

# Renal Abnormalities During Calcinosis Primary Tumor

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## Abstract

Primary tumour calcinosis is a rare condition of unknown etiopathogeny. They are transmitted in an autosomal dominant or recessive mode and predominate in the black race. Calcinoses are manifested by hydroxyapatite deposits in the dermis or hypodermis. The authors report an observation of primary tumour calcinosis associated with renal abnormalities and statureweight retardation in an 11-year-old child.

**Keywords:** Renal abnormalities; tumor calcinosis; child.

## 1. Introduction

Tumorous calcinosis is a rare condition that affects black adolescents or adults [1]. There are several types of tumor calcinosis and their nosology is not well defined. Tumor calcinosis can be primary or secondary, isolated or associated with other conditions [23]. The characteristic feature of tumour calcinosis is the dermal or hypodermal deposition of hydroxyapatite. Primary tumour calcinosis is described as TEUSCHLANDER's lipocalcinogranulomatosis with associated metabolic disorders [5]. The authors report a case of tumour calcinosis associated with renal abnormalities in an 11-year-old child.

## 2. Observation

An 11-year-old black boy was admitted to a dermatology consultation at Brazzaville University Hospital for diffuse hypopigmented macules associated with a diffuse, predominantly acral skin sclerosis that had been evolving for 6 years.

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The general condition was preserved, he was not mentally retarded, his height was 1.28m, weighed 26 Kilos, a standard deviation of -3. The interrogation had been regaining the notion for 2 - 3 months of thirst, of abundant urination. Blood pressure was 80/60 mmHg. The examination found voluminous masses evolving over the last 5 years from 2 to 10 cm in diameter at the hips, knees, elbows and at the upper 1/3 of the right leg, of firm consistency, some ulcerated pseudogumous masses with an inhomogeneous whitish and chalky background. The paraclinical assessment showed: blood glucose 1g/l, positive glycosuria, creatinine 6mg/l, low serum calcium 78 mg/l, low phosphorus 20 mg/l, kalaemia 3 mmol/l, natraemia 134 mmol/l, low proteinuria 0.23 g/24h (9 mg/kg/d). Calciuria was elevated above 0.1 mmol/Kg/24h as was phosphaturia, diuresis at 2.3 l/24h. Standard radiography of the pelvis showed multiple calcifications (photo). The diagnosis of lipocalcino granulomatosis was suggested to be associated with proximal tubulopathy and staturweight retardation.

### **3. Discussion**

Tumor calcinosis or lipocalcino granulomatosis is a rare, benign condition characterized by the deposition of calcium material in extra-articular soft tissues in tumor form [5]. It may be primary or secondary to chronic renal failure. Heredity is found in 30% of cases. Transmission is autosomal recessive or dominant according to the authors with variable penetrance [7]. Three types of mutations have been identified, one affecting fibroblast growth factor 23 (FGF23) [8], the second affecting the phosphaturic hormone and glycosyl transferase GALT3 and the phosphatemic form linked to the SAM D9 gene [8,9]. Disturbances in phosphate metabolism may accompany some tumor calcinosis [10]. Hyperphosphatemia is the rule [11,12], often secondary to chronic renal failure and hyperparathyroidism. The hypophosphatemia and hyperphosphatemia found in our observation are unusual. The existence of normo-glycemic glycosuria, hypercalciuria and hypocalcemia are evidence of an associated proximal tubulopathy. Is this an incidental association of tumour calcinosis and proximal tubulopathy or an unusual type of mutation? Staturponderal retardation has never been described in classical forms of tumour calcinosis. It is a manifestation of the hydroelectrolytic disorders associated with proximal tubulopathy. Our observation is unusual because of the coexistence of staturweight retardation and tumour calcinosis. The occurrence of proximal tubulopathy is paradoxical in the course of tumour calcinosis; it may be related to an exceptional form or may be the translation of an unknown underlying hereditary pathology. We haven't found any link to a sickle cell trait. Hemoglobin electrophoresis was normal. Our limited means of investigation did not allow us to investigate genetics.

### **4. Conclusion**

This observation illustrates the rarity of the association of tumour calcinosis and proximal tubulopathy with staturweight retardation. Exhaustive genetic and metabolic investigations are essential for a better etiopathogenic and therapeutic approach. The relevant or fortuitous link of this association remains to be determined.

### **5. Conflict of interest**

None.

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