**Introduction**

- Tumoral Calcinosis involves the deposition of calcium within periarticular soft tissue forming lobular, radiodense masses.
- It is a rare, benign condition that is usually familial but in some instances can result from metabolic abnormalities.
- The strict definition of tumoral calcinosis requires a hereditary predisposition to the disease and normal calcium levels. However, sporadic or idiopathic occurrences of tumoral calcinosis have also been reported.
- We present a patient whose kidney failure combined with excessive calcium intake led to tumoral calcinosis.

**Case Description**

- A 62 year old man with a history of chronic kidney disease stage 5, diabetes mellitus, gout and hypertension, presented to the hospital after running out of solution for his chronic peritoneal dialysis. He had lost his insurance after an extended trip to his home country of Mexico, and thus was hospitalized for peritoneal dialysis. On review of systems he complained of mild, chronic right knee pain.

**Physical Exam**

- Afebrile, normal vital signs, no apparent distress.
- Firm, fixed, non-tender nodule on the right elbow (diameter 2cm) and over the bilateral metacarpal joints. There was mild tenderness over the right olecranon bursa with extreme flexion and extension.

**Laboratory Results**

- Calcium: 12.0 mg/dL
- Phosphate: 7.4 mg/dL
- Vitamin D 16 ng/mL
- PTHrP: negative

**Hospital Course**

- Patient was restarted on scheduled peritoneal dialysis.
- Joint pain was initially treated with NSAIDs and steroids, however, it persisted and radiographs were obtained.
- On follow up questioning, the patient reported taking high dose calcium acetate while he was in Mexico.
- During hospitalization, calcium acetate was not restarted and his calcium levels normalized.
- Patient was discharged after three weeks with outpatient follow up.

**Take Home Points**

- Tumoral calcinosis, although usually a benign condition, can result in patient discomfort and functional limitations.
- Secondary tumoral calcinosis has been associated with a calcium phosphate product greater than 65-75 with chronic kidney disease, where secondary hyperparathyroidism often coexists.
- Our patient had a calcium phosphate product of 89, but did not have secondary hyperparathyroidism.
- Treatment involves symptomatic management and normalization of phosphate and calcium levels.
- With normalization of calcium and phosphate, deposits can gradually decrease in size.

**References**