FIND THE SOURCE Causes of hyperchloremia vary and include acid-base related, drug related, and renal failure.

INSIGHTS IN ELECTROLYTE DISORDERS

Evaluation and Management of the Hyperchloremic Patient

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Chloride (Cl^-) is the most abundant extracellular fluid anion, and serum chloride concentrations (i.e., [Cl]) are closely linked to sodium (Na^+) status. Processes that increase sodium concentration (i.e., [Na]) are expected to drive [Cl] in the same direction; a disconnect between the two therefore has diagnostic and therapeutic implications.[1-3] Primary derangements in [Cl] are recognized by calculating the "corrected" [Cl]:

Corrected [Cl] = Normal [Na]/Patient [Na] × Patient [Cl]

Normal [Na] = Midpoint of the reference interval

If this is within the reference range, the patient has a free water loss, and we do not need to address the [Cl] specifically.

In healthy animals, serum [Cl] is primarily regulated by the kidney. Chloride is freely filtered across the glomerulus, and about 60% is absorbed along the proximal convoluted tubule.[4] Additional chloride is reclaimed in the loop of Henle and distal convoluted tubule. However, chloride can be either secreted or reabsorbed in the collecting duct; this outcome is influenced by serum aldosterone concentrations and the rates of sodium uptake and/or bicarbonate (HCO_3^-) secretion.

Corrected hyperchloremia is most often driven by changes in acid–base status or the excessive administration of chloride.[1,2,5] Investigation of hyperchloremia often requires concurrent blood gas analysis, or—at the very least—determination of the patient's total carbon dioxide (TCO_2) status. This measurement approximates bicarbonate concentration (i.e., [HCO_3]) within 1 to 2 mEq/L and can be used to identify an acid–base disturbance. However, a subnormal TCO_2 will be noted in patients with either a metabolic acidosis or a compensated respiratory alkalosis.

CAUSES OF HYPERCHLOREMIA

Spurious

Pseudohyperchloremia is expected in patients receiving the salts of other halides (i.e., bromide, iodide, fluoride), as these interfere with chloride determination. The reported hyperchloremia is usually modest, although high values are seen with potassium bromide toxicosis.[6] Lipemia may falsely increase [Cl] if non–ion-selective methods are used.[1]

Addition of Chloride

All replacement fluids contain robust amounts of chloride, but 0.9% sodium chloride (NaCl) has the

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highest chloride content at 154 mmol/L. Acute administration of large volumes of 0.9% NaCl may quickly drive up serum [Cl]. The rapid infusion of 2 L of 0.9% NaCl to healthy human volunteers has been shown to disrupt sodium and chloride balances for up to 2 days.[7] Balanced polyionic replacement fluids (e.g., lactated Ringer's solution, Normosol-R, Plasma-Lyte 148) have significantly lower [Cl], but prolonged administration may result in hyperchloremia. Additives such as potassium chloride (KCl) may also contribute to "chloride creep," along with any 0.9% NaCl used to dilute injectable drugs. Care must be taken to ensure compatibility when selecting alternative diluents; suitable options should be listed in the package insert.

Saltwater ingestion may also cause a true hyperchloremia; however, the associated hypernatremia is the more pressing clinical concern.

Acid–Base Related

A detailed discussion of acid–base regulation is beyond the scope of this article, but essentially, the loss or gain of an acid or an alkali will impact [HCO_3]. Changes in [HCO_3] will in turn impact [Cl], as these are the primary anions in the extracellular fluid compartment and play a key role in the maintenance of electroneutrality. Under normal circumstances, the difference between measured anions (i.e., Cl^- and HCO_3^-) and cations (i.e., Na^+ and K^+ [potassium]) remains fairly constant. This difference is referred to as the "anion gap":

Anion gap = Patient [$Na^+ + K^+$] – Patient [$Cl^- + HCO_3^-$]

Dog: 12 to 24 mEq/L[8]

Cat: 13 to 27 mEq/L[8]

Bicarbonate loss: Any condition associated with the loss of HCO_3^- is expected to result in an increase in [Cl] in order to maintain electroneutrality; this is termed a "normal anion gap metabolic acidosis."[5,9] HCO_3^- may be lost through the gastrointestinal tract (i.e., in patients with diarrhea) or via the kidneys (i.e., renal tubular acidosis [RTA]). The latter condition may be an isolated defect or associated with other renal tubular reabsorptive defects, such as those seen with Fanconi syndrome. Depending on the type of RTA, urine pH may be alkaline despite a significant metabolic acidosis.

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In contrast, a metabolic acidosis driven by the addition of a noncarbonic acid such as lactate, ketones, and uremic acids is characterized by an increased anion gap and is not associated with hyperchloremia.

Respiratory alkalosis: Patients that hyperventilate will blow off carbon dioxide (CO_2); this results in a decrease in hydrogen (H^+) ions and a respiratory alkalosis. In essence, the carbonic acid dissociation equation shifts to the left:

H_2O and CO_2 ← H_2CO_3 ← H^+ and HCO_3^-

H_2CO_3 = Carbonic acid

With acute respiratory alkalosis, CO_2 and chloride move from the intracellular space in exchange for HCO_3^-.[10] If hyperventilation persists, compensatory renal adaptations drive the reclamation of chloride and excretion of HCO_3^- into the filtrate, resulting in progressive hyperchloremia and progressive decreases in [HCO_3]. Chronic hypocapnia (i.e., low venous carbon dioxide concentration [CO_2]) is uncommon in veterinary patients, although it may occur in those that are mechanically ventilated.

Renal Failure

Hyperchloremia has been reported in humans with end-stage renal disease. This probably reflects compromised hydrogen excretion; this leads to the reabsorption of sodium accompanied by chloride, rather than in exchange for hydrogen.[11]

Hypoadrenocorticism

Corrected hyperchloremia has been reported in dogs in a hypoadrenal ("addisonian") crisis. The measured [Cl] in these patients is not increased; however, the corrected [Cl] may exceed the reference range.

The underlying driver is lack of aldosterone; this hormone facilitates the reclamation of sodium in exchange for protons in the distal nephron. Aldosterone deficiency therefore results in H^+ accumulation, depletion of HCO_3^-, and a compensatory increase in chloride.[12]

Drug Related

Spironolactone is an aldosterone receptor blocker and is used in the management of congestive heart failure and cirrhotic ascites. It limits sodium reabsorption in the distal convoluted tubules and therefore has a diuretic effect. However, decreased sodium uptake limits proton excretion; this may result in a hyperchloremic metabolic acidosis.[13]

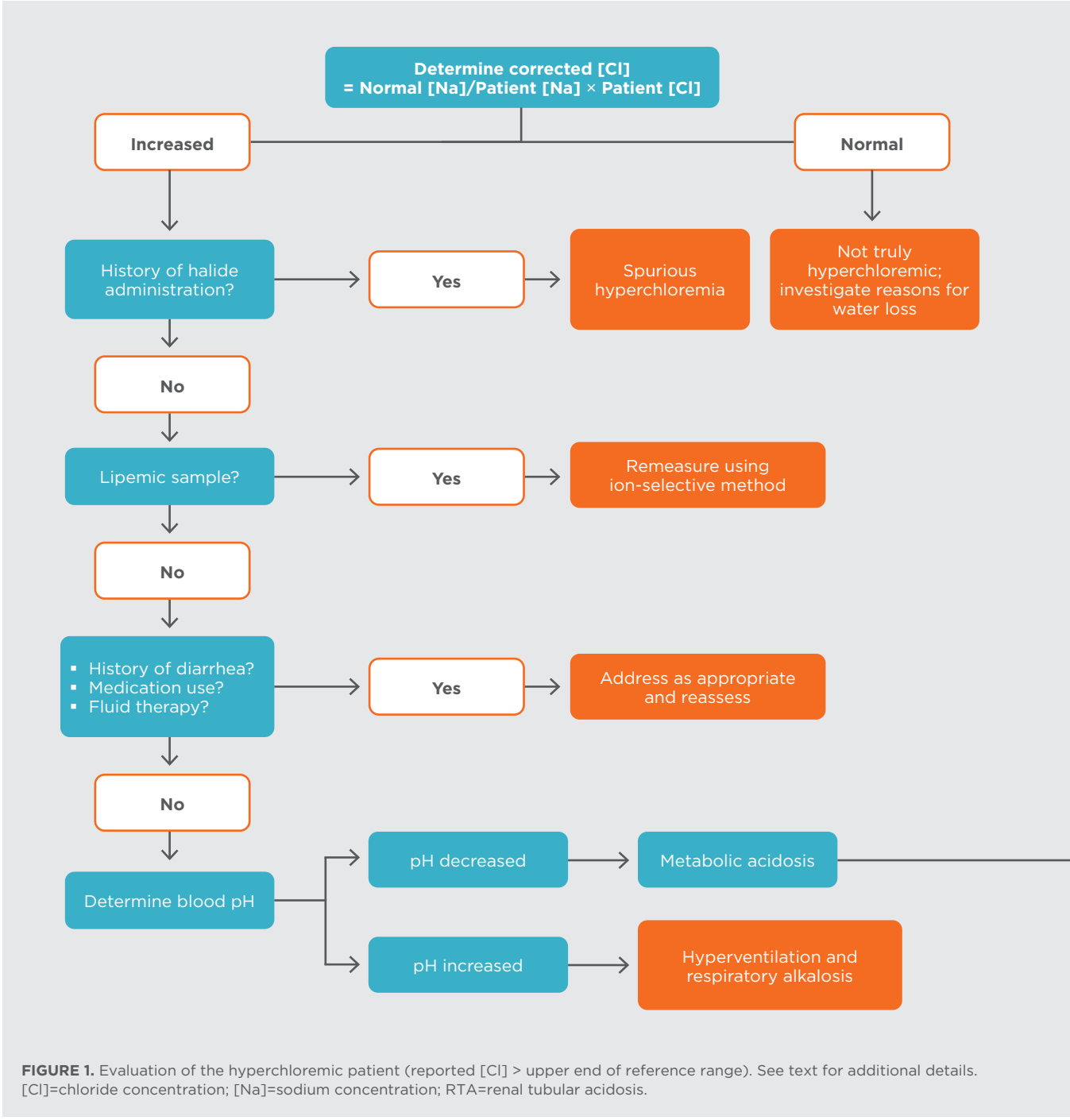
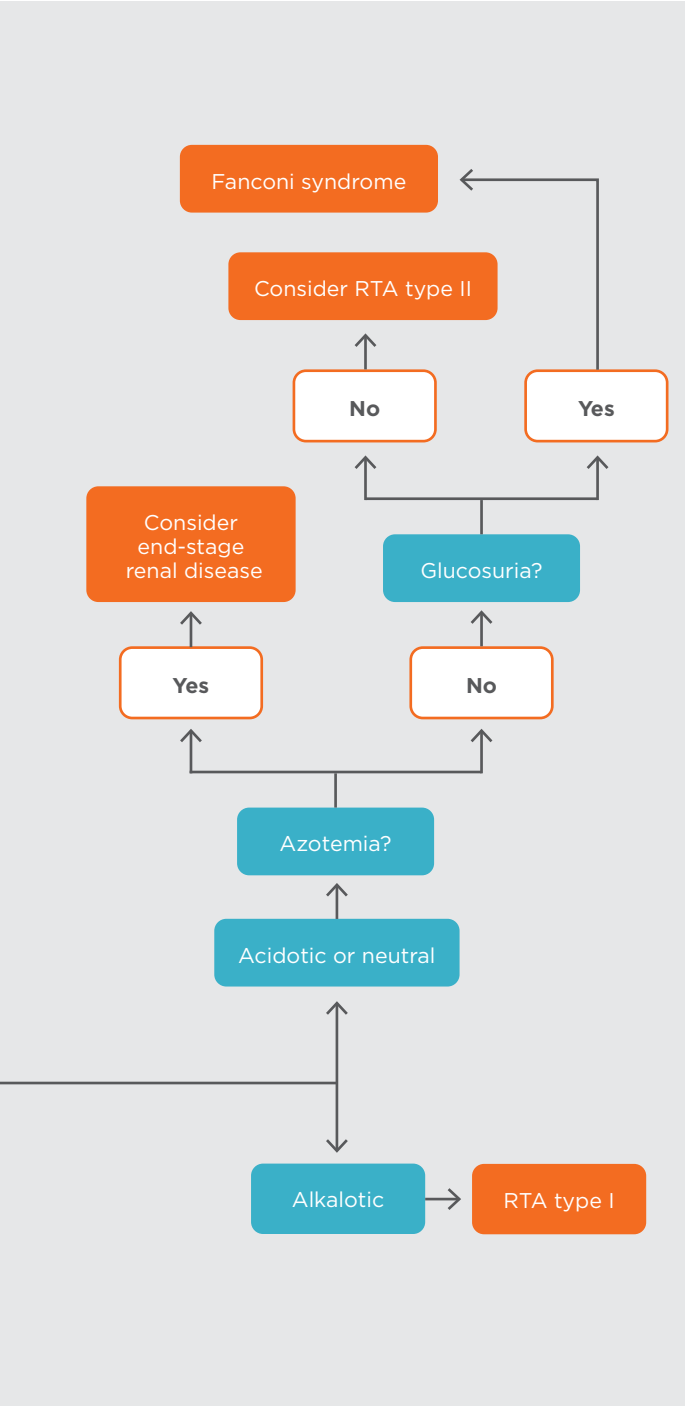


FIGURE 1. Evaluation of the hyperchloremic patient (reported [Cl] > upper end of reference range). See text for additional details. [Cl]=chloride concentration; [Na]=sodium concentration; RTA=renal tubular acidosis.

Zonisamide is an anticonvulsant drug used in the treatment of idiopathic epilepsy. It has been associated with RTA, attributable to inhibition of renal carbonic anhydrase. Discontinuation of the drug reverses this phenomenon.[14]

Ammonium chloride has been used to reduce struvite stone formation by acidifying urine. Its benefits in this regard are questionable, and it may result in hyperchloremia. The condition should resolve quickly when the drug is discontinued.[3]



CONSEQUENCES OF HYPERCHLOREMIA

Hyperchloremia per se is not associated with specific clinical signs, although an accompanying acid–base disorder may result in hyperventilation, changes in mental status, and muscle twitching.[1,2] The impact of hyperchloremia on renal function has recently received attention, and there is evidence that excessive chloride content in renal filtrate may negatively impact renal blood flow (RBF) and glomerular filtration rate (GFR). This is caused by a complex process called tubuloglomerular feedback, in which the nature (i.e., rate of flow and content) of filtrate in distal portions of the nephron impacts vascular events within the glomerulus. This effect is due to the normal homeostatic mechanisms that regulate RBF and GFR and is orchestrated by the macula densa. The exact processes by which the specialized cells of the macula densa determine flow are unclear, but chloride seems to play a role. High [Cl] within the filtrate triggers the release of locally active mediators and results in constriction of the afferent arteriole and a decrease in GFR.[15,16] Infusion of a high chloride fluid such as 0.9% NaCl to healthy human volunteers will decrease RBF by 10% within 20 to 30 minutes. Infusion of a lower-chloride fluid such as Plasma-Lyte 148 (sodium, 140 mmol/L; chloride, 98 mmol/L) does not have the same effect.[17]

Negative impacts on cardiovascular function and hemostasis have also been associated with hyperchloremia, along with increases in inflammatory biomarkers.[18] Unfortunately, it can be difficult to determine the specific effects of [Cl] due to the potentially confounding influences of concurrent acidosis and/or hypernatremia.

Various studies have documented an association between hyperchloremia and increased morbidity in human patients with a range of conditions, including diabetic ketoacidosis, sepsis, and a postoperative state.[19-21] Although there is very little information on this topic in companion animals, one recent study demonstrated an association between higher serum [Cl] and an increased likelihood of hospital-acquired acute kidney injury.[22] It is noteworthy that some dogs developed an acute kidney injury without overt hyperchloremia. Hyperchloremia was associated with

an increase in all-cause mortality in dogs in a large retrospective study; those with a normal or slightly subnormal [Cl] had case fatality rates below that of the whole population.[23]

Interestingly, hyperchloremia does not occur in every patient receiving a chloride-rich crystalloid, and we cannot predict the likelihood of a clinically impactful increase in [Cl] in a hospitalized animal. For this reason, the authors recommend at least daily evaluation of electrolytes (sodium, potassium, chloride) in all patients receiving intravenous fluid therapy.

INITIAL PATIENT ASSESSMENT

If hyperchloremia is noted on a serum biochemical profile, the corrected [Cl] should be calculated (**FIGURE 1**). If this is normal, the animal has a sodium/water imbalance that merits attention. If the corrected [Cl] is above the reference range, reasons for true hyperchloremia should be considered. Current medical treatments should be reviewed, and agents known to cause hyperchloremia (e.g., spironolactone, zonisamide, ammonium chloride) should be discontinued. Hyperchloremia occurring during hospitalization is usually caused by fluid therapy, which should be carefully adjusted.

If hyperchloremia is still unexplained, the patient's acid–base status should be evaluated. A blood gas analysis is ideal, but the TCO_2 may be used if the former is not available. If the patient has a normal anion gap metabolic acidosis, reasons for HCO_3^- loss should be investigated. Diarrhea is a possible cause, which should be apparent by the patient's history. If this possibility is excluded, urine characteristics should be examined. An alkaline pH in a patient with a metabolic acidosis indicates loss of HCO_3^- through the kidneys. It is important to note that the urine may be neutral or even acidic in dogs with type II RTA.[24]

Hyperchloremia occurring during hospitalization is usually caused by fluid therapy, which should be carefully adjusted.

TREATMENT OF HYPERCHLOREMIA

Iatrogenic hyperchloremia is managed by alterations to the patient's treatment plan. Drug-related hyperchloremia should resolve within 7 days. Increases in [Cl] due to fluid therapy may take a few days to reverse. Careful attention should be paid to the patient's renal status during this time, so that acute kidney injury can be promptly recognized.

Although the routine use of HCO_3^- in acidemic patients has fallen out of favor, this is an appropriate option for patients with chronic hyperchloremic metabolic acidosis caused by renal HCO_3^- loss. Powdered sodium bicarbonate ($NaHCO_3$; i.e., baking soda) may be used at a starting dose of 8 to 12 mg/kg PO q8h to q12h. Large doses of baking soda should be given with caution, as CO_2 generation may cause gastric distention and abdominal discomfort. Doses should be titrated based on measurements of venous pH or TCO_2 concentration. Alternatively, potassium citrate may be considered (50 to 75 mg/kg PO q12h); this provides buffering and can address concurrent hypokalemia in patients with other renal tubular reabsorptive disorders. The Gonto protocol should be considered in patients with Fanconi syndrome.[25]

In patients with HCO_3^- loss due to diarrhea, administration of a balanced buffered replacement fluid should be sufficient to mitigate acidemia and hyperchloremia. HCO_3^- may be given intravenously in a significantly compromised patient with a severe normal anion gap metabolic acidosis; the authors reserve this option for severely affected animals, typically with a pH <7.1. The HCO_3^- deficit should first be determined:

HCO_3^- deficit = Weight (kg) × {Desired [HCO_3] – Patient [HCO_3]} × 0.3

Note: Desired [HCO_3] is middle of reference range; alternatively, the reported base deficit may be used in place of {Desired [HCO_3] – Patient [HCO_3]}.

As a general guideline, one-third of the deficit should be delivered over 30 minutes as $NaHCO_3$ solution. The patient's acid–base status should then be reassessed. Excessive or rapid (i.e., bolus) administration of HCO_3^- can result in paradoxical central nervous system acidosis. $NaHCO_3$ should also be used cautiously in patients with impaired ventilation, as rapid pH correction can result in increases in [CO_2].[26]

Hyperchloremia secondary to hyperventilation should be addressed with sedation or analgesia (in anxious or painful patients) or by adjustment of device settings for those on a mechanical ventilator. Abrupt cessation of hyperventilation can leave the patient with a metabolic acidosis caused by renal HCO_3^- loss during protracted respiratory alkalosis.

See **FIGURE 2** for an algorithm of the management of the hyperchloremic patient.

CASE SCENARIO

History

A 6-year-old neutered male Labrador retriever was presented for evaluation of gait changes and apparent pain. The owner reported that the dog whimpered when getting into his crate and struggled to get up on the sofa. Appetite was unchanged, and the owner reported that the dog had "always been a good drinker." Physical examination revealed discomfort on palpation of the thoracic wall and a short-strided gait in the rear.

Results of a complete blood count were unremarkable; the biochemical profile revealed a severe hyperchloremia of 128 mmol/L (reference range, 107 to 116 mmol/L); sodium was at the upper end of the reference range at 146 mmol/L, and TCO_2 was low at 14 mmol/L (reference range, 21 to 28 mmol/L). Blood urea nitrogen was low at 2 mg/dL (reference range, 5 to 29 mg/dL); creatinine was normal at 1.16 mg/dL

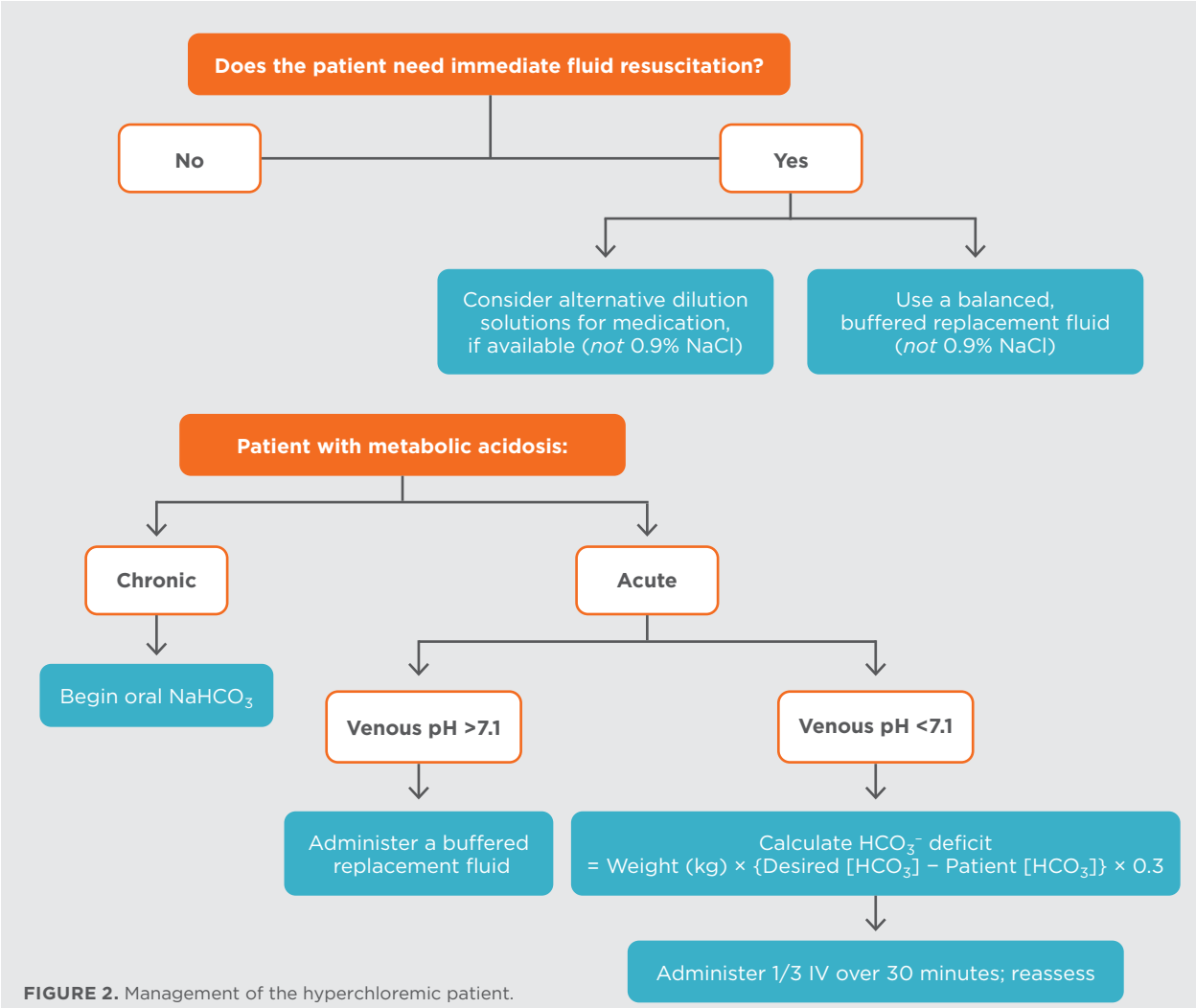


FIGURE 2. Management of the hyperchloremic patient.
See text for additional details.
[Cl]=chloride concentration; HCO_3^-=bicarbonate; NaCl=sodium chloride; $NaHCO_3$=sodium bicarbonate.

(reference range, 0.5 to 1.5 mg/dL). Urine pH was 6.5, specific gravity was 1.007, protein was 30 mg/dL, and glucose was 500 mg/dL. Urine protein:creatinine ratio was 4.1 (reference range, <0.4).

Thoracic radiographs revealed multiple rib fractures in different stages of healing, along with fractures of 2 of the thoracic vertebrae and a possible fracture of the caudal neck of the right humerus (**FIGURES 3 AND 4**).

Due to evidence of multiple renal tubular defects, a urine sample was submitted for a metabolic screen (PennGen Laboratories; **bit.ly/penngen**). This test identified aciduria, glucosuria, and lactic aciduria; these findings are consistent with Fanconi syndrome. Serum calcitriol and 25-hydroxyvitamin D concentrations were both subnormal.

The patient was diagnosed with metabolic bone disease secondary to Fanconi syndrome and chronic metabolic acidosis. Treatment with the Gonto protocol was initiated and included oral $NaHCO_3$ along with vitamin and amino acid supplementation. Pain was managed acutely with transdermal fentanyl; gabapentin and amantadine were prescribed for chronic pain control. Telmisartan was administered for proteinuria.

Drug and supplement protocols have been adjusted as necessary. More than 2 years later, the dog is now moderately azotemic with a creatinine of 1.84 mg/dL. Chloride is at the upper end of the reference range at 116 mmol/L, and urine protein:creatinine ratio is 2.5. The dog appears comfortable, with no evidence of any new fractures.

Comments

While a urinalysis should always be performed in conjunction with routine hematologic and biochemical testing, corrected hyperchloremia should serve as a reminder to evaluate the patient's concentrating ability, urine pH, and tubular function. Determination of the strong ion difference (SID) in a prefluid therapy urine sample can also help identify an RTA, as enhanced Cl^- uptake (and therefore decreased Cl^- excretion in urine) will increase this value.

$SID = Na^+ + K^+ - Cl^-$

Normal urine SID = 42 mEq/L[27]

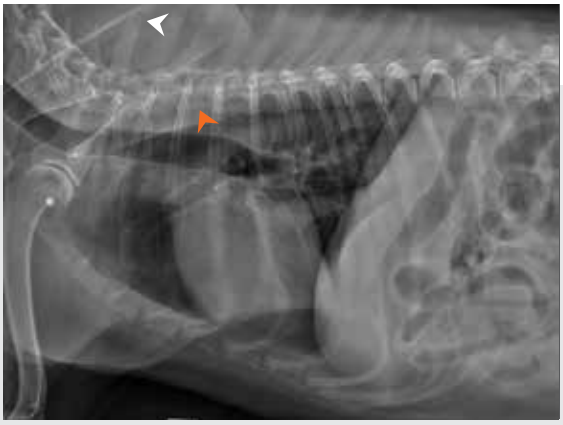


FIGURE 3. Left lateral thoracic radiograph of a 6-year-old neutered male Labrador retriever with Fanconi syndrome and associated metabolic bone disease. Note the fracture of the spinous process of the first thoracic vertebra **(white arrowhead)** and shortening of the fourth thoracic vertebral body with mild dorsal displacement of the cranial aspect **(orange arrowhead)**.

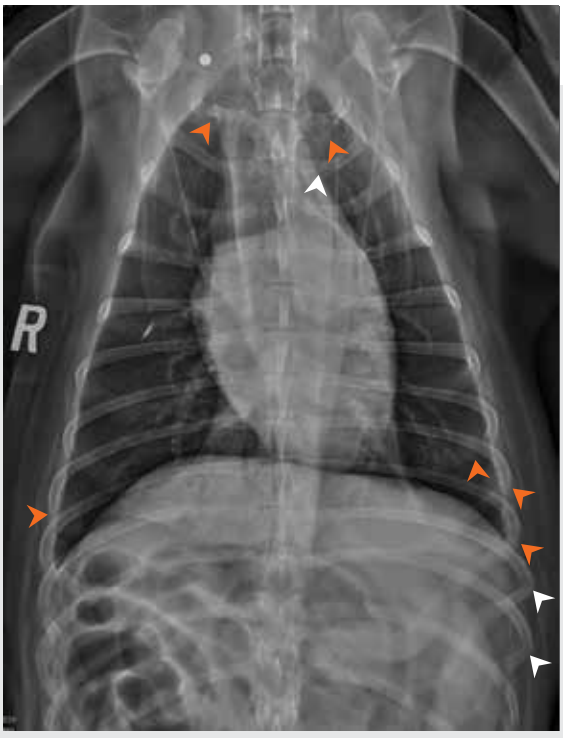


FIGURE 4. Ventrodorsal thoracic radiograph of a 6-year-old neutered male Labrador retriever with Fanconi syndrome and associated metabolic bone disease. There are multiple rib fractures at different stages of healing. Rib fractures with callus formation **(orange arrowheads)** include the following: left first, seventh, eighth, ninth, and right first and eighth. Rib fractures without evidence of callus formation and smooth margins **(white arrowheads)** include the following: left second, 10th, and suspect 11th.

Courtesy of Texas A&M Diagnostic Imaging Service (2)

In this case, the glycosuria indicated a tubular reabsorptive disorder and prompted the consideration of Fanconi syndrome. However, patients may have RTA without glycosuria, and provocative testing may be necessary to establish a definitive diagnosis. TVP

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