

HYPERCALCEMIA AND PHPT PRESENTING WITH MENTAL HEALTH CRISIS – A CASE REPORT

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INTRODUCTION

Primary Hyperparathyroidism is a condition in which parathyroid gland secretes high levels of Parathyroid hormone and loses the ability to regulate Parathyroid hormone secretion via a negative feedback mechanism with serum calcium level (Geffken 1998). This hormone increases serum calcium by increasing bone resorption, intestinal absorption and decreasing renal excretion resulting in vicious cycle of hypercalcemia (Figure 1).

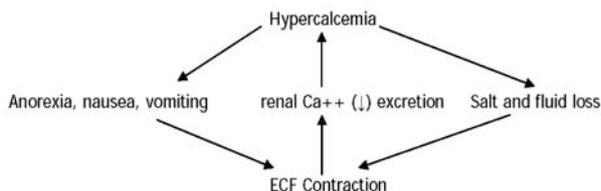


Figure 1. Vicious Cycle of Hypercalcemia (As depicted by Felig P. Endocrinology and Metabolism 3rd edition 1995)

It has long been known to be a general medical condition associated with psychiatric manifestation presenting as depression, anxiety, confusion, disorientation, fatigue, lethargy, emotional lability or occasionally paranoid delusions, hallucinations and catatonia (Gatewood 1998, Spivak 1989). These effects are believed to be mediated through the increased calcium levels seen in hyperparathyroid state (Jimerson 1979).

CASE SUMMARY

AB is a 63 year old single Caucasian man employed as a Transport Manager, living alone was referred by his GP to Crisis Resolution and Home Treatment (CRHT) Team. He was stressed by domestic problems and work related issues. He responded by taking a planned overdose and was not regretful. He had persistent low mood and described irritability, lethargy, poor concentration and depressive cognition. He continued to feel life was not worth living. He was also suffering from dyspepsia and obstructive sleep apnoea. He was being treated with Omeprazole and was using CPAP every night. He also described lower chest pain which was different from his “usual” reflux related discomfort and was associated with exertion for which a cardiology opinion was sought.

He was started on Sertraline which was increased to 100mg/day in a week. His mental state started improving, but he continued to experience fatigue and lethargy. He had a routine blood test, which showed normal results including thyroid function test. However, as an incidental finding we found that his serum calcium was significantly high. His GP was informed and he was referred to an endocrinologist.

During CRHT follow up, we monitored his mental state and also supported him with supportive psychotherapy and psychosocial intervention, in addition to the anti depressant medication mentioned above. During the follow up he had a chest infection, which was treated with appropriate antibiotics. As the crisis resolved and his mental state improved, we transferred his care to the Community Mental Health Team.

While he was being treated for depression and awaiting endocrinologist opinion, his further investigations revealed Primary Hyperparathyroidism (PHPT). His subsequent investigations by endocrinologist revealed Parathyroid adenoma and pituitary adenoma, and the diagnosis of Multiple Endocrine Neoplasia type 1 was established (Table 1).

Table 1. Blood results with Crisis Resolution and Home Treatment Team

Parathyroid Hormone	10.6 pmol/L (↑)
Phosphate	1.01 (=)
Calcium	3.00 (↑)
Corrected Calcium	3.08 (↑)

The diagnosis of primary hyperparathyroidism in hypercalcemia patient is established by determining high level of parathyroid hormones and presence of elevated urinary calcium.

DISCUSSION

PHPT is a relatively rare condition with an incidence of 25 per 100,000 in general population (Bilezikian 2005). It is 2-3 times more common in women (Kelly 2010) with the average age at diagnosis being 55 years (Taniegra 2004). Nearly 80% of patients with nonspecific neuropsychological symptoms are diagnosed mostly after routine laboratory tests showing elevated calcium levels.

Earlier studies have reported psychological symptoms like sleep disturbance, depression, fatigue, irritability, lack of concentration and somatic symptoms (McAllion & Peterson 1989). A study by Rao et al (2004) found worsening of social functioning and emotional problems in patients followed without intervention for 2 years. Up to 10% of patients with PHPT may meet DSM IV-TR criteria for major depression or "major depression due to a general medical condition" (Wilhelm 2003). Our patient presented with a picture suggesting major depression affecting his social functioning and quality of life. Following the treatment of depression, his mood showed most significant improvement however, fatigue showed least improvement. His social functioning and quality of life improved to an extent that he could return to work.

A study by Wilhelm (2003) showed that after Parathyroidectomy, 55% of patients report no further depressive episodes and those who continue to experience depressive symptoms, 90% state that their depressed mood no longer affected their quality of life. In the same study, overall quality of life improved in more than 50% of patients and roughly same numbers of patients were able to discontinue or reduce the dose of the antidepressant. A study by Weber (2007), indicate that patients show substantial improvements in neurocognitive symptoms following Parathyroidectomy.

fMRI has showed changes in medial prefrontal and dorsolateral prefrontal cortex. The changes in Cerebral Blood Flow as shown on SPECT by Mjaland (2003) did not correlate with the level of serum calcium, intact parathyroid hormone or depression score. A study of CSF correlated with decrease in 5HIAA, a serotonin metabolite (Joborn 1991). Ex vivo studies of human cells have demonstrated calcium-mediated modulation of 5-HT₃ receptors, although specific binding site is yet to be identified (Thompson 2009). Therefore, the mechanism by which calcium levels disturbances contribute to mood changes remains largely unknown.

CONCLUSION

PHPT is an endocrine disorder presenting with non-specific neuropsychiatric symptoms, which shows excellent improvement with surgical treatment. This also results in improvement in the quality of life. However, further research is needed in calcium and other related ions to understand the complex mechanism behind neuro-psychopathological conditions.

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Conflict of interest : None to declare.

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References

1. Bilezikian JP, Brandi ML, Rubin M: Primary hyperparathyroidism: new concepts in clinical, densitometric and biochemical features. *Journal of Internal Medicine* 2005; 257:6-17.
2. Gatewood JW, Organ CH Jr, and Mead BT: Mental changes associated with hyperparathyroidism. *Am J Psychiatry* 1975; 132:129-132.
3. Geffken GR, Ward HE, et al: Psychiatric morbidity in endocrine disorders. *Psychiatr Clin North Am* 1998; 21:473-489.
4. Jimerson DC, Post RM, Carman JS, et al: CSF calcium: clinical correlates in affective illness and schizophrenia. *Biol Psychiatry* 1979; 14:37-51.
5. Joborn C, Hetta J, Niklasson F, et al: Cerebrospinal fluid calcium, parathyroid hormone, and monoamine and purine metabolites and the blood-brain barrier function in primary hyperparathyroidism. *Psychoneuroendocrinology* 1991; 16:311-22.
6. Joborn C, Hetta J, Lind L, Rastad J, Akerstrom G, Ljunghall S: Self-rated psychiatric symptoms in patients operated on because of primary hyperparathyroidism and in patients with long-standing mild hypercalcemia. *Surgery* 1989; 105:72-78.
7. Kelly KJ, Chen H, Sippel RS: Primary hyperparathyroidism. *Cancer Treat Res* 2010; 153: 87-103.
8. McAllion SJ, Paterson Colin R: Psychiatric morbidity in primary hyperparathyroidism. *Postgraduate Medical Journal* 1989; 628-631.
9. Mjaland O, Normann E, Halvorsen E, Rynning S, Egeland T: Regional cerebral blood flow in patients with primary hyperparathyroidism before and after successful parathyroidectomy. *Br J Surg* 2003; 90:732-7.
10. Rao DS, Phillips ER, Divine GW, Talpos GB: Randomized controlled clinical trial of surgery versus no surgery in patients with mild asymptomatic primary hyperparathyroidism. *J Clin Endocrinol Metab* 2004; 89:5415-5422.
11. Spivak B, Radvan M, Ohring R, et al: Primary hyperparathyroidism, psychiatric manifestations, diagnosis and management. *Psychother Psychosom* 1989; 51:38-44.
12. Taniagra ED: Hyperparathyroidism. *Am Fam Physician* 2004; 69:333-9.
13. Thompson AJ, Lummis SCR: Calcium modulation of 5-HT₃ receptor binding and function. *Neuropharmacology* 2009; 56:285-291.
14. Weber T, Keller M, Hense I, Pietsch A, Hinz U, Schilling T, Nawroth P, Klar E, Buchler MW: Effect of parathyroidectomy on quality of life and neuropsychological symptoms in primary hyperparathyroidism. *World J Surg* 2007; 31:1202-1209.
15. Wilhelm, Lee, Prinz, et al: Major depression due to primary hyperparathyroidism: a frequent and correctable disorder. *The American Surgeon* 2004; 70:175-179.