中文題目：高血鈣在一位肺部感染的病人

英文題目：Hypercalcemia in a patient with pulmonary infection

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Case presentation:

An 87-year-old woman without specific medical history presented to our hospital because of intermittent nausea and vomiting for one month.

She has a history of stroke with left hemiplegia and bed-ridden status with total dependent ADL for 20 years. About one month ago, she suffered abdominal pain, conscious disturbance. It had been accompanied by fever, dyspnea and productive cough. She was sent to our emergent department, where physical examination revealed disorientation to time, place, body temperature of 36 degrees Celsius, pulse rate of 75 per minutes, respiratory rate of 20 per minutes, tenderness over epigastric area and decreased muscle power. Biochemistry data revealed a severe hypercalcemia with corrected serum calcium level of 14.0 mg/dL (8.5-10.5 mg/dL), progression of impaired renal function of 5.28 mg/dL (0.4-1.1 mg/dL), normal liver enzyme with elevated total bilirubin level of 1.9mg/dL (0.2-1.3 mg/dL). The chest radiography revealed diffused infiltration with multiple small nodular lesions in bilateral lungs (Figure 1). Empiric antibiotic for community-acquired pneumonia was prescribed. Her hypercalcemia and acute kidney injury became improving after hydration. The sputum culture of tuberculosis showed Mycobacterium tuberculosis complex. The pulmonary tuberculosis was diagnosis. Anti-tuberculosis agents with isoniazid, rifampicin, ethambutol, and pyrazinamide were prescribed. Further laboratory investigation of the hypercalcemia revealed normal level of serum phosphorus level and the serum intact parathyroidhormone (iPTH) was 30 pg/mL (normal range: 15-88 pg/mL). Protein electrophoresis of serum revealed elevated M-protein of 12% (normal range: 0%) but immunofixation electrophoresis revealed normal ratio of monoclonal immunoglobulin IgG kappa/lambda in serum and urine. No punched-out lesions or lesion of brain was found in computed tomography of brain. Examination of bone marrow was suggested to exclude multiple myeloma after the pulmonary tuberculosis under control and the family refused the examination.

She received fluid replacement for the acute severe hypercalcemia. The hypercalcemia, hyperbilirubinemia, and conscious disturbance improved after hydration. No recurrent hypercalcemia was found during follow-up period.

Discussion:

Primary hyperparathyroidism and malignancy was the most common cause of hypercalcemia and account for 90 percent of cases. The other cause include vitamin-D induced

(4.3 percent) and miscellaneous or unknown cause include granulomatous disease.[1] Granulomatous disease including sarcoidosis, tuberculosis, fungal granuloma, berylliosis are associated with metabolism of calcium.[2] There are variant frequency between countries and primary hyperparathyroidism is the most frequent cause. This case remind us cases of pulmonary tuberculosis could presented hypercalcemia and resulted in impairment of renal function, acute pancreatitis, and consciousness disturbance.

The survey of hypercalcemia should be conducted after excluding drug-induced hypercalcemia. The level of parathyroid hormone is important for hypercalcemia to distinguish parathyroid dependent or parathyroid independent. Low or normal level of intact parathyroid hormone favors parathyroid independent etiology.[3]

The pathophysiology of hypercalcemia resulted from tuberculosis has variant proportion in articles. Abnormal regulation of 1,25 (OH2)D3 contributed to the hypercalcemia and spontaneous production of vitamin D metabolite by alveolar macrophage has a major contribution to hypercalcemia. But calcium metabolism abnormalities do not always correlate with the serum levels of 1,25(OH2)D3. Increased production of 1,25(OH2)D3 is associated with the increased proportion of T-lymphocyte, suggesting an intense alveolitis, African ethnic origin, and presentation of tuberculosis as isolated hilar adenophy.[4] Prolonged duration and level of hypercalcemia also resulted in renal failure. Severe hypercalcemia of 12–15 mg/dl can cause acute renal failure by direct renal vasoconstriction and volume contraction causing a decrease in glomerular filtration rate.[5] The treatment for severe hypercalcemia with consciousness disturbance or concentration > 14 mg/dL is indicated. Volume expansion with isotonic saline, calcitonin, bisphosphonate which was contraindicated with severe renal impairment. Avoidance of calcium-containing foods and supplements and vitamin D. Disease-specific approach is also indicated.

Figure

Figure 1. Radiograph of chest shows bilateral lung reticular/miliary infiltrates.

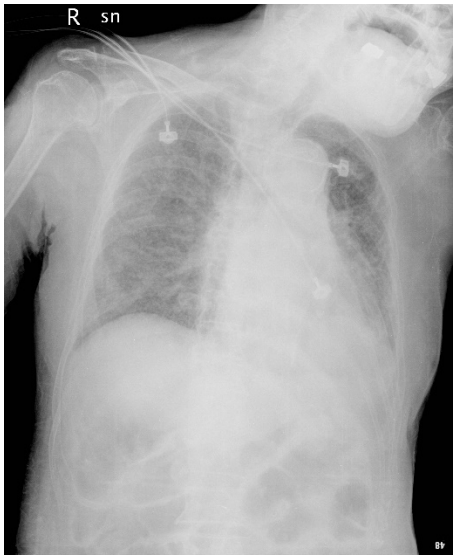

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Figure 2. Graph shows the change in serum calcium levels and serum creatinine in the months before and after starting antitubercular therapy.