Hypercalcaemic crisis presentation and management

Hypercalcaemic crisis: presentation and management

Hypercalcaemia is documented in 0.5% of the general population and in up to 5% of hospitalised patients. The vast majority are asymptomatic with a mild to moderate elevation of serum calcium (<3 /uni00A0 mmol/L and 3–3.5 /uni00A0 mmol/L, respec tively) and respond to treatment of the underlying aetiology with associated dietary modification. A small proportion of patients will present symptomatically with a total calcium of >3.5 /uni00A0 mmol/L. This is referred to as a hypercalcaemic crisis and requires ag gressive medical management. Although symptoms can be varied, the typical presentation is of acute confusion, abdominal pain, vomiting, dehydration and anuria. Prolongation of the PR interval with a shortened QT interval can be identified on an electrocardiog ram (ECG) prior to potentially lethal cardiac arrhythmias. Where the cal cium is >4.5 /uni00A0 mmol/L, coma and cardiac arrest can occur. Treatment revolves around increasing renal excretion of calcium, reducing skeletal release of calcium and treatment of the underlying cause. Aggressive rehy dration plays a pivotal role. Typically , 200–500 /uni00A0 mL/h of normal saline is given to maintain a urine output >100 /uni00A0 mL/h, with the caveat that this may be modified to account for associated patient comorbidi ties. Once intravascular volume has been adequately restored, loop diuretics, such as furosemide, can be used to enhance the renal excretion of calcium. The majority of patients will have normalisa tion of their calcium with these simple measures. In patients with advanced malignancy and a serum calcium level >3 /uni00A0 mmol/L, agents that blunt the release of calcium from skeletal stores may be required. First-line treatment includes administration of bisphosphonates. These are pyrophosphate analogues that inhibit osteoclast activity in areas of high bone turnover. In the acute setting, these are given intravenously owing to poor absorption in the gastrointestinal tract. Calci tonin can be used to both decrease osteoclastic activity and increase renal excretion of calcium. It has a short duration of action and is usually used as a bridge to reduce calcium until the sustained action of the bisphosphonates is seen. Finally , glucocorticoids (prednisolone) can be used to enhance the action of calcitonin. T hey increase calciuresis and decrease intestinal absorption of calcium. As a result, they may also play a role in diseases associated with vitamin D excess. Hypercalcaemic crisis: presentation and management

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Revision #1
Created 2025-12-31 15:21:03 UTC by Omar Ayman
Updated 2025-12-31 15:21:03 UTC by Omar Ayman