Tumoral Calcinosis: What Is the Treatment? Report of Two Cases of Different Types and Review of the Literature

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Abstract

Tumoral calcinosis (TC) is a rare problem that can be idiopathic (primary) or secondary to other diseases, particularly end-stage renal disease. Although these 2 types of TC do not differ in their radiologic and histopathologic presentations, their treatment modalities may be changed on the basis of etiology. Surgical resection of the calcified mass is the main treatment for the primary type, but should be avoided in hemodialysis-related types, which are instead often treated with parathyroidectomy.

In this article, we report on 2 cases of TC (different types). We also describe several aspects of diagnosis and different therapeutic approaches through a review of the literature.

Tumoral calcinosis (TC) is a histopathologic syndrome characterized by calcium deposits in periarticular areas, most often the hips, shoulders, and elbows, and less often the hands, feet, and knees.[1] Only clinical and radiographic findings are universally congruous, whereas the epidemiology and etiology are still under discussion, according to the literature.[2]

TC is an uncommon entity. It has a primary type with no associated disease and a secondary type that follows other disorders, particularly chronic renal failure.[1] In 1935, Teutschländer[3] was the first to use the term progressive lipocalcinogranulomatosis for a case of TC. There has been a trend from the almost exclusively idiopathic cases reported in the early literature to more cases associated with other diseases, particularly end-stage renal disease (ESRD), in the more recent literature. Although surgical resection has been the main treatment for primary TC for decades,[4] the preferred surgical treatment for TC associated with ESRD has been changed from surgical resection of the calcified mass to a more etiologic approach of subtotal parathyroidectomy for recurrence prevention.[3]

Here we report on 2 cases of TC—a case associated with ESRD and an idiopathic case—and review the English literature on the etiology, diagnosis, and treatment of TC. The patients provided written informed consent for print and electronic publication of their respective case reports.

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Case Reports

Case 1

In April 2008, a 50-year-old man was referred to our orthopedic clinic with a massive enlargement on the posterior aspect of the right elbow. The mass had developed gradually over 3 weeks. The patient reported minor

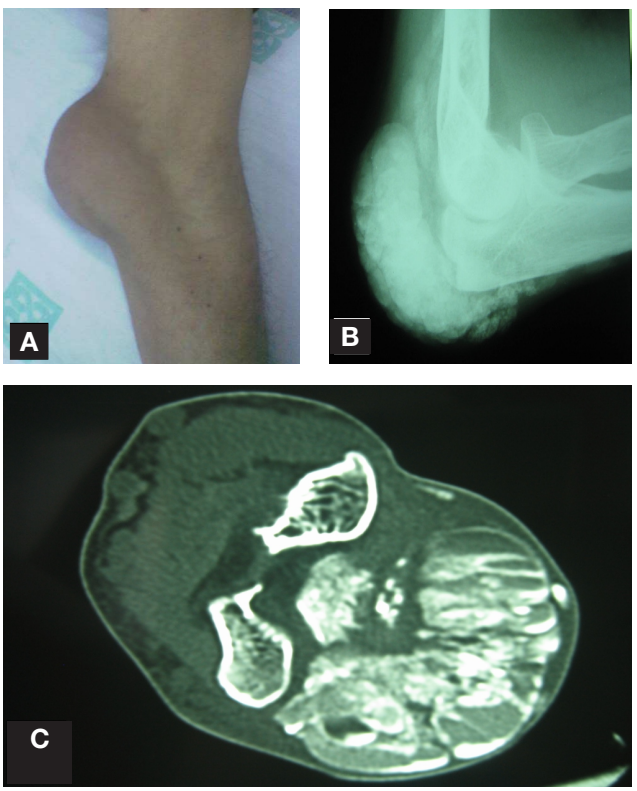

Figure 1. Case 1. Patient had had end-stage renal disease for 5 years. (A) Mass on posterior aspect of right elbow. (B) Simple radiography shows mass with calcified nodules. (C) Axial computed tomography shows mass with calcification and cyst formation.

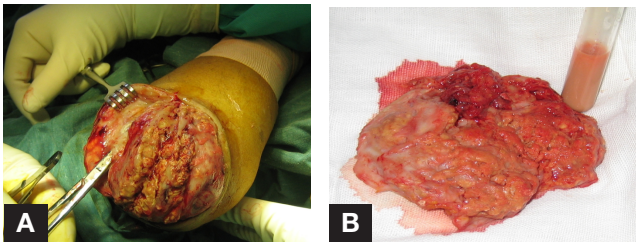

Figure 2. Case 1. Intraoperative view of calcified mass before resection (A) and after resection (B).

pain, without restriction of elbow range of motion (ROM), and a chalky white material that had begun discharging occasionally, 7 days after an iatrogenic attempt to aspirate the mass at another center. There were no associated constitutional symptoms and no trauma history. As the patient had polycystic kidney disease, identified by hypertension and hematuria 6 years earlier, and then ESRD, he had been undergoing hemodialysis 3 times per week over the past 5 years. Hepatitis B surface antigen was detected in a previous serologic profile, so there was the added concern about transmitting hepatitis B virus to his family. The patient was taking aspirin 80 mg/d, atenolol 50 mg twice per day, captopril 12.5 mg twice per day, and lamivudine 10 mg daily on order of a nephrologist and infectious disease experts.

Physical examination revealed a firm, lobular, 8×8-cm soft-tissue mass on the posterior aspect of the right elbow and on the distal portion of the humerus (Figure 1A) associated with a pinpoint fistula near the dome of the mass, and slight erythema and warmness. Compression of the mass led to extraction of a creamy, white material that, after desiccation, remained as a white powder on the skin. Elbow ROM was painless, and only 10° of extreme extension had been lost (Figure 1A). There was no sign of involvement of the regional neurovascular and lymphatic structures. Radiography showed a lobulated calcified mass in the posterior soft tissue of the humerus and elbow with no bony erosion (Figure 1B). Findings were similar on computed tomography (CT), which also showed an intact elbow joint space (Figure 1C). On laboratory testing, calcium level was 8.5 mg/dL (normal range, 8.4-10.5 mg/dL), phosphate level was 7 mg/dL (normal range, 2.7-4.5 mg/dL), alkaline phosphatase level was 581 U/L (normal range, 94-270 U/L), parathyroid hormone (PTH) level was 60 pg/mL (normal range, 15-65 pg/mL), alanine aminotransferase and aspartate aminotransferase were normal, serum albumin (3.9 g/dL) and total protein were normal, slightly normochromic-normocytic anemia was present (hematocrit, 41%), erythrocyte sedimentation rate was 35 mm/h, and C-reactive protein was negative. The aspirated sample from the mass demonstrated only calcium amorphous crystals and no growth of bacteria.

Ongoing discharge, along with lack of diagnosis of secondary hyperparathyroidism, made open excision of the calcified mass the most appropriate measure for this patient. A posterior approach was used. Just after incision of the superficial fascia, the mass was seen as a fibrous thickening of the well-formed capsule (Figure 2A), and a large amount (almost 50 cm^3) of a high-consistency puslike fluid exited the weakened areas of the wall (Figure 2B). The mass surrounded the ulnar nerve, extended to the proximal triceps tendon and muscle, and completely involved the olecranon bursa. The nerve was dissected and freed completely. In addition, as parts of the posterior capsule would need to be resected for removal of the entire mass, the elbow joint had to be opened. Time-consuming irrigation with several liters (almost 12) of sterile saline was required to clear the joint and surrounding tissues of calcified debris.

Histopathologic evaluation of the excised mass showed nodules containing amorphous calcified material, macrophages, and multinucleated giant cells. Bands of dense fibrous tissue separated these nodular structures. After 7 days with the elbow splinted in near full extension because of the potential risk for skin necrosis, ROM exercises were started, and full ROM was achieved in a few days. The patient was followed up for 2 months. There were no complications. During this period, hemodialysis was modified to prevent recurrence. A kidney the patient had been awaiting for

Table I. Review of Literature on Differences Between Hemodialysis-Related and Classic Tumoral Calcinosis[4]

	Tumoral Calcinosis in Hemodialysis Patients	Classic Tumoral Calcinosis
Age distribution, y	35–46 (average; 40.4)	3–60 (average; 16.7)
Male/female, No.	3/2	15/9
Normal serum calcium, No. (%)	None	6/9 (66.7%)
Hypercalcemia, No. (%)	3/4 (75%)	3/9 (33.3%)
Normal serum phosphate, No. (%)	None	6/12 (50%)
Hyperphosphatemia, No. (%)	4/4 (100%)	6/12 (50%)
Normal alkaline phosphatase, No. (%)	2/3 (66.7%)	8/11 (72.7%)
Multiple/solitary, No. (%)	2/3 (66.7%)	6/13 (46.1%)
Osteopenia, No. (%)	1/5	None
Renal failure, No. (%)	5/5 (100%)	None
Hemodialysis duration	3 mo–6 y	None
Constituent, No. (%)	4/5 calcium phosphate	All calcium phosphate
Spontaneous remission	Maybe	Maybe

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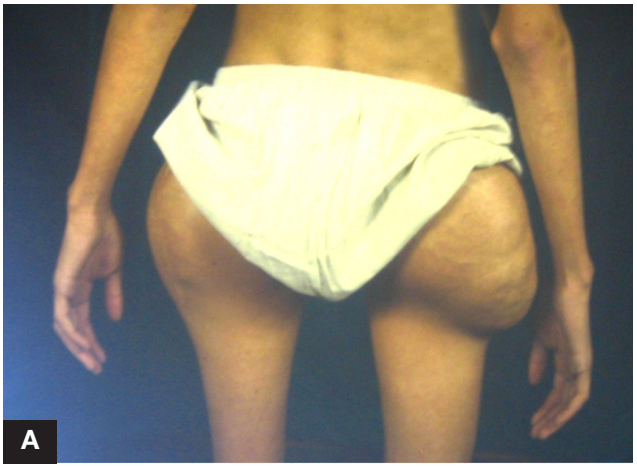
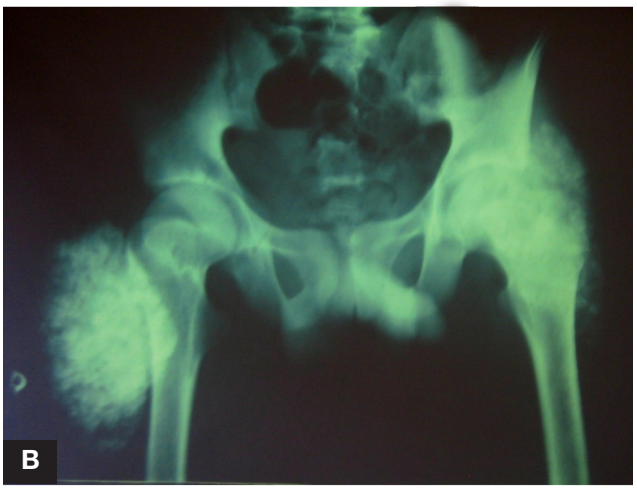
Table II. Review of 18 Primary/Secondary Tumoral Calcinosis Cases Reported in the English Literature

Case	Reference	Age, y	Sex	Presentation	Involvement Location	Renal Failure	Dialysis Duration, y	Kidney Transplant	Laboratory Data	Medical Treatment	Surgical Treatment	Outcome
1	Mozaffarian et al[10] (1972)	46	M	Painless mass	Hips, shoulders	—	—	—	Ca normal, P increased	P deprivation by antacids	Previous resections	Response
2	Suzuki et al[4] (1979)	37	M	Mass, decreased ROM	L cubital area, hips	+	2	—	Ca & P increased, PTH ?	Low-Ca dialysis	Incomplete resection	Response
3	Eisenberg et al[11] (1990)	50	M	Decreased ROM	Shoulders, R fibular head	+ (HTN)	4	—	Ca increased, P & PTH normal	Modified hemodialysis	—	Dead
4	Pecovnik-Balon & Kramberger[12] (1997)	60	F	Pain	R clavicle	+	4	—	Secondary hyperparathyroidism	Low-P diet	Total parathyroidectomy	Response
5	Minisola et al[13] (2000)	37	M	Pain	Hips, shoulders	+ (GN)	3	—	Ca increased, P & PTH normal	Sodium thiosulfate	—	Patient failed to follow up
6	Phanish et al[14] (2000)	25	M	Pyrexia, pain, decreased ROM	L shoulder	+ (RN)	8	—	Secondary hyperparathyroidism	Intravenous pamidronate, P binders, low-Ca diet, dialysis	Transplantation after 6 months	Response to nonoperative Treatment
7	Möckel et al[3] (2005)	40	F	Swelling, joint discomfort	Hips, shoulders	+ (GN)	16	2× rejected	Secondary hyperparathyroidism	P binders, modified diet, dialysis	Resection	Temporary improvement, local recurrence
8	Möckel et al[3] (2005)	20	M	Swelling, decreased ROM	R hip & shoulder, L elbow & wrist	+ (GN)	5	1× rejected	Secondary hyperparathyroidism	—	Subtotal parathyroidectomy	Rapid regression, except of hip
9	Huang et al[15] (2006)	54	M	Pain, mass	L shoulder	+	6	—	Ca normal, P & PTH increased	Low-Ca dialysis	?	?
10	Ovali et al[9] (2006)	23	F	Mass	Hips, R elbow	—	—	—	?	—	Resection	Response
11	Magarkar et al[16] (2006)	13	F	Swelling, normal ROM	L elbow	—	—	—	?	?	Resection	?
12	Seyahi et al[17] (2006)	63	F	Severe pain, decreased ROM	R hip	+ (GN)	7	—	Ca normal, P & PTH increased	Vinpocetine, P binder, paricalcitol	—	Response
13	Alam et al[18] (2007)	2	M	Decreased ROM	R hip & knee, chest	—	—	—	Normal	?	?	?
14	Kadowaki et al[1] (2008)	67	F	Mass, pain after 3 y	L great toe	—	—	—	Normal	—	Excisional biopsy	Response
15	Tuy et al[19] (2008)	60	F	Pain, decreased ROM	Neck, gluteal area	+ (SCD)	2	—	Ca normal, P increased	—	Resection	Response
16	Tarrass & Benjelloun[20] (2008)	23	F	Swelling, discharge	Elbows	+ (GN)	9	—	Secondary hyperparathyroidism	—	Subtotal parathyroidectomy	?
17	Cohen & Parikh[21] (2007)	36	F	Painless nodules	Hips, shoulders, L wrist	+ (GN)	10	1× rejected	Secondary hyperparathyroidism	PTH suppression?	Partial parathyroidectomy	Response
18	Aghaghazvini & Shirani[22] (2008)	17	M	Mass, decreased ROM	Hip	—	—	—	Ca & PTH normal, P & 1,25 dihydroxyvitamin D increased	—	Resection suggested	?

Abbreviations: Ca, serum calcium; GN, glomerulonephritis; HTN, hypertension; P, serum phosphate; PTH, serum parathyroid hormone; RN, reflux nephropathy; ROM, range of motion; SCD, scleroderma.





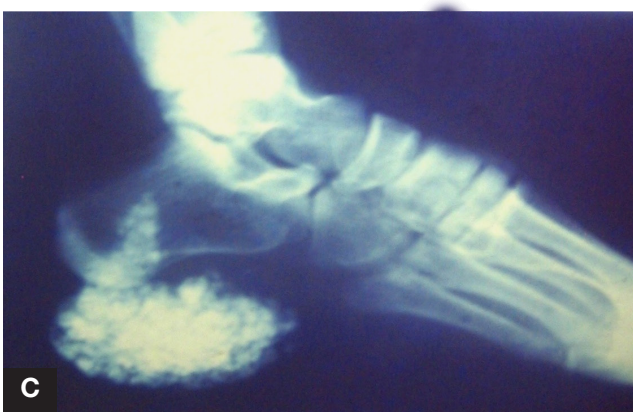


Figure 3. Case 2. Patient had primary tumoral calcinosis. (A) Asymmetric bilateral masses on lateral aspect of hips. (B) Simple radiography shows typical view of tumoral calcinosis, both masses excised during a single surgical session. (C) Recurrence of disease in left foot after 7 years.

several years became ready 2 months after surgery, and he was admitted for renal transplantation. Twenty-five days after successful transplantation, the patient, who had been undergoing immunosuppressive treatment, experienced a full-blown fatal sepsis and, after 5 days in the intensive care unit, expired. Even during this critical period, there was no sign of recurrence or local infection in the operated elbow.

Case 2

In June 1999, a 22-year-old man presented with a painless mass around the right hip. The mass had been insidiously enlarging for about 6 months. Two months after onset, the left hip became similarly involved. There was no history of trauma or other significant point in the patient's medical history. Family history was negative for any related problem, as was history of medication use. Physical examination revealed only firm lobulated masses around the hips and no constitutional signs (Figure 3A) or erythema or warmness on the overlying skin. The nontender, 10×18-cm mass on the right hip moderately limited the abduction/adduction and rotational motions of that hip, whereas the slightly tender, 10×12-cm mass on the left hip interfered only with the extremes of motions. The right hip had a positive Trendelenburg sign, and the patient exhibited a subtle limp while undressing. Simple radiographs showed lobular calcifications where the masses were located (Figure 3B). Serum calcium; phosphate; 1,25 dihydroxyvitamin D; and alkaline phosphatase levels were normal, and no abnormalities in renal or other functions were detected in repetitive sampling. Serum PTH level was not assessed.

Given the diagnosis of primary TC made on the basis of the clinical and radiologic presentation, the 2 lesions were resected through lateral incisions during a single operation. Viscous, chalky, white material (containing calcium salts, according to later analysis) exited the masses immediately after the surrounding well-formed capsules were punctured. Both masses involved the hypodermis, and to some extent, the bulk of the tensor fascia and gluteus medius; the right mass extended to the gluteus maximus. On the right side, the sciatic nerve was explored and preserved, but it had not been in the mass. Histopathologic studies showing calcified nodules separated by fibrovascular bands confirmed the diagnosis of TC.

The patient was ambulated within 2 days, and, after wound healing and 2 months of physical exercises, near normal ROM and good function were achieved. As of 10-year follow-up, there was no local recurrence; after 7 years, however, a painful mass with similar characteristics gradually developed in the plantar surface of the left foot. Although the laboratory profile showed no changes with this development, simple radiography showed another lobular calcified mass (Figure 3C). Pain and difficult shoe-wearing led to another surgical resection, using a medial approach. Difficult exposure and complex local anatomy resulted in incomplete resection of the calcified lesion, and pathologic studies led to the same diagnosis. Although wound healing and easy, unsupported walking took longer now than after the first operation, after 4 months the patient had no complaint about his shoes and no significant pain. There has since been no recurrence, either locally or in other locations.

Discussion

TC, first described by Duret[5] in 1899, is a rare disease characterized by calcified soft-tissue masses near large joints.[6] Progressive lipocalcinogranulomatosis, the term introduced by Teutschländer[7] in 1935, was replaced with tumoral calcinosis, proposed by Inclan and colleagues[8] in 1943. Renal failure, genetic disorders, and recurrent soft-tissue microtrauma are among the main causes of TC.[9] Tumoral calcinosis, at that time, applied to idiopathic cases. Associated disorders, including chronic renal failure and hypervitaminosis D, were classified separately. Although the nomenclature differs in published reports, most authors apply the clinicopathologic term tumoral calcinosis to all types of significant soft-tissue calcification around large joints. TC can be subdivided etiologically into a primary type with no associated disease, and a secondary type that follows other disorders, particularly chronic renal failure.[1,9] In some reports, especially earlier ones, primary TC was the most common type.[1] With routine use of hemodialysis for ESRD over recent decades, however, most cases of TC are now associated with ESRD. In 1979, Suzuki and colleagues[4] reviewed the literature and identified several differences regarding classic and hemodialysis-related types of TC (Table I). In Table II, we present our brief review of reports of TC in the English literature over the past 4 decades.[10-22] This review can be helpful in evaluating the etiology and the related treatment options for this rare entity.

Patients with idiopathic TC typically present with painless masses in juxta-articular locations. Hips are most often involved, followed by elbows and shoulders, though knees, hands, and feet also can be affected.[18] Idiopathic TC usually occurs in the first or second decade of life, typically in young African men, [3] and approximately one-third of the cases exhibit familial inheritance. It typically presents with swelling around large joints. The swelling is generally painless and increases slowly over a year. It predominantly affects the extensor surfaces, but the joints themselves are normal. Symmetrical involvement is common, but constitutional signs and symptoms are characteristically absent.[5,17,18]

Idiopathic TC has 2 distinct subtypes: primary normophosphatemic TC, in which serum calcium and phosphate levels are normal, and primary hyperphosphatemic TC, in which calcium level is normal but phosphate level is elevated.[18,23] Case 2 in the present report and 6 cases (1, 10, 11, 13, 14, 18) listed in Table II had the primary form of TC; our case and cases 13 and 14 in Table II had normal serum phosphate levels. These patients ranged in age from 2 years to 67 years—similar to the age range (3 years-60 years) noted by Suzuki and colleagues[4] (Table I). With our case 2 added to the primary TC cases listed in Table II, the male:female ratio is 4:3, compared with 15:9 in the review by Suzuki and colleagues. In our review (Table II), hips (cases 1, 10, 13, 18, plus our case 2) and elbows (cases 10, 11) were the most common sites of involvement of primary TC.

In recent years, secondary TC associated with renal failure and prolonged hemodialysis is detected more often. Although other conditions, including hyperparathyroidism, pseudoxanthoma elasticum, malignancy, sarcoidosis, scleroderma, and hypervitaminosis D, can underlie secondary TC,[1] ESRD seems the most common cause. Persistently elevated serum calcium–phosphate product is the major contributing factor in development of TC. Etiologically, this product, under the influence of elevated pH and serum magnesium levels and local tissue injuries, sometimes leads to extremely pronounced soft-tissue calcifications.[24] In healthy adults, this product ranges from 25mg/dL to 45 mg/dL. In cases of chronic renal failure, serum phosphate rises, and serum calcium may drop.[25,26] When their product is more than 45 mg/dL (~5.8 mmol/L), calcium phosphate precipitates in the soft tissues.[27,28] Patients with ESRD often have secondary or tertiary hyperparathyroidism.[29,30]

In our case 1, and in cases 3 and 5 in our review (Table II), serum PTH level was normal, and hyperparathyroidism could not be detected. However, all the patients undergoing hemodialysis treatment had elevated serum levels of calcium, phosphate, or both (high calcium–phosphate product). Age ranged from 35 years to 46 years in the review by Suzuki and colleagues[4] and from 20 years to 63 years in ours. With our case 1 added to the hemodialysis-related cases listed in Table II (cases 2-9, 12, 15-17), the male:female ratio is 7:6, compared with 3:2 in the review by Suzuki and colleagues. Duration of dialysis in our review ranged from 2 years to 16 years, obviously longer than the range of 3 months to 6 years found by Suzuki and colleagues. In our review, shoulders (cases 3-9, 17), hips (cases 2, 5, 7, 8, 15, 17), and elbows (cases 2, 8, 16, plus our case 1) were the sites most commonly involved in TC associated with ESRD.

The diagnosis of TC can be rendered on the basis of radiographic findings plus knowledge of the patient's medical history.[3] Plain radiographs in both primary and secondary TC are often diagnostic and show multiple areas of well-circumscribed nodular masses with fibrous septa giving a cobblestoned or chicken-wire appearance. Radiographs used with a horizontal beam and CT scans may show the "sedimentation sign" caused by dependent pooling of the mineral portion, creating a fluid calcium level. Bone scintigraphy with radiolabeled phosphate compounds (technetium-99m methylene diphosphonate) demonstrates increased uptake in the soft-tissue masses. On magnetic resonance imaging (MRI), the lesion is hypointense on all sequences, with areas of high signal intensity on T-2 and inversion recovery sequences corresponding to edema caused by an inflammatory response to pathologic calcification. The sedimentation sign can exist on MRI sequences as well.[18,31]

On the basis of differing etiologies for the 2 almost distinct types of TC, treatment can be expected to differ to an extent as well, and this is backed up by a literature review. In primary TC, spontaneous regression seldom

occurs. Approaches for managing primary (idiopathic) TC are the treatment mainstay, surgical resection of the calcified mass (may need to be performed several times given the recurring nature of the problem, as in our case 2), and other, more comprehensive therapeutic and perhaps prophylactic measures, such as low-calcium and low-phosphate diets, phosphate deprivation by oral aluminum hydroxide, and induction of phosphaturia by acetazolamide.[32,33] Indications for surgical removal are pain, recurrent infection, ulceration, and functional impairment.[18]

It is clear that calcium- and phosphate-restricted diets, and use of a phosphate binder with a meal or acetazolamide, are helpful only in patients with defective phosphate metabolism (hyperphosphatemia).[18] Radiotherapy,[8] special diets, and oral and parenteral medications, including corticosteroids and corticotropin, are treatments that have not proved curative.[34] Our review (Table II) revealed that, except for case 1 (treated with oral antacids) and case 13 (treatment unknown), the performed or suggested treatment was resection (cases 10, 11, 14, 18, plus our case 2). The outcome of resection in cases 10 and 14 and our case 2 was acceptable.

The results of Mozaffarian and colleagues[10] with administration of oral aluminum hydroxide to decrease body phosphates were promising, and this treatment could be used in hyperphosphatemic primary TC.

Hemodialysis-related or, better, ESRD-related TC as the idiopathic type has been addressed with many therapeutic approaches. Given the pivotal role of high serum calcium and phosphate levels in the pathophysiology of this type of TC, calcium- and phosphate-restricted diets and dialysated, and phosphate binders were the first measures used for its management. Of course, phosphate binders, except aluminum-containing binders, are more acceptable for this purpose when aluminum toxicity has been implicated in the pathogenesis of ESRD-associated TC.[35]

Sodium thiosulfate was used to manage hemodialysis-related TC by Minisola and colleagues[13] and was suggested by Drüeke,[36] but their results were not clear. Vinpocetine is a semisynthetic vinca alkaloid that acts through calcium channel–blocking activity and through voltage-gated sodium channel–blocking activity. Seyahi and colleagues[17] were successful in using vinpocetine in the medical treatment of TC in a patient (case 12, Table II) on long-term dialysis.[17] Ueyoshi and Ota[37] found elimination of calcified masses in 8 patients after administration of vinpocetine 15 mg/d for 3 to 12 months.[37]

Phanish and colleagues[14] achieved good results with administration of intravenous pamidronate in a patient (case 6, Table II) with TC and pyrexia in the context of ESRD. Kidney transplantation 6 months after symptom onset, along with use of phosphate binders and a low-calcium diet and dialysate, might also help in the treatment of this case. In fact, surgical removal is not routinely indicated in these patients, and even a biopsy should be avoided because of the risk of infection. Möckel and colleagues[3] noted local recurrence and temporary recovery with use of surgical resection in 1 patient (case 7, Table II). In our case 1, aspiration of the patient's mass led to permanent fistula discharge of hepatitis B virus–containing materials—which forced us to resect the mass. Tarrass and Benjelloun[20] faced the problem of discharge of calcified material too, but they performed a parathyroidectomy and achieved a good outcome (case 16, Table II). Overall, because of probable recurrence and no correction of the cause of TC, the indication for removal of the calcified mass in patients undergoing hemodialysis is limited to those unresponsive to other measures and those with an infected or fistulated mass (opposite to the treatment of the idiopathic type of TC). Also in contrast to the idiopathic types, because of underlying secondary or tertiary hyperparathyroidism in most of these patients, subtotal or total parathyroidectomy seems the most reasonable etiologic treatment when medical treatment fails. Cases 4, 8, 16, and 17 (Table II) involved parathyroidectomy after failure of medical measures, and the response to treatment was appropriate and significant.

It is to be concluded that, for diagnosis and management of TC, symptomatic periarticular soft-tissue swellings initially require clarification with conservative radiographs. Whenever the diagnosis of TC is made, calcium–phosphate metabolic parameters and renal function must be checked. In primary TC, resection of the mass should be considered on the basis of the patient's symptoms. In hyperphosphatemic types of TC, oral aluminum hydroxide may be helpful and may eliminate the need for a surgical procedure, which may be followed by recurrence locally or at another site. In hemodialysis-related types of TC (the most common form of secondary TC), a conservative approach is initially indicated—use of phosphate binders, optimization of dialysis, and start of a suitable diet. If this conservative approach fails, or the therapeutic outcome is unsatisfactory, subtotal parathyroidectomy should be performed. Surgical resection of TC can be performed as a last resort, though this is insufficient therapy on its own.[3]

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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