More about Tumoral Calcinosis in Maintenance Hemodialysis Patients

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Dear Sir,

Tumoral calcifications are an infrequent finding in hemodialysis patients. In reference to the paper published by Zins et al. [1], we report upon a hypothyroid female patient aged 51 years who developed this kind of metastatic calcification after 7 years of maintenance hemodialysis in a unit with water treatment including reverse osmosis. She was the only case who developed this complication among 971 patients followed up between 1974 and 1992.

She was started on hemodialysis in 1980. Arterial hypertension and hypothyroidism were reported in her clinical history. She was never compliant respective to thyroid hormone replacement and phosphorous chelators (aluminum hydroxide and calcium carbonate), thus she received vitamin D supplements, irregularly. A transiliac bone biopsy was performed in 1984 and osteitis fibrosa cystica was informed from a decalcified specimen.

Nonparathyroid enlargement was shown by ultrasonographic scan. In 1987, roentgeno-graphic features of ectopic calcifications sized 20 × 20 cm were observed in the adductor muscles (fig. 1) and periarticular zones bilaterally, as well as a compressive fracture of D11 and a periarticular calcification of the shoulders. Computed tomography revealed intrapulmonary and mitral valve calcium deposits as visceral calcifications. Disseminated vascular calcifications had previously been detected. The patient refused a second bone biopsy which was suggested in order to investigate aluminum bone disease. A septic complication caused her death in 1988. Visceral and nonvisceral metastatic calcifications represent different etiological mechanisms and rarely coincide [2]. Nonvisceral calcifications are related to: (a) hyperphosphatemia and (b) increased calcium-phosphate product. Instead, visceral calcifications are associated to other factors such as tissue dystrophy [3].

In our patient, laboratory data revealed the following (table 1): persistent hyperphosphatemia, alkaline phosphatase progressive elevation without hepatic disease, and increased PTH levels without echographic confirmation of an enlarged gland [4].

The possible coexistence of bone and parathyroid aluminum deposits are suggested by PTH levels and the absence of an increased size of the gland. Also, aluminum [1] has been implicated among the mechanisms of metastatic calcification, but unfortunately, it was not investigated in our patient.

Nevertheless, the etiological importance of aluminum must be reviewed due to the greatest frequency of aluminum bone disease compared to that of tumoral calcifications.

Thus, hyperparathyroidism, biochemical abnormalities, tissue dystrophy related, in this case, to hypothyroidism, and aluminum bone disease all might play a possible role in the mechanism of metastatic calcification.

Finally, up to now, one of the best available approaches to prevent this complication among hemodialysis patients should be a careful control of phosphate levels, even with the use of aluminum chelators when phosphate levels are superior to 6-7 mg/dl [5].

Fig. 1. Radiological aspect of tumoral calcification in the right thigh region of the patient.

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Table 1. Patient yearly laboratory data

Results are expressed as mean $\pm$ SD. Normal values are given in parentheses. Levels were determined by a carboxiterminal RIA.

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