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Special Feature

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Knuckle cracking: secondary hyperparathyroidism and what your mother did not tell you

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Abstract

Introduction. Secondary hyperparathyroidism in end-stage renal disease patients has protean musculoskeletal manifestations. Some of our dialysis patients spontaneously vocalized that they had lost the ability to crack their knuckles and then experienced gratifying restoration after surgical parathyroidectomy. We propose that the physiology and mechanical basis of knuckle cracking would be affected by parathyroid-related mineral and bone disorders.

Methods and results. We surveyed all of our chronic dialysis outpatients who had undergone surgical parathyroidectomy. Thirteen (∼12% of the population) individuals were identified: eight males, age 37.7 ± 12.5 years old, dialysis duration of 10.2 ± 7.0 years and peak preoperative intact parathyroid hormone (PTH) levels of 2344 ± 900 pg/mL. Seven patients had no recollection of knuckle cracking issues, with surgery as remote as decades. Six individuals were able to provide adequate histories: four had postoperative restoration of knuckle cracking and expressed great satisfaction from the emotional relief from what appeared to be habitual knuckle cracking. Two of these patients reported rapid return of cracking, occurring in less than ∼2 weeks.

Conclusions. This is the first report of loss of knuckle cracking due to hyperparathyroidism and its cure in 67% of patients, following surgical parathyroidectomy. We propose that parathyroid (e.g. calcific) changes in articular structures (tendons, ligaments) cause reduced elasticity, limited joint surface separation upon flexion, attenuation of cavitation and thus, loss of the audible crack upon vacuum collapse. The psychological 'release' from habitual knuckle cracking may be a motivator from some patients to adhere to complex parathyroid regimens or to pursue surgical intervention.

Keywords: hyperparathyroidism; parathyroidectomy; renal osteodystrophy

Introduction

Articular and other musculoskeletal manifestations of secondary hyperparathyroidism have been a longstanding concern for patients with chronic kidney disease. In the setting of abnormalities in divalent ion and parathyroid homeostasis, there is much involvement of tendons leading to a loss of elasticity and fragility due to soft tissue calcium deposition. Dialysis patients are known to be particularly susceptible to tendon ruptures at multiple sites, including the fingers, quadriceps, patella, Achilles, triceps and biceps [1–5]. In the presence of ongoing disorders of bone and mineral metabolism, these tendon ruptures result in swan neck as well as other finger deviation deformities, which lead to loss of function requiring difficult reconstructive surgeries.

The prevention or treatment of secondary hyperparathyroidism relies on patient adherence to complex treatment regimens. Treatment compliance is challenging in the dialysis population as these patients require significant motivation in the form of being convinced of the risks from inadequate control for motivation. Unfortunately, educational discussions, review of parathyroid laboratory results or even symptomatic arthritis are frequently insufficient to convince patients that renal osteodystrophy has commenced. Ultimately, to maximize patient outcomes, acknowledgement of disease activity should occur early, and treatment compliance must be maximized prior to severe manifestations such as fractures.

We believe that this case series is the first report of the loss of ability to crack one's knuckles as being a manifestation of hyperparathyroidism: a self-recognized sign of advancing disease that dismayed our patients might be a motivator for improved adherence, which was often reversible upon surgical parathyroidectomy. We hypothesize that soft tissue calcification reduces tendon elasticity and restricts finger joint motion. This limitation reduces the vacuum and cavitation caused by phalanx flexion, and thereby attenuates or eliminates the audible 'pop' or cracking of the knuckles.

Methodology and results

This series represents the clinical experience at the University of Florida Health Shands Hospital, Gainesville, FL, following surgical subtotal parathyroidectomy in patients either referred to or originating in our chronic outpatient dialysis program. Details about the parathyroid status were only available for the latter group; however, all patients underwent surgery due to failure to control parathyroid disease with medical regimens that included dietary restrictions, phosphate binders, vitamin D analogs and calcimimetics. Patient symptoms were noted in real time as part of their postoperative care or long term during their ongoing follow-up in our clinical program. Data collection and reporting adhere to our institutional review board policies (University of Florida, Gainesville, FL, USA).

We provided clinical follow-up to 13 patients (12 hemodialysis and 1 peritoneal dialysis) who had undergone subtotal parathyroidectomy, representing ∼12% of the total population. There were eight (62%) males, aged 37.7 ± 12.5 (mean ± SD) years, and with a dialysis duration of 10.2 ± 7 years. Of the 10 patients for whom there were preoperative data available in our medical records, the peak intact PTH levels were 2344 ± 900 pg/mL. Postoperative levels in all patients confirmed adequate reduction in parathyroid gland mass, with levels initially being <150 pg/mL.

Of the 13 patients, seven were unable to provide any information as to their ability to crack their knuckles. The reasons provided were as follows: they would not know since they had not practice that behavior; they did not notice or they had no recollection since the surgery was in the distant past (e.g. decades). Thus, there were six evaluable patients:

There was no improvement in four patients two patients spontaneously vocalized their having completely lost the ability to crack their knuckles and how happy they were that postoperatively knuckle cracking was completely restored. In one of these individuals, the return of cracking occurred within 2 weeks of their parathyroid surgery. One additional patient described how preoperatively he could only crack two knuckles in one hand and three on the other. Postoperatively, this patient reported restoration of knuckle cracking in all fingers. The remaining patient described a total loss of all audible knuckles popping with maintenance of only an inaudible cracking sensation. This patient stated that after adequate surgical intervention his audible knuckle cracking ability was completely restored.

There was no perception of a deficit before or after surgery in two patients.

Discussion

There is longstanding interest in articular 'noises', which have been described as joint cracking and a variety of other articular pops, clunks, releases, snaps or ligamentous strums and clicks [6]. There has also been ongoing concern that habitual knuckle cracking might eventually injure joints. Indeed, there is rare case reporting of complications related to frequent knuckle cracking, including the development of knuckle pads, fractures or dislocation [7, 8]. Nevertheless, and despite the frequent attestations to 'stop cracking your knuckles!' from lay counselors (e.g. your mother), there is yet to be clear evidence of harm. In a prospective study, Castellanos and Axelrod [9] reported that, compared with 226 control subjects, 74 habitual knuckle crackers had functional hand impairment (hand swelling, lower grip strength), but this was associated with a history of manual labor and the behavior characteristics of nail biting, smoking and alcohol consumption. There was, however, no objective difference in the incidence of arthritis between the groups. In Deweber *et al.* retrospective case–control study [10], osteoarthritis was no more prevalent in subjects who knuckle-cracked than in those who did not. Similarly, Swezey and Swezey [11] were not able to demonstrate a correlation between knuckle cracking and metacarpal phalangeal joint degeneration in geriatric patients. Individuals, especially habitual crackers, do subjectively report the importance of a vaguely described emotional relief or 'release' upon cracking.

Perhaps the paucity of harm is due to the benign mechanism of the 'pop' [6, 12, 13]. Based on the elegant studies of Unsworth *et al.* [13], it is believed that rapid flexion of the joint separates the articular surfaces, and the resultant vacuum creates cavitation within the articular space. Subsequent sudden collapse of the vacuum then causes the popping sound. The extent of bubble formation within the joint space depends on the articular forces and the vapor pressure of the synovial fluid. The joint cannot be re-cracked until the residual gas bubbles are fully reabsorbed into the fluid, which takes ∼20 min. Protopapas *et al.* [6] described clinical entities that would prevent the negative pressures or joint separation necessary for joint cracking, and these include tense or rigid periarticular muscles, abnormally short ligaments, invagination of abnormally lax synovial soft tissue and low elasticity of the connective tissue.

We believe that, in secondary hyperparathyroidism, the mineral abnormalities induce soft tissue calcification that limits tendon elasticity and deformability. When combined with joint discomfort, the resultant restriction to the speed, range of motion and separation of the articular surfaces would reduce the vacuum, which, in turn, would attenuate or eliminate the noisy collapse of cavitation. This pathophysiologic mechanism would be exacerbated by, and in addition to, structural bone changes in the joints from hyperparathyroidism, osteoarthritis or other articular disorders that would limit flexion and thus cavitation. Abnormalities in tendons due to parathyroid disease have been recognized since the 1960s [14, 15].

Numerous case reports, series and literature reviews describe tendon rupture in primary [16, 17] and secondary hyperparathyroidism [2, 4, 14, 15, 18–21]. Most articles reported tendon rupture in patients on long-term hemodialysis, as well as some individuals who were on peritoneal dialysis or had received kidney transplantation [5]. The term 'uremic tendonopathy' was coined by Hofmann *et al.* [22]. In one of the largest reviews, Jones and Kjellstrand [1] describe 44 patients with tendon ruptures that were related to the duration of end-stage renal disease, as well as the degree of hyperphosphatemia and hyperparathyroidism. In a review of 24 cases, Shah [4] also related tendon problems to the length of time on dialysis. De Franco *et al.* [23] and Shiota *et al.* [21] highlighted that these articular problems were associated with hyperparathyroidism without there being evidence for β2-microglobulin amyloid involvement. The proposed pathophysiology is two-fold: (i) dystrophic calcification with diminished integrity of the tendon, which we believe corresponds to the loss of joint cracking ability; and (ii) subperiosteal bone resorption leading to repeated micro- or eventual macro- (e.g. total) avulsion of the tendon [14, 15, 18, 24]. The latter's clinical presentation would depend on whether

the rupture occurred within the fragile tendon or at the site of bone insertion, and may be complicated by hemarthrosis. Most reports describe the association of weight-bearing joints [2] with tendon ruptures. The tears are seemingly spontaneous or can occur after minimal exertion or trauma, and are not rarely bilateral. Although most cases, thus, are at the quadriceps, patella or Achilles tendons, there are also patients with ruptures at the triceps, biceps or abdominal muscle tendons. Involvement of the hands is also well recognized [3] and leads to limited function, decreased range of motion, deformities, finger deviation and pain.

Of the six patients in our series that had any recollection of their knuckle signs or symptoms (e.g. excluding those with surgery in the remote past), 67% reported return of what had been the lost ability to fully crack their knuckles. Quite notable was the rapidity at which knuckle cracking improved postparathyroidectomy: between 1 and 2 weeks. It also highlights the potential reversible nature of these soft tissue disorders and is reminiscent of effective treatment, leading to improvements in skin and conjunctival calcium depositions as well as in tumoral calcinosis. Most surprising to us was how important knuckle cracking was to some of these patients, aptly described as 'habitual crackers'. Although difficult to quantify, individuals appeared very dismayed by loss of this chronic habit and delighted to spontaneously report to us the return of this ability (we have yet to receive comment from their family members). One wonders whether, in select patients, the loss of knuckle cracking will be a sentinel event that with proper counseling could lead to improved adherence to the bone and mineral disease regimen. We propose to add this end-organ manifestation to our armamentarium of counseling tools to promote better compliance to these complex dietary and pharmaceutical strategies.

In conclusion, we report a previously unappreciated yet potentially reversible end-organ manifestation of secondary hyperparathyroidism. The self-described importance of some patients' ability to crack their knuckles may be a motivator to adhere to the prescribed regimen and improvements can be a gratifying response to successful treatment.

Conflict of interest statement. None declared.

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