Calcinosis cutis in a pediatric patient with Burkitt's lymphoma

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Turk J Haematol 2005;22(3): 151-154

Received: 28.12.2004 Accepted: 10.03.2005

ABSTRACT

Calcinosis cutis, an uncommon disorder characterized by hydroxyapatite crystals of calcium phosphate deposited in the skin, has been described infrequently in childhood. Cutaneous calcification may be divided into four major categories: dystrophic, metastatic, idiopathic, and iatrogenic. Here, we report an example of iatrogenic type with a 4-year-old boy who diagnosed with Burkitt's lymphoma, and developed calcinosis cutis secondary to a tumour lysis syndrome with induction chemotherapy.

Key Words: Calcinosis cutis, Malignancy, Tumour lysis syndrome.

ÖZET

Burkitt lenfomalı pediatrik hastada kalsinozis kutis

Kalsinozis kutis, deride kalsiyum fosfat hidroksiapatit kristallerinin birikimi ile karakterize çocukluk çağında nadir görülen bir patolojidir. Derideki kalsifikasyon, distrofik, metastatik, idiyopatik ve iyatrojenik olarak dört ana gruba ayrılabilir. Burada, Burkitt lenfoma tanısı konulan dört yaşında bir erkek olguda indüksiyon tedavisi sırasında gelişen tümör lizis sendromuna sekonder iyatrojenik kalsinozis kutis tablosu sunulmakta ve literatür bilgileri ışığında tartışılmaktadır.

Anahtar Kelimeler: Kalsinozis kutis, Malignite, Tümör lizis sendromu.

INTRODUCTION

Calcinosis cutis, an uncommon disorder characterized by hydroxyapatite crystals of calcium phosphate deposited in the skin, has been described infrequently in childhood. Cutaneous calcification may be divided into four major categories: dystrophic, metastatic, idiopathic, and iatrogenic[1-7]. The rare types, calcinosis cutis cicumscripta, universalis and tumoral calcinosis could be included in the group of dystrophic and also in the group of iatrogenic[8-10]. Here, we report a 4-year-old boy with Burkitt's lymphoma, who developed calcinosis cutis secondary to a tumour lysis syndrome with induction chemotherapy.

A CASE REPORT

Four year old boy with Burkitt's lymphoma had generalised tonic-clonic convulsions secondary to hypocalcaemia after induction chemotherapy. At this time, laboratory examinations showed calcium: 4.5 mg/dL, phosphorus: 9.5 mg/dL, potassium: 5.7 mEq/L, magnesium: 2.7 mg/dL, magnesium: 2.7 mg/dL, uric acid: 8.7 mg/dL, blood urea nitrogen: 7 mg/dL, albumin: 3.2 g/L, and urine pH: 8.0.

The focuses of tissue necrosis in diameters of 2-3 millimetres were occurred after intravenous infusion of calcium gluconate especially around intravenous injection sites and extremities.

A few weeks later, white coloured, firmly, palpable and sensitive nodular lesions developed in this sites (Figure 1). Bone scintigraphy revealed the rising in activity of uptake. Incisional biopsy showed mononuclear cell Infiltration in perivascular and periadnexial tissue of superficial and deep dermis focuses of degeneration and calcification in collagen suggested calcinosis cutis (Figure 2).

DISCUSSION

Calcinosis cutis is characterized by hydroxyapatite crystals of calcium phosphate deposited in the skin. Cutaneous calcification may be divided into four major categories: dystrophic, metastatic, idiopathic, and iatrogenic[5,6].

Dystrophic calcification occurs as a result of local tissue injury, inflammation, necrosis, or neoplastic changes. It occurs without calcium and phosphorus metabolic abnormalities and may be localized or generalized[3,4,6,9].

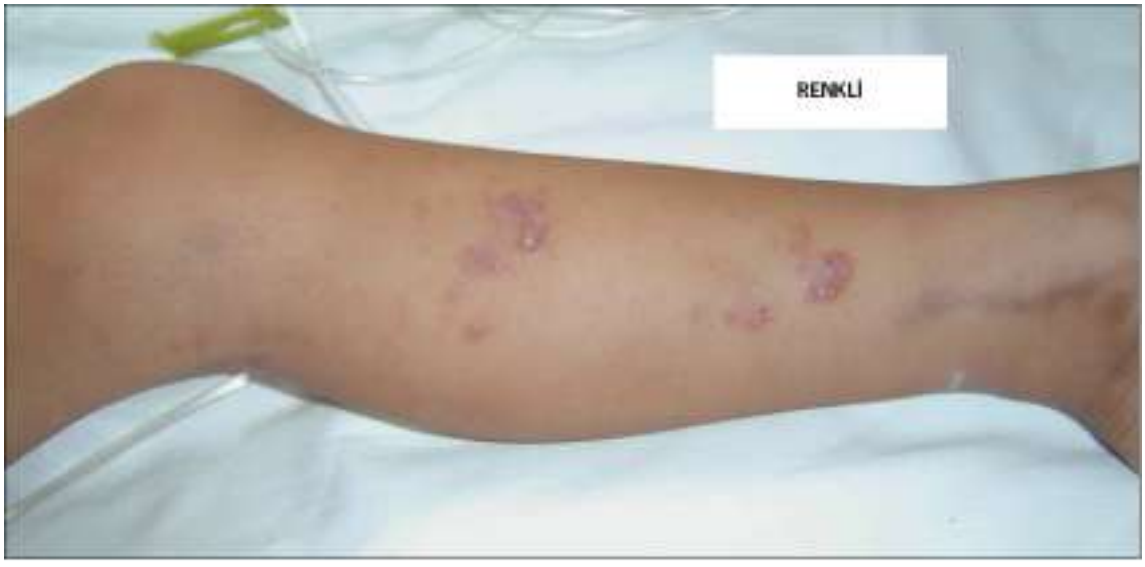

Figure 1. Skin lesions of the patient.

Figure 2. Histopathologic appearance of the skin biopsy.

Metastatic calcification results from abnormal calcium and/or phosphate metabolism especially hypercalcaemia and/or hyperphosphatemia[6]. But there is no known etiologic factor or systemic disease and local tissue damage in idiopathic type[4,6,7,11].

Iatrogenic calcinosis cutis appears after a treatment procedure such as parentheral calcium or inorganic phosphorus treatment, tumour lysis syndrome, and long term application of calcium containing gels used in electromyography, electroencephalography[5,9]. Tumour lysis syndrome causes hyperkalemia, hyperphosphatemia, hyperuricemia and secondary hypocalcaemia as a result of rapid potassium and phosphorus releasing and production of uric acid. This condition results in serious and life-threatening complications like acute renal failure, multiple organ dysfunction and probably death.

In our case, calcinosis cutis occurred due to accumulation of calcium-phosphate deposits in dermis as a result of intravenous calcium replacement for hypocalcaemic convulsion.

Elevated extracellular phosphorus level that also increases intracellular phosphorus lead to calcium x phosphorus > 70 in a tumour lysis syndrome. This causes precipitation of calcium-phosphate crystals in dermis[5].

In addition to biochemical parameters for diagnosis, X-ray may show soft-tissue calcification. A rare case of extensive dermal uptake of Tc-99 m MDP associated with renal failure is reported. The mechanism of Tc-99m MDP uptake in such examples of metastatic calcification is not proved, but may relate to adsorption onto hydroxyapatite crystals. This phenomenon is useful in demonstrating distribution of nonvisceral metastatic calcification[1].

Computerized tomography can be used in differentiation of visceral and non-visceral calcinosis and rarely in diagnosis of primary tumoral calcinosis. The value of magnetic resonance imaging to evaluate the calcified tissues is limited. Biopsy and histopathology are diagnostic tools. Sometimes fine-needle aspiration biopsy may be diagnostic[4-6].

The main principle of medical treatment is to treat underlining disease. The application of corticosteroids directly to the lesions may be useful via inhibition of fibroblastic activity. Probenecid and colchicines have effect on some cases. Magnesium and aluminium antacids are powerful phosphate binders, but can cause magnesium and aluminium toxicity in cases of renal failure. Natrium etidronate and diphosphanates reduce the occurrence of ectopic hydroxyapatite crystals, but can cause paradoxically hyperphosphatemia when it is used for a long time. A calcium-canal blocker, diltiazem has some benefits when used more than five years[9,12,13]. Cutaneous calcification may be divided into four major categories: dystrophic, metastatic, idiopathic, and iatrogenic. The probable indications for surgical treatment are pain, recurrent infections, ulceration and functional disability. Because the surgical procedure it self can also stimulate calcification, it must be tested in a narrow area before total excision. Relapsing after excision is also frequent[9,10,13]. In conclusion, we must be careful when apply intravenous treatment especially in a tumour lysis syndrome and follow serum concentrations of electrolytes carefully.

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