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Opinion Article

Hyperphosphatemia: Causes, consequences and management

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ABOUT THE STUDY

Hyperphosphatemia refers to an abnormally high level of phosphate in the blood. Phosphate is an essential mineral that plays a vital role in various physiological processes, including bone formation, energy metabolism, and cellular signalling. However, when phosphate levels become elevated beyond the normal range, it can lead to a range of complications and adverse effects on multiple organ systems.

Causes

Chronic Kidney Disease (CKD): One of the primary causes of hyperphosphatemia is impaired renal function, particularly in patients with CKD. The kidneys play a crucial role in maintaining phosphate homeostasis by excreting excess phosphate through urine. In CKD, the kidneys lose their ability to efficiently eliminate phosphate, resulting in its accumulation in the bloodstream.

Medications: Certain medications, such as phosphate-containing laxatives, phosphate-based enemas, and certain antacids, can contribute to elevated phosphate levels. Additionally, the use of phosphate-containing intravenous fluids and some chemotherapeutic agents can also cause hyperphosphatemia.

Endocrine disorders: Hormonal imbalances can disrupt phosphate regulation. Hypoparathyroidism, a condition characterized by decreased parathyroid hormone levels, leads to impaired phosphate excretion and subsequent hyperphosphatemia. Similarly, acromegaly, a disorder associated with excessive growth hormone production, can also result in elevated phosphate levels.

Tumor lysis syndrome: Hyperphosphatemia can occur as a consequence of rapid cell destruction, commonly observed in certain cancers, such as leukemia and lymphoma. The release of intracellular phosphate from lysed cells overwhelms the body's capacity to eliminate it, causing an increase in serum phosphate levels.

Consequences

Calcification of soft tissues: Hyperphosphatemia disrupts the balance between phosphate and calcium, leading to abnormal deposition of calcium phosphate crystals in soft tissues throughout the body. This calcification process primarily affects blood vessels, heart valves, and organs such as the lungs and kidneys, impairing their function and potentially leading to organ failure.

Secondary hyperparathyroidism: Persistent elevation of phosphate levels triggers compensatory mechanisms, such as increased Parathyroid Hormone (PTH) secretion. PTH acts to mobilize calcium from bones and enhance renal phosphate excretion. However, prolonged stimulation of the parathyroid glands can result in secondary hyperparathyroidism, leading to bone demineralization and an increased risk of fractures.

Cardiovascular complications: Hyperphosphatemia has been linked to increased cardiovascular morbidity and mortality. Elevated phosphate levels promote vascular smooth muscle cell proliferation and calcification, leading to the development of atherosclerosis and cardiovascular disease. Additionally, hyperphosphatemia impairs endothelial function, disrupts nitric oxide availability, and promotes inflammation, all of which contribute to the development of cardiovascular complications.

Management

Dietary modifications: Dietary phosphate restriction is a cornerstone of managing hyperphosphatemia. Limiting the intake of high-phosphate foods, such as dairy products, processed meats, and carbonated beverages, can help reduce phosphate absorption. Additionally, consumption of phosphate binders, which bind dietary phosphate in the gastrointestinal tract, can be prescribed to limit its absorption.

Phosphate binders: Phosphate binders are medications that reduce phosphate absorption from the diet. They work by binding to dietary phosphate in the gastrointestinal tract, forming an

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insoluble complex that is excreted in the faeces. Commonly used phosphate binders include calcium-based binders (calcium carbonate, calcium acetate), sevelamer, and lanthanum carbonate. The choice of phosphate binder depends on factors such as the patient's underlying condition and serum calcium levels.

Dialysis: In individuals with advanced CKD, dialysis is often necessary to manage hyperphosphatemia. Dialysis helps remove excess phosphate from the bloodstream, restoring phosphate levels to a more acceptable range. Hemodialysis and peritoneal dialysis are the two main modalities used for phosphate clearance in individuals with end-stage renal disease.

Causes: Managing hyperphosphatemia involves addressing the underlying causes whenever possible. This may involve optimizing renal function in CKD patients, adjusting medication regimens, and treating endocrine disorders such as hypoparathyroidism or acromegaly.

Hyperphosphatemia, characterized by elevated phosphate levels in the blood, can have significant consequences on various organ systems. It is crucial to identify and manage the underlying causes of hyperphosphatemia to prevent complications such as soft tissue calcification, secondary hyperparathyroidism, and cardiovascular disease.

Through dietary modifications, phosphate binders, and dialysis, healthcare professionals can effectively manage hyperphosphatemia and improve patient outcomes. Early detection and prompt intervention are key to mitigating the adverse effects of hyperphosphatemia and ensuring optimal phosphate balance in the body.