CASE REPORT

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Hyperparathyroidism and Psychosis: Possible Prelude to Murder

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ABSTRACT: The authors present a case of a middle aged attorney who suffered from hyperparathyroidism and a psychotic disorder. It is possible that the hyperparathyroidism may have precipitated an acute psychotic delusional rage leading to an attempted mass murder. They discuss the relationship between hyperparathyroidism and neuropsychiatric symptoms in consideration of available research.

KEYWORDS: forensic science, hyperparathyroidism, psychosis, insanity defense

Primary hyperparathyroidism is defined as a generalized disorder of calcium phosphate and bone metabolism that results from the increased secretion of parathyroid hormone (PTH). The excessive concentration of the circulating hormone can lead to hypercalcemia and hypophosphatemia, with great variation in manifestations (1). Excess PTH results in increased resorption of calcium from the skeletal system and increased absorption of calcium by the kidneys and gastrointestinal system (2). The disorder often is asymptomatic for years, but eventually leads to a variety of clinical presentations including nephrolithiasis (kidney stones), peptic ulcers, mental changes, and pathological bone fractures. The disorder can be remembered by the medical pneumonic: bones, kidney stones, abdominal groans, and psychic moans (and fatigue overtones) (3). Further, this disorder can produce a variety of other symptoms related to the urinary system such as nocturia, polydypsia, polyuria, and gastrointestinal symptoms such as nausea, heartburn and constipation as well as pruritis.

Psychiatric symptoms include depression, concentration and memory problems, and sometimes, even psychosis. Other symptoms such as anorexia, muscle weakness, and fatigue are sometimes attributed to psychiatric causes. This condition, however, is detected by elevated calcium levels together with elevated parathyroid hormone levels. A high level of suspicion is warranted, as even modestly elevated calcium levels less than 12 mg/dL have been associated with significant psychiatric symptoms (4). Clouding the

picture further, the severity of psychiatric symptoms is not directly related to the level of calcium increase (5).

In a recent review of the literature, Zahrani and Levine (6) found that some patients with primary hyperparathyroidism exhibit "symptoms that constitute a poorly characterized neuropsychiatric syndrome, from decreased attentiveness and ability to concentrate to severe depression." Further, it was noted that in ten percent of persons with primary hyperparathyroidism, parathyroidectomy is not completely curative. That is, some persons continue to suffer from residual symptoms post-operatively. These findings support an earlier study (7) in which it was shown that the majority of persons who suffer from hyperparathyroidism with psychiatric symptoms typically have affective syndromes and/or cognitive disturbance and most symptoms are reversed in a majority, but not all patients by parathyroid surgery.

There have been case reports in the literature characterizing persons with hyperparathyroidism and psychotic symptomatology, but no large well controlled studies. Spivak, Radvan, Ohring, and Weizman (8) described two such cases. In one case, a 68-year-old woman showed "disorganized thought process with delusional persecutory ideation." In the other case, a 47-year-old female was noted to suffer from disorganized thought process, hallucinations, and delusions of persecution and reference. Both cases showed a complete resolution of psychotic symptoms following surgical excision of growths in the parathyroid glands.

Cunningham and Anderson (9) described the case of a 67-yearold woman diagnosed with hyperparathyroidism and delusional depression with mood congruent delusions. Interestingly, this woman experienced visual hallucinations that were attributed to her problems with hypercalcemia. She experienced a remission of neuropsychiatric symptoms, albeit slowly, with a treatment regimen of electroconvulsive therapy and stabilization of blood calcium levels.

Thurling (10) described a 52-year-old woman. This case is somewhat analogous to the case report presented below. The woman, called "Ms. J," had a past history of schizophrenia. It was found that Ms. J experienced a marked deterioration in mental state following elevated PTH and hypercalcemia secondary to a parathyroid adenoma. In this case, her mental state quickly cleared following surgical removal of the growth. The author hypothesized that the hypercalcemia likely caused a "dysequilibrium" of brain functioning. This was explained as "the synthesis and release of central neurotransmitters and post-synaptic receptor sensitivity are calcium dependent. Calcium facilitates dopaminergic activity at the various stages of dopamine synthesis

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and release." Of course, disruption of dopamine synaptic functioning has been implicated in the expression of psychotic symptoms such as those found in schizophrenia or secondary to dopamine agonists prescribed for persons with Parkinson's disease.

Case Report

On a fall day at a major midwestern university in 1992, a graduate student pulled his car alongside the building where his scheduled course was taught. He got out of the car and proceeded to the classroom where his afternoon course was meeting. In his hands he carried an M-1 .30-caliber carbine with a folding stock. He entered the classroom whereupon he lifted the gun, pointed it at the class full of students, pulled back the bolt, and pulled the trigger. The students observed their classmate, C.K., moving the gun in a sweeping motion toward them. C.K. seemed alarmed when the gun jammed. He attempted to work the bolt to clear the jam; two bullets were stuck in the chamber. The students, scared and panicked, fled quickly from the class. Dismayed, C.K. left the building and returned home in his car. He was later arrested and charged with multiple counts of attempted murder. If C.K.'s gun had not jammed on that brisk October day, one of the largest mass murders in U.S. history could have been perpetrated.

C.K. was arrested shortly after the incident described above. Police found the M-1 .30-caliber carbine in the back seat of his car. Upon arrest, a police officer noted that C.K. was talking to himself quietly. His speech was inaudible. C.K. remarked aloud that his classmates were "all members of a conspiracy in order to prevent him from listening to the class professor." While searching his home, investigators found numerous handwritten notes containing what appeared to be surveillance information. That is, C.K. had taken notes on various classmates' behavior he observed and considered to be suspicious. Other guns were found in the apartment along with blood stained clothing. It was later determined that the bloody clothing found in his apartment was blood from his urine due to increased levels of PTH and calcium in the blood.

The police collected evidence, interviewed eyewitnesses, and overall, built a solid case against C.K. As more information was gathered on C.K.'s background history, what emerged was a quite unusual profile. C.K. was a single man in his early forties who lived alone. He had completed his undergraduate studies in the late 1970s and successfully obtained a Juris Doctorate in 1989. His education also included numerous courses in accounting and actuarial sciences. The records showed that he had made three unsuccessful attempts to gain acceptance to a doctoral program in actuarial sciences.

C.K. had no significant history of previous arrests or incarcerations. However, it was noted that he had assaulted his mother during his teenage years and that they had not spoken to each other for over two decades. Reportedly, he never knew his father. C.K. was described by many as someone who preferred to be alone. Although he worked consistently over the years, there were many accounts of C.K. not getting along with his co-workers, or his supervisors. After working for an insurance company for about seven years, he applied for a promotion, but was denied. C.K. was convinced that the denied promotion was a product of racial discrimination (he is of minority status). He became so obsessed with the denied promotion that it began to interfere with his work performance. His co-workers observed that C.K. was sometimes talking to himself. He often voiced complaints about other staff persons taunting him in various ways. For example, he described how a woman employee bumped into him and he interpreted this as a form of sexual harassment. Another co-worker would sometimes tap his fingers on his desk while working. C.K. interpreted this as a provocative act, meant to disturb his concentration. He voiced numerous complaints about other employees and their actions, which he saw as malicious acts intended to provoke him. Finally, his employer insisted that he undergo an evaluation by a psychiatrist. C.K.'s company-ordered evaluation was interpreted by him as a ploy "to get rid of him." The psychiatrist who assessed him concluded that C.K. suffered from paranoid schizophrenia.

C.K. was so incensed by this experience with his employer that he attempted to solicit attorneys who would help him sue the company. However, it appears that no attorney would take the case. He decided to obtain a law degree so that he could protect himself from persons who intended to harass him or discriminate against him. After months of preparation, he applied and was accepted into law school.

Early in his first year, he became seriously ill. He suffered a significant decline in weight, notable loss of energy, and fractured his arm. It was discovered upon further diagnostic testing that C.K. suffered from a parathyroid adenoma with primary hyperparathyroidism. C.K. was hospitalized and underwent various surgical procedures. The parathyroid adenoma was removed and surgical repair to weight-bearing bones was performed to correct pathology (e.g., a steel rod and a bone graft was done to support his legs). He was stabilized on medications to correct abnormally high blood calcium levels.

After a medical leave of absence, C.K. struggled, but managed to complete law school. In addition, he took several courses in actuarial sciences. He planned to obtain a doctorate in this area next. He worked as a law clerk for a period of time. Again, it was documented that he had multiple interpersonal problems with co-workers and supervisors. At one point, he filed a civil law suit against a fellow worker accusing him of assault and libel. The accusations were found to be baseless. He maintained an isolated, schizoid demeanor. Also during this time, the records showed that C.K. had significant financial difficulties and filed for bankruptcy (apparently he had accumulated medical bills well beyond his means to pay). Clearly at this time, C.K. was struggling to cope with numerous problems, at work, in school, with his social life, and financial status.

Discussion

Both professors and fellow students described C.K. as a "loner," an unusual man who sometimes mumbled to himself and did not seem "all there." One professor described that C.K. often made a "clicking" type noise in class and seemed to increasingly labor, as if in pain, while walking. Upon arrest and incarceration, C.K. exhibited behaviors suggesting he was markedly paranoid and acutely psychotic. He was diagnosed with a paranoid delusional disorder by an examining psychiatrist and prescribed an anti-psychotic drug. While awaiting trial, he was hospitalized with renal failure and hypercalcemia. Blood calcium levels were well above normal. A CT scan done at the time showed clear evidence of cerebral atrophy. Eventually, he was stabilized and returned for safe keeping at a forensic psychiatric hospital. Some time later, the court found that C.K. was not responsible by reason of insanity for the crimes as charged, based in part on the evaluation of one of the authors (W.S.L.). It was decided that he should remain hospitalized in a secure setting until removal of disability and minimization of dangerousness could be attained. This decision was based on the post-adjudication assessment conducted by another one of the authors (S.A.B.).

It was clear from the records that C.K. maintained persecutory delusional beliefs over a number of years. Others observed that he talked to himself on occasion. This strongly suggested that C.K. experienced hallucinations, an expression of his schizophrenia. Yet, despite these obvious psychotic symptoms, C.K. was able to complete law school (though his grades were near the bottom of the class) and manage his daily affairs. The fact that the impetus for completing a law degree was to defend himself from others conspiring to wrong him, is quite remarkable. It appears that C.K.'s problems with hyperparathyroidism may have triggered an acute psychotic episode, exacerbating pre-existing psychotic symptoms. It seems that his hyperparathyroidism never fully resolved once his adenoma was removed. He continued to suffer from symptoms and eventually he was rehospitalized years later with renal failure. It is believed that C.K.'s hyperparathyroidism affected him in multiple ways, both physically and mentally, and likely predisposed him to act out in a criminal manner.

Presently, C.K. remains hospitalized. He continues to take antipsychotic medications, but with little effect. Though he does not evidence significant positive symptoms (e.g., hallucinations), he seems paralyzed by negative symptoms (e.g., anhedonia, abulia, asociality). Neuroimaging shows cerebral atrophy which is consistent with a diagnosis of chronic schizophrenia. His prognosis for recovery seems poor.

References

- Fauci. Harrison's principles of internal medicine. 14th ed. New York: McGraw-Hill, 1998;2228.
- Segre GV, Potts JT Jr. Differential diagnosis of hypercalcemia. In: De-Groot L et al, editors. Endocrinology. Philadelphia: Saunders, 1995; 1075.
- St. Goar, W. Gastrointestinal symptoms as due to the diagnoses of primary hyperthyroidism: a review of 45 cases. Ann Intern Med 1956; 46:102
- Okamoto T, Gerstein HC, Obara T. Psychiatric symptoms, bone density and non-specific symptoms in patients with mild hypercalcemia due to primary hyperparathyroidism: a systematic review of the literature. Endocr J 1997;44:367–74.
- Alarcon RD, Franceshoini JA. Hyperparathyroidism and paranoid psychosis: case report and review of the literature. Br J Psychiatry 1984; 145:477–86.
- Zahrani AA, Levine M. Primary hyperparathyroidism. Lancet 1997; 349:1233–8.
- Joborn C, Hetta J, Johansson H. Psychiatric morbidity in primary hyperparathyroidism. World J Surg 1988;(12):476–81.
- Spivak B, Radvan M, Ohring R, Weizman A. Primary hyperparathyroidism. Psychiatric manifestations, diagnosis and management. Psychother Psychosom 1989;51:38

 –44.
- Cunningham SJ, Anderson DN. Delusional depression, hyperparathyroidism, and ECT. Convuls Ther 1995;11(2):129–33.
- Thurling ML. Primary hyperparathyroidism in a schizophrenic woman. Can J Psy 1987;23:785–7.

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