African Journal of Biomedical. Research. (2002): Vol 5; 91 - 92

TUMORAL CALCINOSIS: REPORT OF A CASE

OGUNLADE S.O.[1*], SALAWU S.A[1] AND EYELADE R.A[2]
[1]Department of Surgery, College of Medicine, University of Ibadan, Nigeria.
[2]Department of Anesthesia, College of Medicine, University of Ibadan, Ibadan, Nigeria.

Tumoral calcinosis is a rare syndrome characterized by massive subcutaneous soft tissues deposits of calcium phosphate near the large joints. We report herein a 20 old boy with calcified lesions bilaterally involving the soft tissue over the greater trochanter. The serum calcium, phosphate and urea were normal.

Key words: *tumoral calcinosis, calcified lesions, greater trochaner, exclusion*

** Author for correspondence*

INTRODUCTION

Tumoral calcinosis is a rare syndrome characterized by massive subcutaneous soft tissues deposits of calcium phosphate near the large joints. The condition has been reported in patients ranging from age 5 months to 83 years (Bostrom *et al*, 1981; Aprin *et al* 1986), it usually becomes manifest in the second decade of life (Aprin *et al*, 1986). Men and non-whites are affected more commonly than women and Caucasians.

We report herein a 20 old boy with calcified lesions bilaterally involving the soft tissue over the greater trochanter.

CASE REPORT

A 20 year old boy presented in the surgical outpatient Clinic with swelling of both hip region of 1½ years duration, he complained of aching pain in both hips over the swelling. He denied any history of trauma.

The right trochanteric measure 15cm by 10cm, firm while and the left trochanteric mass measured 10cm by 7cm and was firm (Plate 1). X-ray of the pelvis revealed circumscribed multiple calcification over greater trochanter. Hip joints are not involved Plate 2.

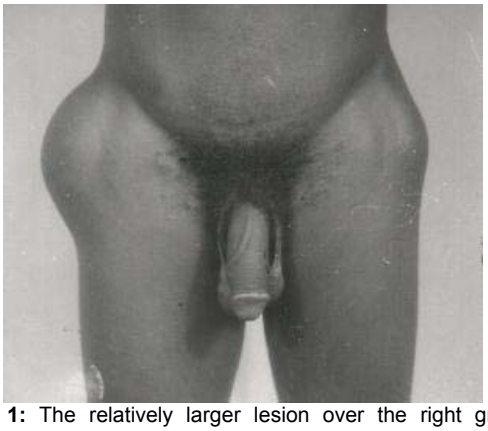

Plate 1: The relatively larger lesion over the right greater trochanter is demonstrated

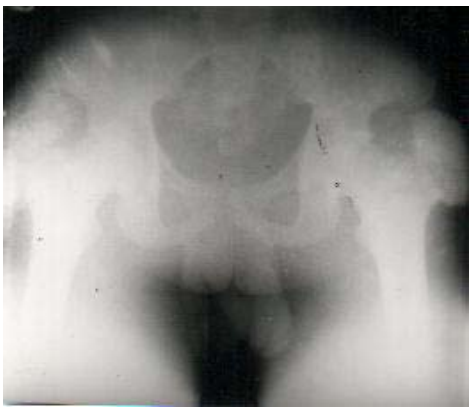

Plate 2: Plain radiography of the pelvis showed massive soft tissue calcification over the greater trochanter. The hip joints are unaffected

At surgery, the capsule was adherent to subcutaneous tissue, the tissue, the mass was dissected free by sharp dissection; there was significant blood loss. Postoperative recovery was uneventful.

The patient's Clinical photograph five months post operatively is shown in Fig 3. No recurrent at 7 month follow up. Histology confirmed Tumoral calcinosis.

DISCUSSION

Tumoral calcinosis is characterized by massive subcutaneous soft tissue deposit of calcium phosphate near large joints such as hip, the shoulder, and the elbow, in addition to the wrist, feet and hands (Mitnick *et al*, 1980). It has also been reported in subcutaneous tissue of the abdomen and thigh following trauma (Arikawa *et al*, 2002).

The majority presented with a painless swelling in single or multiple periarticular regions. The hip is the most commonly affected (Pakassa *et al*, 1997). Idiopathic tumoral calcinosis should be diagnosed by eliminating the other diseases in which the same calcified masses are seen. Some

of these disease are chronic renal disease, hypervitaminosis, milk-alkali syndrome, Sacoidosis, and primary hyper parathyroidism (Lafferty *et al*, `1965; Clarke *et al*, 1984) the serum calcium concentration are high in these disease, normocalcemi and hyperphosphatemia or normal phosphate level in some cases exist in tumoral calcinosis (Clarke *et al*, 1984; Lakhkar *et al*, 1991).

Plate 3: 5 months post-surgery. Patient's condition satisfactory

The condition has been reported in patient ranging from age 5 months to 83 years (Bostrom *et al*, 1981; Aprin *et al* 1986), it usually becomes manifest in the second decade of life (Aprin *et al*, 1986). Men and non-whites are affected more commonly than women and Caucasians.

A family history is apparent in 30 to 40 percent of cases, and an autosomal recessive pattern of inheritance is suggested (Harwood *et al*, 1996); Nedin *et al*, 2000. The finding by Mitnick *et al* (1980) support the concept of a specific enhancement in renal tubular phosphate reabsorption, probably originating in the proximal tubule. Their data also suggest that a direct effect of hyperphosphatemia to reduce urinary calcium excretion may be the mechanism through which hormonal ionized calcium and serum parathormone levels are maintained in this condition.

The valid medical treatment are surgical excision or a low-phosphorus, low calcium diet with phosphate – binding antacids. Complete surgical excision of early lesions has been recommended (Bostrom *et al*, 1981); Baldursson *et al*, 1969), but recurrence is common Baldursson *et al*, 1969).

The growth of recurrent masses is frequently more rapid than of the initial lesions, especially in patients with incompletely excised tumours (Kirk *et al*, 1981), administration of steroid, prebenecid, phenylbutazone, diphosphonates and thyrocalcitonin, and radiation therapy have proved unsuccessful. Calcium administration is contraindicated (Lafferty *et al*, 1965, Kirk *et al*, 1981).

Spontaneous regression of the lesion have been reported while in patient with uraemic tumoral calciunosis, resolution have been seen after renal transplantation and also using continuous ambulatory peritoneal dialysis (CAPD) combined with hemodialysis with low-calcium dialysate (Niall *et al* 1998; McGregor *et al* 1999; Kuriyama *et al* 1998).

REFERENCES

Aprin, H; and Sinha, A. (1986): Tumoray Calcinosis: Report of a case in a one – year old child. Clin. Orthop. 185:83

Arkawa J, Higaki, Y, Mizushima, J. *et al* (2000). Tumoral calciunosis: A case report with an electron microscopic study. Eur J Dermatol Jan-Feb; 10(1): 52-4

Baldurssion, H., Evans, e.B., Dodge, W.F., *et al* (1969): Tumoral calcinosis with hyperphosphatemia: a report of a family with incidence in four sublings. J. Bone joint Surg. Am. 51; 913.

Bostrom, B (1981): Tumoral calcinosis in an infact. Am. J. Dis. Child. 135:246.,

Clarke, E., Swischuk, L.e., and Hayden, C.K., jr. (1984): Tumoral Calcinosis: an unsual cause of Cutaneous calcification. Clin Exp Dermatol mar, 21(2): 163-6.

Kirk, T.S. and Tomonari, H., Nakayama, M., *et al* D.H. (1965): Tumoral Calcinosis: A metabolic disease of obscure etiology Am. J. Med. 38: 105.

Kuriyama, S., Tomonari, H., Nakayama, m *et al* (1998): Successful treatment of tumoral calcinosis using CAPD combined with hemodialysis with low-calcium dialysate. Blood purif; 16(1): 43-8

Lafferty, F.W., Reynoids, E.S., and Pearson, D.H. (1965): Tumoral Calcinosis: A metabolic disease of obscure etiology Am. J.Med 38: 105.

Lakhkar, B.B, and Lakhkar, B.N (1991): Tumoral calcinosis. Acta Paediatr. Scand. 80: 475,

McGregor, D, Burn, J Lynn, K; *et al* (1999): Rapid resolution of tumoral calcinosis after renal transplantation. Clin. Nephrol Jan, 92: 8.

Mitnick, P.O., Goldfarb S. Slatopolsky, E, *et al* (1980): .Calcium and Phosphate metabolism in tumoral calcinosis. Ann. Intern. Med. 92: 482,

Nedin, S., Mustafa, C.A., Zekeriya, T., *et al* (2000): Hyperphosphatemic Tumoral clacinosis. Plastic and reconstructive surgery 105: 162-165.

Niall, D.M., Forgarty, E.E, Dowling, F.E., *et al*. (1998): Spontaneous regression of Tumoral Calcinosis in an infant; a case report. J Pediatrsurg Sep; 33(9): 1429-31.

Pakassa, N.M., Kalengayi, R.M (1997): Tumoral Calcinosis a clinicopathological study of III cases with emphasis on the earliest changes. Histopathology Jul; 31 (1): 18-24

Received: September, 2000
Accepted in final form: September 2001