic (weighing just 38 kg) and poorly hydrated (Fig. 1). Examination of his skin showed generalised xerosis. On the right side of his anterior chest wall, there was a 5 cm wide erythematous area of keratotic follicular papules. These are 3–4 mm reddish-brown papules with a prominent keratotic follicular plugging (Fig. 2). His hair was coarse, dry and messy, but his nails were normal.

There were no mucosal lesions noted. There were also multiple shallow ulcers and sores noted over the bony prominences of his ankles bilaterally, which were probably due to decubitus pressure or self-inflicted scratches. Ophthalmological examination

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Correspondence to: Dr Chia Min Wee Tel: (65) 8121 1536 Fax: (65) 6788 0933 Email: chia_min_ wee@yahoo.com.sg showed xerophthalmia with no other ocular or corneal involvement. His dentition was poor with multiple oral cavities.

Laboratory investigation showed normocytic anaemia with a haemoglobin level of 11.1 g/dL (normal range 14.0-18.0 g/dL). However, the iron panel, vitamin B12 and folate levels were within normal range. Although his serum albumin level was low (22 g/L [normal range 37-51 g/L]), his inflammatory markers and other liver panel were within normal limits. Nutritional studies were done in view of his poor nutritional status. The serum zinc (9.3 µmol/L [normal range 8.4-22.9 µmol/L]) and vitamin E levels (6.5 mg/L [normal range 5.0–18.0mg/L]) were borderline low, while the serum vitamin A level was grossly deficient (0.12 mg/L [normal range 0.30-0.80 mg/ L]). A stool sample was sent for evaluation but no parasite was found. No skin biopsy was done as consent could not be obtained from the patient, and he had neither family nor kin.

Based on his clinical presentation and the nutritional study results, a diagnosis of phrynoderma was made. The patient was commenced on oral vitamin A and multivitamins. He was also reviewed by the dietician and given supplemental protein and caloric intake. The patient was discharged when his medical condition improved. He was seen in the outpatient clinic one month later. As his nutrition became better, there was also improvement in his cutaneous lesions.

DISCUSSION

The term, phrynoderma, meaning "toad skin", was first coined in 1933 by Nicholls, who described the condition in East African labourers thought to be deficient in vitamin A.⁽¹⁾ Characteristic skin lesions are follicular papules with conical keratotic plugs on the extensor surfaces of the extremities. The lesions may also involve the abdomen, back and buttocks, typically sparing the hands, feet and face. (2) The patient had a clinical picture consistent with phrynoderma. He developed the characteristic hyperkeratotic papules and plaques on his trunk in a setting of low serum level of vitamins A and malnutrition. He also had marginal serum levels of vitamin E and zinc, and overt hypoalbuminaemia. He was treated with supplemental vitamins and improved nutrition, which led to improvement of his skin lesions. Besides follicular hyperkeratosis, hypovitaminosis A also affects the skin by causing xerosis, generalised hyperpigmentation and sparse and fragile hair. (3,4) Ocular involvement is common in vitamin A deficiency, frequently causing night blindness. (3-5) Severe deficiency can even lead to destructive eye lesions affecting the conjunctiva (xerosis conjunctiva and Bitot's spots) and cornea (xerosis corneae and keratomalacia).(3) Our patient exhibited other skin signs of hypovitaminosis A, namely: general xerosis and dry, sparse hair. Fortunately, his ocular involvement was mild with only xerophthalmia and no other permanent ocular or corneal sequelae.

Treatment consists of high dose vitamin A replacement therapy. Visual disturbances resolve within days, although corneal scarring is permanent. (6) The skin lesions usually take one to four months to heal. (7)

Vitamin A deficiency was the first cited cause of phrynoderma, but the aetiology has been a question of controversy. Deficiencies of other factors, such as vitamins B, C, E, caloric, and essential fatty acids, have been incriminated. Some authors even believed that general malnutrition has the strongest association. The clinical picture typically improves with enhanced nutritional status and vitamin A, as it did in our patient.

Although phrynoderma is most commonly seen in South and East Asia, it is rarely reported in Singapore. (9) The rare cases of phrynoderma reported in developed countries have been due to vitamin A deficiency secondary to malabsorption. Those previously reported include malabsorption secondary to small bowel bypass surgery for obesity, (5,7) as a complication to pancreatic insufficiency, (4) and even associated with chronic giardiasis. (14)

To the best of our knowledge, this report is the first documented case of phrynoderma in Singapore. Malnourishment and vitamin A deficiency is more common from developing countries, (15) while in developed countries like Singapore, it is usually a complication of malabsorption of animal fats, anorexia nervosa or in fad dieters. (6) However, there is a population at risk in Singapore. Raja et al studied the prevalence of malnutrition in hospitalised patients in Singapore, and found that the overall prevalence was 14.7%. (16) A study on plasma vitamins A, C and E in a random sample of the Singapore population found that, while no person had vitamin A deficiency (defined as < 0.01 mg/L), 0.1% had vitamin E deficiency (< 5.0 mg/L) and vitamin C levels were relatively low.(17) There have also been increasing literature and reports about anorexia nervosa and other eating disorders in Singapore, with a prevalence believed to be as rampant as the West. (18,19) Therefore, the population at risk for nutritional skin diseases is not insignificant here. Phrynoderma must, therefore, be considered in the differential diagnosis in patients presenting with hyperkeratotic folliculitis in the setting of malnourishment, and should prompt the clinician to evaluate the nutritional status, not just the vitamin A level.

Phrynoderma is a distinctive form of follicular hyperkeratosis associated with nutritional deficiency. Originally thought to represent vitamin A deficiency, several studies have now demonstrated that it has multiple aetiologies. Although rare in developed countries, there is still a population at risk, such as patients with malabsorption (from various causes) and eating disorders,

and institutionalised persons. Phrynoderma must, therefore, be considered in the differential diagnosis in patients with hyperkeratotic folliculitis in the setting of malnourishment.

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