

## Letters to the Editor

**Regression of Tumoral Calcinosis  
After the Appropriate Control of a  
Deranged Mineral and Bone  
Metabolism, in Conjugation With  
Cinacalcet Hydrochloride Treatment,  
in a Chronic Hemodialysis Patient**

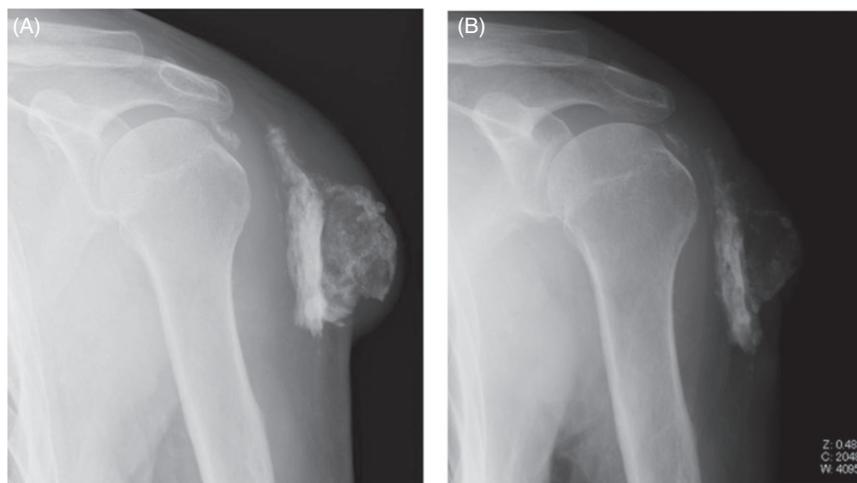
Dear Editor,

Uremic tumoral calcinosis is an ectopic calcification that is frequently associated with persistent hypercalcemia and hyperphosphatemia in dialysis patients (1). In recent years, calcimimetic compounds have become widely used in dialysis patients for controlling secondary hyperparathyroidism (SHPT). Because cinacalcet can lower the serum parathyroid hormone (PTH), calcium (Ca), and phosphorus (P) levels simultaneously, it can therefore potentially improve the tumoral calcinosis resulting from mineral disorders associated with SHPT.

We herein present the case of a 62-year-old male who had received 12 years of HD therapy, who was hospitalized for painful calcified masses around the left shoulder and bilateral iliac crests. He had undergone a total parathyroidectomy with forearm auto-transplantation for SHPT 5 years before hospitalization. However, his SHPT relapsed, and intravenous maxacalcitol therapy (15 µg/week) with calcium carbonate (3 g/day) had been administered for the

last 4 years; the serum Ca and P levels remained above the upper limit recommended by the guidelines ( $10.7 \leq \text{serum Ca} \leq 12.8 \text{ mg/dL}$ ,  $6.5 \leq \text{serum P} \leq 8.2 \text{ mg/dL}$ ) (2).

On admission, the patient showed serum levels of corrected Ca of 12.6 mg/dL, P of 6.8 mg/dL, intact PTH of 320 pg/mL, and bone specific alkaline phosphatase of 34 U/L. He had been receiving HD during 4-h sessions three times per week with 3.0 mmol/L of Ca dialysate. Tc<sup>99m</sup> methoxy-isobutylisonitrile scintigraphy and neck ultrasonography disclosed two hyperactive parathyroid glands; one in the forearm and the other in the neck. Radiology disclosed a calcified mass around the left shoulder and bilateral iliac crests (Fig. 1). We concluded that the calcified masses were uremic tumoral calcinosis induced by persistent hypercalcemia and hyperphosphatemia, which could be attributed to the recurrent SHPT and inappropriate use of vitamin D and a Ca-based P binder. We discontinued the administration of maxacalcitol and calcium carbonate, and started treating him with sevelamer hydrochloride (3 g/day) and alphacalcidol (0.25 µg/day). A dose of 25 mg/day of cinacalcet hydrochloride was also started for the recurrent SHPT. At one month after adopting the new treatment, the serum Ca, P, and intact PTH levels were 9.2 mg/dL, 5.8 mg/dL, and 84.5 pg/mL, respectively, and these parameters remained within the recommended ranges thereafter ( $8.9 \leq \text{serum Ca} \leq 9.9 \text{ mg/dL}$ ,  $4.3 \leq \text{serum P} \leq 5.8 \text{ mg/dL}$ ,  $76.5 \leq \text{intact PTH} \leq 145.8 \text{ pg/mL}$ ). At



**FIG. 1.** X-ray films of the tumoral calcinosis before (A) and after (B) treatment. The tumoral calcinosis around the left shoulder regressed 6 months after the patient started cinacalcet hydrochloride treatment (arrows).

6 months after changing treatment, the tumoral calcinosis around the left shoulder had markedly decreased (Fig. 1).

The regression of tumoral calcinosis can be promoted by the normalization of the serum Ca and P levels to within the target ranges (1,3). In the present case, cinacalcet effectively lowered the serum PTH level, thus leading to the amelioration of high bone turnover and the normalization of the serum Ca and P levels, although both conversion of the Ca-containing P-binder to a non-Ca containing agent, and the discontinuation of intravenous vitamin D therapy could also have contributed to the normalization of the serum Ca and P. This total management of mineral and bone disorders could accelerate the passive and active resolution of the Ca-P complex from the tumoral calcinosis (3). Additionally, a recent clinical report indicated that cinacalcet has a direct effect on vascular calcification (4), and it may have directly acted on the tumoral calcinosis. Therefore, further studies are needed to determine the indirect and direct effects of cinacalcet on soft tissue calcification, including vascular calcification.

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## Catheter Related Infection Due to *Mycobacterium abscessus* in a Patient Under Peritoneal Dialysis

Dear Editor,

According to Kameyama et al. (1), catheter removal was mandatory if *Mycobacterium abscessus*

related peritonitis was reported. We present a case of exit site infection (ESI) due to such an unusual pathogen. The current data indicates rareness of infection from peritoneal dialysis-related nontuberculous mycobacteria (NTM). There is one published study from Hirohama et al. (2) reporting a successful experience of treating *Mycobacterium gordonae* related infection. Due to the lack of research, there is scarcity of treatment experiences for *Mycobacterium abscessus*. Therefore, it is crucial that we present our experience, which is the second case presented of *Mycobacterium abscessus* related ESI and tunnel infection (TI).

The 50-year-old man was diagnosed with herb related end-stage renal disease (ESRD) receiving continuous ambulatory peritoneal dialysis (CAPD). The exit site became purulent after one year of CAPD and the bacterial culture of the discharge was negative. We prescribed topical gentamicin ointment and oral amoxicillin/clavulanate for one month. Then, oral cephalexin and gentamicin ointment were prescribed for another month. The condition of his ESI fluctuated for a year. He applied gentamicin intermittently and took  $\beta$ -lactam antibiotics orally for six months. One year later, ESI progressed to TI because sonography revealed an abscess near his Tenckhoff catheter. We then tracked back his previous specific cultures and discovered an overlooked report showing NTM infection. Therefore, we cultured the pus again with special media for *Mycobacterium*, and the result was *Mycobacterium abscessus*. During this course, all seven culture reports were negative, except two positive results. One was NTM and the other was *Mycobacterium abscessus*. A surgeon performed debridement, followed by three combined drugs (via in vitro susceptibility) for two months: ciprofloxacin, clarithromycin, and rifampin. Although the wound healed well, there was still a delay in diagnosing *Mycobacterium abscessus* owing to its rarity, albeit there was successful preservation of the catheter. The following culture for *Mycobacterium abscessus* was negative.

He was the fourth *Mycobacterium abscessus* related CAPD infection reported before 2011 (1–3), and the second for ESI or TI. *Mycobacterium abscessus* belongs to a family of rapidly growing NTM, in which *Mycobacterium fortuitum* and *chelonae* infections are more common. However, cases of *Mycobacterium abscessus* related CAPD infection have increased since the advent of molecular identification methods, which distinguishes *Mycobacterium abscessus* from *Mycobacterium chelonae* (4). Importantly, NTM CAPD infections are more common in Asia because of warmer weather and humidity.