

Hypocalcemia and hypercalcemia

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Case Presentation 1

The patient is a 45-year-old female with a history of hypothyroidism and chronic kidney disease. She has been on levothyroxine for the past 10 years and has been undergoing regular hemodialysis for the past 5 years.

Present Illness:

The patient presents to the emergency department with complaints of muscle cramps, tingling in her fingers and toes, and frequent episodes of numbness. She also reports experiencing fatigue and weakness over the past few days. On further questioning, she mentions that she has been feeling more irritable and anxious than usual.

Physical Examination:

On physical examination, the patient appears fatigued and slightly pale. Her blood pressure is 130/80 mmHg, heart rate is 90 beats per minute, and respiratory rate is 18 breaths per minute. Upon palpation, her muscles are found to be tender to touch. Chvostek's sign is positive, with facial twitching elicited upon tapping the facial nerve just anterior to the ear. Trousseau's sign is also positive, with carpal spasm induced by inflating a blood pressure cuff on her arm.

Laboratory Findings:

Laboratory tests reveal a serum calcium level of 7.5 mg/dL (normal range: 8.5-10.5 mg/dL), along with low levels of albumin at 3 g/dL (normal range: 3.5-5 g/dL). The patient's serum phosphorus level is elevated at 6 mg/dL (normal range: 2.5-4.5 mg/dL), while her parathyroid hormone (PTH) level is low at 15 pg/mL (normal range: 15-65 pg/mL).

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Introduction to Acute Hypocalcemia

Acute hypocalcemia refers to a sudden drop in the levels of calcium in the blood, potentially leading to serious complications. It can be caused by various factors, such as inadequate calcium intake, thyroid or parathyroid issues, and certain medication side effects.

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Definition and Causes of Acute Hypocalcemia

① Definition

Acute hypocalcemia is defined by a significant decrease in serum calcium levels, usually below 8.5 mg/dL.

② Causes

This condition can be caused by factors such as hypoparathyroidism, renal failure, and vitamin D deficiency, leading to impaired calcium absorption.

③ Underlying Conditions

Additionally, conditions like pancreatitis and cancer can contribute to acute hypocalcemia due to altered calcium metabolism.

Clinical Presentation and Symptoms

① Hypocalcemia Symptoms

Patients may experience symptoms like muscle cramps, tingling in the extremities, lethargy, and even seizures.

② Chvostek's Sign

A positive Chvostek's sign, characterized by facial muscle spasm upon tapping the facial nerve, is indicative of hypocalcemia.

③ Cardiac Manifestations

Cardiac manifestations can include arrhythmias and prolonged QT intervals on the ECG.

Diagnostic Tests for Acute Hypocalcemia

Blood Tests

Tests for serum calcium levels, along with magnesium and vitamin D levels, aid in diagnosing acute hypocalcemia.

Electrocardiogram (ECG)

An ECG can reveal changes associated with low calcium levels, such as QT interval prolongation.

Chvostek's & Trousseau's Signs

Observing these signs can also aid in confirming a diagnosis of acute hypocalcemia.

Differential Diagnosis of Acute Hypocalcemia

Hypoparathyroidism

One of the primary disorders to exclude due to its direct impact on calcium regulation.

Renal Failure

Considered because impaired kidney function can result in abnormalities in calcium metabolism.

Management of Acute Hypocalcemia

① Calcium Replacement

Oral or intravenous calcium supplementation is often necessary to elevate calcium levels swiftly.

② Vitamin D Therapy

Vitamin D analogs can aid in calcium reabsorption, particularly in cases of ongoing malabsorption.

③ Underlying Cause Management

Treating underlying issues like renal failure or hypoparathyroidism is crucial for long-term management of acute hypocalcemia.

Pharmacological Interventions for Acute Hypocalcemia

1

Calcium Gluconate

This medication is often administered intravenously to rapidly boost calcium levels in severe cases.

2

Alfacalcidol or Calcitriol

Vitamin D analogs such as these can enhance calcium absorption if there is an associated deficiency.

3

Bisphosphonates

For chronic hypocalcemia cases, bisphosphonates can prevent bone resorption and maintain calcium levels.

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Non-pharmacological Interventions for Acute Hypocalcemia

Dietary Modifications

Increasing intake of calcium-rich foods like dairy products and leafy greens can support long-term management.

Lifestyle Adjustments

Avoiding excess caffeine and sodium can assist in maintaining calcium balance within the body.

Exercise Programs

Weight-bearing exercises can help preserve bone mass and prevent calcium loss in the long run.

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Complications and Prognosis of Acute Hypocalcemia

① Cardiac Complications

Severe hypocalcemia can lead to life-threatening arrhythmias and heart failure if left unmanaged.

② Neurological Complications

Protracted low calcium levels can result in seizures, neuromuscular irritability, and long-term neurological effects.

Conclusion and Key Takeaways

1

Early Recognition is Vital

Timely identification and treatment of acute hypocalcemia are crucial to prevent severe complications.

2

Comprehensive Approach

Combining pharmacological and non-pharmacological measures is essential for effective management.

3

Long-term Surveillance

Regular monitoring post-treatment is important to prevent recurrence and manage potential complications effectively.

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Case Presentation 2

The patient is a 55-year-old male with a history of hypertension and chronic kidney disease. He has been on medication for his hypertension for the past 10 years and has been undergoing regular monitoring for his kidney function.

Present Illness:

The patient presents to the emergency department with complaints of fatigue, weakness, and frequent urination. He also reports experiencing nausea and constipation over the past several days. On further questioning, he admits to having bone pain and difficulty concentrating. His family members have also noticed a change in his behavior, describing him as being more irritable than usual.

Physical Examination:

Upon physical examination, the patient appears lethargic and dehydrated. His blood pressure is elevated at 160/90 mmHg, and he has a rapid pulse of 110 beats per minute. There are no signs of respiratory distress, but he appears pale. Upon palpation of his abdomen, there is tenderness in the epigastric region. His neurological examination reveals decreased deep tendon reflexes and confusion.

Laboratory Findings:

Laboratory investigations reveal an elevated serum calcium level of 12 mg/dL (normal range: 8.5-10.2 mg/dL). His creatinine level is also elevated at 2.5 mg/dL (normal range: 0.6-1.3 mg/dL), indicating impaired kidney function. The patient's parathyroid hormone (PTH) level is low at 15 pg/mL (normal range: 15-65 pg/mL), suggesting primary hyperparathyroidism as the cause of hypercalcemia.

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Introduction to Acute Hypercalcemia

Acute hypercalcemia is a rare but potentially life-threatening condition characterized by an elevated level of calcium in the blood. It can lead to a variety of symptoms and complications, making it crucial to diagnose and manage promptly.

Definition and Causes of Hypercalcemia

1

Definition

Hypercalcemia refers to high levels of calcium in the blood, typically beyond 10.4 mg/dL. It can result from various underlying conditions.

2

Common Causes

Causes include primary hyperparathyroidism, malignancy, hyperthyroidism, and excessive vitamin D intake. Each has distinct pathophysiological mechanisms.

3

Less Common Causes

Less common causes include granulomatous diseases, familial hypocalciuric hypercalcemia, and drug-induced hypercalcemia, each requiring specific diagnostic considerations.

Symptoms of

HY Clinical Presentation and Symptoms

1

Acute Presentation

Acute hypercalcemia may lead to nausea, vomiting, dehydration, constipation, polyuria, and muscle weakness due to derangement of neuromuscular function.

2

Cardiovascular Symptoms

Severe cases can result in arrhythmias, hypertension, and even cardiac arrest, emphasizing the need for vigilant monitoring and prompt intervention.

3

Neurological Manifestations

Central nervous system effects include confusion, lethargy, and coma, necessitating immediate medical attention and intervention.

Diagnostic Evaluation and Laboratory Findings

Laboratory Tests

Assessment includes serum calcium, albumin-corrected calcium, PTH, Vitamin D, and other relevant tests to identify the underlying cause.

Imaging Studies

Radiological investigations such as bone densitometry, ultrasound, MRI, or CT scans may be needed to visualize the underlying conditions contributing to hypercalcemia.

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Differential Diagnosis of Hypercalcemia

1 Primary Hyperparathyroidism

A common cause, characterized by overproduction of PTH and hypercalcemia, often with other mineral disturbances.

2 Malignancy-Associated Hypercalcemia

Arises from paraneoplastic syndromes, ectopic hormone production, bone metastases, or hypercalcemia of malignancy related to tumor-associated factors.

3 Vitamin D-Related Hypercalcemia

Including primary hypervitaminosis D or intoxication from excessive supplementation.

Management of Acute Hypercalcemia

1

Fluid Resuscitation

Addressing dehydration and enhancing urinary calcium excretion.

2

Calcium-Lowering Agents

Administration of calcitonin or bisphosphonates to decrease calcium levels.

3

Enhanced Renal Clearance

Utilizing loop diuretics to promote urinary excretion of calcium ions.

Pharmacological Interventions and Treatment Options

1

Bisphosphonates

2

Calcitonin

3

Steroids

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Non-Pharmacological Approaches and Lifestyle Modifications

1

Dietary Adjustments

Adherence to a low calcium diet and avoidance of Vitamin D supplements.

2

Hydration

Increased water intake to promote urinary calcium excretion.

3

Activity Modifications

Avoidance of excessive physical activities and immobilization if indicated.

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Complications and Long-Term Consequences

Renal Complications

Prolonged hypercalcemia can lead to renal impairment due to calcium deposition in renal parenchyma.

Skeletal Manifestations

Long-term hypercalcemia can result in osteoporosis and bone fractures.

Conclusion and Future Directions

The management of acute hypercalcemia requires a comprehensive approach including accurate diagnosis, timely intervention, and long-term monitoring. Advancements in therapeutic modalities and explorations into underlying mechanisms will likely shape future treatment strategies.