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Management of hypokalaemia and hyperkalaemia

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Over 90% of the potassium in the body is located within cells. External balance for potassium is maintained by matching output to input. Internal balance is maintained by translocation of potassium between intracellular and extracellular fluid. Any change in plasma potassium concentration must arise from a change in intake, distribution, or excretion.

Hypokalaemia

Causes

Decreased intake of potassium alone is unlikely to cause hypokalaemia but, in chronically ill animals, prolonged anorexia, loss of muscle mass, and ongoing urinary potassium losses may combine to cause hypokalaemia. Alkalemia contributes to hypokalaemia as potassium ions enter cells in exchange for hydrogen ions. Insulin promotes uptake of glucose and potassium by hepatic and skeletal muscle cells. A syndrome characterised by recurrent episodes of limb muscle weakness and neck ventroflexion, increased creatine kinase concentrations, and hypokalaemia has been reported in related young Burmese cats.

Gastrointestinal loss of potassium (especially vomiting of stomach contents) is an important cause of hypokalaemia in small animals. Chloride depletion and sodium avidity due to volume depletion contribute to perpetuation of potassium depletion and metabolic alkalosis by enhancing urinary losses of potassium and hydrogen ions. Urinary loss of potassium is another important cause of hypokalaemia and hypokalaemia is common in cats with chronic renal failure. Hypokalaemia also may occur in distal renal tubular acidosis in cats. Finally, hypokalaemic nephropathy characterised by tubulointerstitial nephritis may develop in cats fed diets marginally replete in potassium and containing urinary acidifiers. Hypokalaemia commonly occurs during the post-obstructive diuresis that follows relief of urethral obstruction in cats. Mineralocorticoid excess is a rare cause of urinary potassium loss and hypokalaemia in dogs and cats. Administration of loop or thiazide diuretics may cause hypokalaemia by increased flow rate in the distal tubules and increased secretion of aldosterone secondary to volume depletion. Peritoneal dialysis can be complicated by hypokalaemia if potassium-free dialysate is used over an extended period of time.

Clinical signs

Muscle weakness may be observed when serum potassium concentration falls below 2.5–3.0 mEq/l. Rear limb weakness and, in cats, weakness of neck muscles with ventroflexion of the head are commonly observed. Cardiac arrhythmias may develop because hypokalaemia increases automaticity and delays ventricular repolarisation. In dogs and cats, the electrocardiographic changes associated with hypokalaemia are inconsistent but ventricular arrhythmias may be observed. Polyuria, polydipsia, and

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defective urinary concentrating ability may be observed in hypokalaemia.

Diagnosis

The clinical history often will provide information about the likely source of potassium loss (eg, vomiting, diuretic administration). Determination of the fractional excretion of potassium (FE_K as a percentage=$U_K P_{Cr}/P_K U_{Cr} \times 100$) may help differentiate renal and non-renal sources of potassium loss. The FE_K should be <4% for non-renal sources of loss and values >4% indicate inappropriate renal loss in the face of hypokalaemia. The occurrence of hypokalaemia with metabolic alkalosis suggests vomiting of stomach contents or diuretic administration as likely causes of potassium loss.

Treatment

Potassium chloride is the additive of choice for parenteral therapy because chloride repletion also is very important if vomiting or diuretic administration is the underlying cause of hypokalaemia. When administered intravenously, potassium should not be infused at a rate greater than 0.5 mEq/kg/hr. Infusion of potassium-containing fluids initially may be associated with a decrease in plasma potassium concentration as a result of dilution, increased distal tubular flow, and cellular uptake of potassium, especially if the infused fluid also contains glucose. This effect may be minimised by using a fluid that does not contain glucose and by administering it at an appropriate rate. Potassium gluconate is recommended for oral supplementation. In cats with hypokalaemic nephropathy, the initial oral dosage of potassium gluconate is 5–8 mEq per day divided q 12 h or q 8 h whereas the maintenance dosage can usually be reduced to 2–4 mEq per day.

Careful potassium supplementation is very important when using insulin to treat diabetic ketoacidosis. Chronic potassium depletion usually is present in affected patients as a result of loss of muscle mass, anorexia, vomiting, and polyuria. Serum potassium concentrations, however, often are normal or even increased due to the effects of insulin deficiency and hyperosmolality on serum potassium concentration. As blood glucose concentration falls with insulin treatment, marked hypokalaemia may develop if supplementation is not diligent.

Hyperkalaemia

Causes

Hyperkalaemia occurs uncommonly if renal function is normal. Even in chronic renal failure, potassium excretion is maintained by enhanced tubular secretion in remnant nephrons so that hyperkalaemia only develops if oliguria supervenes. Thus, chronic hyperkalaemia is almost always associated with impaired renal excretion. Increased intake is likely to be contributory only during excessive infusion of potassium-rich fluids or in the face of impaired renal excretion.

Serum potassium concentrations exceed plasma concentrations because potassium is released from platelets during clotting. The difference between serum and plasma potassium concentrations is most pronounced in animals with thrombocytosis. Haemolysis can result in hyperkalaemia in species with high red cell potassium content. Normal canine and feline red cells contain potassium in concentrations similar to those of plasma and haemolysis usually is not associated with hyperkalaemia. In some Akitas, however, red cell potassium concentration may be as high as 70 mEq/L and haemolysis may result in progressive hyperkalaemia during storage of blood. Haemolysis in Akitas and thrombocytosis cause what has been called pseudohyperkalaemia because these effects occur in vitro.

Translocation of potassium from intracellular to extracellular fluid can cause hyperkalaemia. Metabolic acidosis due to mineral acids (eg, NH_4Cl, HCl) but not organic acids (eg, lactic acid, ketoacids) causes potassium to shift out of cells in exchange for hydrogen ions that enter cells to be buffered. The effect of inorganic metabolic acidosis on serum potassium concentration is very variable. Insulin deficiency and hyperosmolality contribute to hyperkalaemia in diabetic patients. Acute tumour lysis syndrome complicated by renal failure and hyperkalaemia has been reported in dogs with lymphoma after radiation or chemotherapy.

Decreased urinary excretion is the most important cause of hyperkalaemia in small animal practice. The most common associated disorders are urethral obstruction, ruptured bladder, anuric or oliguric renal failure, and hypoadrenocorticism. The time required for development of hyperkalaemia in cats after urethral obstruction is variable but it may occur within 48 h. After relief of obstruction,

hyperkalaemia resolves within 24 h whereas azotemia and hyperphosphatemia require 48–72 h to resolve. After experimental bladder rupture in dogs, azotemia, hyperphosphatemia, and mild hyponatraemia developed within 24 h whereas hyperkalaemia did not develop until after 48 h. Hyperkalaemia, hyponatraemia, and Na/K ratios <27:1 are usually (but not always) found in dogs and cats with hypoadrenocorticism and similar findings can occur in dogs with gastrointestinal disease due to trichuriasis, salmonellosis, or perforated duodenal ulcer. Hyperkalaemia only occurs in renal failure when anuria or oliguria develops. This occurs more commonly in acute renal failure (eg, ethylene glycol ingestion) but may occur terminally in chronic renal failure. Potassium-sparing diuretics (eg, spironolactone) reduce urinary excretion of potassium and can cause hyperkalaemia.

Clinical signs

Muscle weakness develops with hyperkalaemia, usually when serum potassium concentration exceeds 8 mEq/l. The electrocardiographic findings caused by hyperkalaemia include peaked narrow T waves and shortened QT interval reflecting abnormally rapid repolarisation. These changes are followed by widened QRS complexes and decreased amplitude, abnormally wide or absent *P* waves associated with delayed depolarisation. A sinoventricular rhythm develops followed by ventricular fibrillation and cardiac standstill. These electrocardiographic findings represent the life-threatening functional consequences of hyperkalaemia.

Diagnosis

Increased intake, translocation from intracellular to extracellular fluid, and impaired excretion must all be considered when evaluating a patient with hyperkalaemia. Except in a hospital setting where infusion of potassium-containing fluids may be the source of hyperkalaemia, decreased urinary excretion of potassium or translocation are the usual causes.

Treatment

Hyperkalaemia can be treated by antagonising its effects on cell membranes with calcium gluconate, driving extracellular potassium into cells with sodium bicarbonate or glucose, or by removing potassium from the body with a cation exchange resin or dialysis. First, any source of intake must be discontinued (eg, potassium-containing fluids, potassium penicillin). Hyperkalaemia decreases the resting potential of cells. By administering calcium gluconate, the extracellular fluid concentration of calcium is increased and the threshold potential is decreased thus normalising the difference between the resting and threshold potential and restoring normal membrane excitability. Administered calcium begins to work within minutes but its effect lasts less than an hour. The dosage of calcium gluconate is 2–10 ml of a 10% solution to be administered slowly with electrocardiographic monitoring. Glucose works by increasing endogenous insulin release and moving potassium into cells. Its effects begin within an hour and last a few hours. Glucose-containing fluids (5 or 10% dextrose) or 50% dextrose (1–2 ml/kg) can be used for this purpose. Unless the patient is diabetic, administration of insulin with glucose usually is unecessary and may cause hypoglycaemia. Sodium bicarbonate also works by moving potassium into cells as hydrogen ions come out to titrate the administered bicarbonate. Bicarbonate begins to work within an hour and its effects last a few hours. The usual dosage is 1–2 mEq/kg intravenously and it can be repeated if necessary. Loop or thiazide diuretics increase distal tubular flow rate and potassium secretion and may have adjunctive value in the treatment of hyperkalaemia. The cation exchange resin polystyrene sulfonate can be used to bind potassium and release sodium in the gastrointestinal tract. Each gram will bind one mEq of potassium and release 1–2 mEq of sodium. If these measures fail, the clinician must consider peritoneal dialysis.

Suggested reading

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