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Severe hyponatraemia in foals: clinical findings, primary diagnosis and outcome

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Objective To evaluate severe hyponatraemia in foals presenting as medical emergencies to an intensive care unit (ICU) in order to determine the prevalence, clinical findings, primary diagnosis and outcome.

Design Retrospective case study of records from Thoroughbred foals aged less than 3 months presenting to an ICU as medical emergencies in 2002–12; foals with severe hyponatraemia (serum sodium <122 mmol/L) on admission laboratory data were identified. Data retrieved included signalment, clinical findings, laboratory results, primary diagnosis, treatment and outcome.

Results Severe hyponatraemia was identified in 69/1718 Thoroughbred foals (4%) presenting to the ICU during the study period. Of the 69 foals, 11 (15.9%) presented with neurological signs attributable to hyponatraemic encephalopathy and 7 of these foals had seizures; other neurological signs included obtundation, ataxia and apparent blindness. The three most common primary diagnoses of the 69 foals with severe hyponatraemia were renal disease (18/69, 26.1%), enterocolitis (16/69, 23.2%) and uroperitoneum (15/69, 21.7%). Treatment was directed at the primary disease and correction of the hyponatraemia. A total of 50 of the 69 foals (72.5%) with severe hyponatraemia survived to hospital discharge and 38 of them (76%) survived at least 12 months following discharge.

Conclusion The prevalence of severe hyponatraemia in this study population was 4%. The majority of foals with severe hyponatraemia did not demonstrate direct clinical manifestations as a result of the low serum sodium concentration. The outcome of foals with severe hyponatraemia was mostly favourable.

Keywords critical care; foals; hyponatraemia; hyponatraemic encephalopathy

Abbreviations ICU, intensive care unit; IQR, interquartile range; Na^+, serum sodium concentration; TB, Thoroughbred

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Sodium is the dominant cation in the extracellular fluid and has an essential role in determining its tonicity and volume.[1] Despite wide fluctuations in water and salt intake, appropriate water and sodium balance usually maintains both the serum sodium concentration and serum osmolality within a relatively narrow range.[2] Hyponatraemia represents a relative excess of total body water to sodium[3] and occurs because of retention of free water and/or excessive loss of sodium.[1] Although there are many possible pathophysiological mechanisms for the development of severe hyponatraemia, most are associated with increased production or release of arginine vasopressin, an interruption in production or action of aldosterone, altered renal sodium transport and/or a reduction in glomerular filtration rate.[4] In foals, severe hyponatraemia has been defined as a serum sodium concentration of less than 122 mmol/L.[5]

Potential causes of severe hyponatraemia in foals include diarrhoea,[5,6] uroperitoneum,[7,8] renal disease,[9–11] rhabdomyolysis,[12] suspected transient pseudohypoaldosteronism secondary to urinary tract disorders such as hydroureter syndrome,[10,13] adrenal insufficiency[14] and over-zealous administration of water enemas, hypotonic fluids or excessive water intake.[3,10]

Severe hyponatraemia can result in a hyponatraemic encephalopathy with subtle to severe neurological deficits because of intracellular water accumulation and the development of cerebral oedema and a generalised encephalopathy.[15] The serum sodium concentration necessary to initiate abnormal neurological signs has been reported to be variable and depends on both the severity of the hyponatraemia and the duration over which the hyponatraemia occurs, with hyponatraemic encephalopathy being more likely with an acute (<48 h) drop in the serum sodium concentration.[3] To date, only 10 cases of hyponatraemic encephalopathy have been reported in the peer-reviewed literature as individual cases and small case series.[5,6,9,11,13,14]

The objectives of the present study were to establish the prevalence of severe hyponatraemia in Thoroughbred (TB) foals admitted as medical emergencies to an intensive care unit (ICU) and to collate the clinical findings, primary diagnosis and outcome of these foals with severe hyponatraemia.

Materials and methods

Case selection

Data including signalment, admission physical parameters and clinical pathological measurements were obtained from medical records of all TB foals aged less than 3 months presenting to Scone Equine Hospital's ICU as medical emergencies between 2002 and 2012. Severe hyponatraemia was defined as a serum sodium concentration (Na^+) less than 122 mmol/L.[5] Foals with severe hyponatraemia at admission were identified and selected for further evaluation.

Data collection

Signalment (sex, age of foal at admission (days), year of admission), history, physical examination findings at presentation, presence or absence of neurological signs, primary diagnosis, treatment and the

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short- and long-term outcomes were reviewed for each foal with severe hyponatraemia at admission. A complete blood count, fibrinogen, serum biochemistry, blood glucose and blood L-lactate concentrations were analysed from blood samples withdrawn from a jugular catheter during catheter placement at admission. Admission physical examination findings recorded by the attending clinician included heart rate, respiratory rate, rectal temperature and neurological signs (if present).

Foals with neurological signs attributable to hyponatraemic encephalopathy were defined as foals that were normal at birth but subsequently developed acute onset of neurological signs with concurrent severe hyponatraemia that quickly and completely resolved following an increase in the serum sodium concentration. The clinician's primary diagnosis was the single diagnosis most responsible for the foal's condition, even if other contributing diseases or problems were present. Details of treatments given during hospitalisation were recorded (including crystalloid and colloid fluid therapy, total parenteral nutrition, electrolyte supplementation, antimicrobial therapy, anti-diarrhoea medications and anti-ulcer medications).

Outcome variables evaluated were length of hospitalisation and short- and long-term survival. Short-term outcome was determined by whether the foal was discharged, euthanased or died. If the foal was euthanased, the reason for euthanasia was recorded when available (clinician-determined poor prognosis or financial reasons). Long-term outcome was based on follow-up information obtained from the *Australian stud book* (Racing Australia, Randwick, NSW). Registration was associated with survival beyond hospital discharge greater than 12 months. For foals that survived for more than 12 months following hospital discharge, whether the foal raced or not was recorded as an indicator of whether the foal achieved its intended use.

Statistical analysis

Summary statistics are reported as median and interquartile range (IQR) (25–75th percentile). Continuous data (e.g. admission rectal temperature, heart rate, respiratory rate, results of laboratory data) were not normally distributed. Wilcoxon rank-sum tests were used to determine the association of continuous data with (a) short-term survival (alive vs dead at hospital discharge) and (b) hyponatraemic encephalopathy. Categorical data were analysed via a Pearson's Chi-squared test. When there were less than five values per cell, a Fisher exact test was used. For all tests, $P \leq 0.05$ was considered statistically significant. All statistical analyses were performed using Microsoft Office Excel 97–2003 Worksheet (Microsoft Corporation, Redmond, WA, USA) and JMP 11 software (SAS Institute Inc., Cary NC, USA).

Results

Subjects' details and clinical findings

During the 11-year study period (2002–12), 1718 TB foals less than 3 months of age were admitted to the Scone Equine Hospital's ICU as medical emergencies and had complete admission biochemical results available for further analysis. All 1718 foals required intravenous fluid support and close monitoring as a minimum. Severe hyponatraemia (serum sodium concentration <122 mmol/L) was identified in 69 of the 1718 TB foals (4%); there were 40 colts and 29 fillies. Their median age at presentation to the ICU was 4 days (IQR 2–12 days).

Of the 69 foals with severe hyponatraemia, 58 (84.1%) did not demonstrate direct clinical manifestations as a result of the low serum sodium concentration and 11 (15.9%) presented with neurological signs attributable to hyponatraemic encephalopathy. The prevalence of hyponatraemic encephalopathy in foals admitted as medical emergencies to the ICU in the present study was 0.6% (11/1718).

Recorded neurological signs of hyponatraemic encephalopathy included obtundation (11/11), seizures (7/11), ataxia (7/11), apparent blindness with walking into walls (5/11), hyperaesthesia (3/11), head pressing (2/11) and circling (1/11) (Table 1). All foals presented with two or more of these neurological signs in varying combinations. Of the 7 foals with seizures, 3 had partial seizures, 3 had generalised seizures and 1 foal was in status epilepticus.

The 11 foals with hyponatraemic encephalopathy had significantly lower admission serum sodium (median serum sodium concentration 106 mmol/L, IQR = 101–111, n = 11) than foals without hyponatraemic encephalopathy (median serum sodium concentration 117.5 mmol/L, IQR 111–120, n = 58) (P = 0.0017) (Figure 1).

All historical data for foals with and without hyponatraemic encephalopathy were similar. The only physical examination finding recorded to be more common in foals with hyponatraemic encephalopathy than those foals without encephalopathy was visible oedema (P = 0.018, relative risk 12.9). There were no other differences in clinical pathology measurements between the two groups of foals.

Data on the exact duration of clinical signs prior to referral were not consistently available for all cases and few foals had a recently measured serum sodium concentration; therefore, the exact duration of the severe hyponatraemia could not be determined in most cases.

The three most common primary diagnoses in the 69 foals with severe hyponatraemia were renal disease (18/69, 26.1%), enterocolitis (16/69, 23.2%) and uroperitoneum (15/69, 21.7%). The primary

Table 1. Summary of the serum sodium concentration and neurological signs of the 11 obtunded foals with hyponatraemic encephalopathy and a primary diagnosis of renal disease

Case no.	Admission Na^+(mmol/L)	Neurological signs
1	110	Status epilepticus
2	103	Generalised seizures, ataxia, hyperaesthesia, circling
3	111	Generalised seizures, ataxia, apparent blindness
4	112	Generalised seizures, ataxia, hyperaesthesia, apparent blindness
5	99	Partial seizures, head pressing
6	101	Partial seizures, ataxia
7	114	Partial seizures, ataxia
8	99	Head pressing
9	104	Ataxia, apparent blindness
10	106	Ataxia, apparent blindness
11	106	Hyperaesthesia, apparent blindness

Na^+, serum sodium concentration.

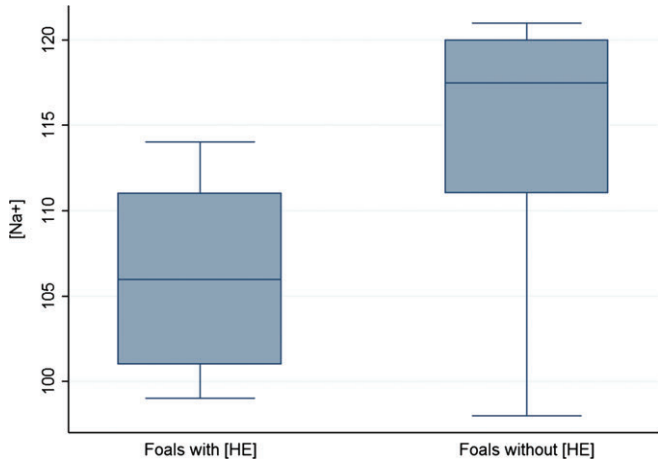

Figure 1. Box and whisker plot of the serum sodium concentration (mmol/L) at admission (Na^+) by foals with neurological signs of hyponatraemic encephalopathy (foals with (HE); n = 11) or foals without neurological signs of hyponatraemic encephalopathy (foals without (HE); n = 58) in the 69 foals with severe hyponatraemia at admission to the intensive care unit. The 'box' represents the 25–75th percentile range, the horizontal line within the 'box' represents the median and the 'whiskers' show the minimum to maximum value.

diagnoses in the remaining foals were colic (6/69, 8.7%), neonatal syndrome (6/69, 8.7%), sepsis (5/69, 7.2%), severe haemorrhage secondary to congenital coagulopathy (2/69, 2.9%) and rhabdomyolysis (1/69, 1.5%). In the subset of foals with hyponatraemic encephalopathy (n = 11), all had renal disease as the primary diagnosis. Of the 18 foals with severe hyponatraemia and a primary diagnosis of renal disease, 9 had received drugs with nephrotoxic potential in the preceding 3-week period prior to admission to the ICU (gentamicin, 7/9; phenylbutazone, 1/9; high-dose oxytetracycline, 44 mg/kg, (1/9)); 7 of the 18 foals had been treated for presumed sepsis in the neonatal period by the referring veterinarians and all had received gentamicin as part of their treatment.

Treatment

Treatment was directed at the primary disease and correction of the hyponatraemia.

Foals with hyponatraemic encephalopathy. All foals with hyponatraemic encephalopathy received 3% hypertonic saline administered via an infusion pump to increase the serum sodium concentration, with frequent monitoring of serum electrolyte concentrations (every 4–6 h) and repeated adjustment of the infusion rate as required; the remainder of their calculated fluid requirements was provided by 10% dextrose in water. Ingestion of free water and milk was restricted. The rate of administration of the 3% hypertonic saline was calculated using the Androgue formula[16] to calculate the sodium replacement therapy. This formula estimates the effects of 1 L of intravenous fluids on the patient's serum sodium concentration:

$$\text{Change in } Na^+ = \frac{(\text{infusate } Na^+ + \text{infusate } K^+) - Na^+}{\text{total body water} + 1}$$

The aim of treatment was to raise the serum sodium concentration by approximately 1.0–1.5 mmol/L/h for the first 3 h, but not to exceed a total increase of 10–12 mmol/L in the first 24 h. The serum sodium concentration was re-assessed every 4–6 h during this active phase of treatment in order to adjust therapy so that the correction stayed within these recommended limits. Once the serum sodium concentration had exceeded 120 mmol/L, the foal was then treated with balanced polyionic isotonic crystalloid fluids (Hartmann's solution).

Anticonvulsant medications (diazepam (4/11), phenobarbital (1/11), continuous-rate infusion of midazolam (1/11)) were used in the 4 foals with hyponatraemic encephalopathy and generalised seizures or status epilepticus. Systemic antimicrobials (11/11), anti-ulcer medications (3/11), hyperimmune plasma (3/11), dopamine (1/11) and fenoldopam (1/11) were also administered as part of the treatment plan. Oral electrolyte supplementation was used in 6 of the 11 foals once their serum sodium concentration exceeded 120 mmol/L. In 10 of the foals, the neurological signs had significantly decreased within 12 h of initiating treatment and had completely resolved within 24 h of initiating treatment. One of the foals went into respiratory arrest within the first hour of initiating treatment and attempts at resuscitation were unsuccessful. This foal was in status epilepticus on presentation to the ICU. A postmortem examination was not performed.

Foals without hyponatraemic encephalopathy. Of the 58 foals with severe hyponatraemia but without hyponatraemic encephalopathy, 6 received 3% hypertonic saline and 10% dextrose in water in a similar manner as described above. However, the desired correction rate was notably slower, with the goal of increasing the serum sodium concentration by not greater than 0.5 mmol/L/h and again to substitute with balanced polyionic crystalloid fluids when the foal's serum sodium exceeded 120 mmol/L. The foals' serum electrolytes were monitored every 4–6 h so that the infusion rate could be adjusted as necessary. The remaining 52 foals received isotonic crystalloid fluids with regular (4–6 h) monitoring of serum electrolyte concentrations. Systemic antibiotics (50/58), oral electrolyte supplementation (20), hyperimmune plasma (16), flunixin (16), anti-ulcer medications (14), intranasal oxygen (13), anti-diarrhoea medications (10), furosemide (6), total parenteral nutrition (5), inopressors (6), dopamine (3) and a blood transfusion (2) were also administered. Corrective surgery was performed in the 15 foals with uroperitoneum when they had been medically stabilised.

Outcomes

A total of 50 of the 69 foals (72.5%) with severe hyponatraemia survived to hospital discharge. Of the 19 foals that did not survive, 5 died in hospital and 14 were euthanased because of clinician-determined poor prognosis. None were euthanased at admission without an attempt at stabilisation nor were any euthanased because of financial constraints during the hospitalisation period. The median duration of hospitalisation of surviving foals was significantly longer than that of non-surviving foals: 7 days (IQR 5–9, n = 50) and 3 days (IQR 1–5, n = 19), respectively (P = 0.0007). There was no significant difference between the admission serum sodium in foals with severe hyponatraemia that survived to hospital discharge (median serum sodium concentration 116 mmol/L, IQR

111–119, n = 50) and those that did not (median serum sodium concentration 114 mmol/L, IQR 109–120, n = 19) (P = 0.56).

Long-term follow-up through the *Australian stud book* was available for all foals that survived to hospital discharge. Of the 50 foals that survived to hospital discharge, 38 (76%) were registered (estimated survival at least 12 months after hospital discharge). There was no significant difference in registration for hyponatraemic foals with hyponatraemic encephalopathy (7/10; 70%) and hyponatraemic foals without hyponatraemic encephalopathy 31/40 (77.5%) (P = 0.69). Of the 38 confirmed long-term survivors, 34 (89.5%) went on to race.

Discussion

To the authors' knowledge, this is the first large, retrospective study evaluating severe hyponatraemia at admission in hospitalised TB foals. The prevalence of severe hyponatraemia in TB foals less than 3 months of age presenting as medical emergencies to the ICU was low. The majority of foals with severe hyponatraemia (84.1%) did not demonstrate neurological signs as a result of the low serum sodium concentration; 15.9% of foals with severe hyponatraemia showed neurological signs consistent with hyponatraemic encephalopathy.

Only 10 cases of hyponatraemic encephalopathy have been described in the peer-reviewed literature as individual cases or small case series thus far.[5,6,9,11,13,14] In the present study performed over an 11-year period the prevalence of hyponatraemic encephalopathy in foals admitted as medical emergencies to the ICU was very low. Although hyponatraemic encephalopathy was found to be an infrequent cause of neurological signs in young foals, it still should be considered in the differential diagnoses for foals showing neurological signs such as obtundation, seizures, ataxia and other non-specific central signs, particularly as resolution of neurological signs with appropriate treatment can be relatively quickly achieved in the majority of cases.

Prior descriptions of neurological signs associated with hyponatraemic encephalopathy have included obtundation,[5,9] seizures,[5,6,9,11,14] blindness,[5,6] head pressing[5,11] and ataxia.[5,11,13] The neurological signs in the 11 foals in the present study were similar to those previously reported.

It is reportedly difficult to accurately predict the serum sodium concentrations above or below which the clinical signs of hyponatraemic encephalopathy are likely to occur.[17] A review of severe hyponatraemia in humans found that severe neurological complications were most commonly observed when serum sodium decreased at a rate >0.5 mEq/L/h.[18] If osmolar change occurs too rapidly, outpacing the brain's ability to adapt, cerebral oedema can result in severe neurological deficits.[19] Given time, brain cells extrude organic solutes from their cytoplasm, allowing intracellular osmolality to equal plasma osmolality without a large increase in cell water. Therefore, when hyponatraemia develops over several days, brain swelling is minimised so that in patients with chronic (>48 h) hyponatraemia, minimal neurological signs may be exhibited despite very low serum sodium concentrations.[19]

Hurcombe previously noted that the presence of neurological signs in horses with severe hyponatraemia was variable when the serum sodium concentration was 115–120 mmol/L and that neurological signs were more reliably present when the serum sodium concentration was less than 115 mmol/L.[3] In the previously reported cases of hyponatraemic encephalopathy in foals in the veterinary literature,[5,6,9,11,13,14] 7 of the foals had concurrent serum sodium concentrations less than 115 mmol/L,[5,6,9,13,14] and 3 of the foals had serum sodium concentrations between 115 and 120 mmol/L.[11] All of the foals with hyponatraemic encephalopathy in the present study had serum sodium concentrations less than 115 mmol/L. Foals with hyponatraemic encephalopathy in this study were found to have significantly lower serum sodium concentrations than foals with severe hyponatraemia without hyponatraemic encephalopathy (P = 0.0017) (Figure 1). Although the absolute value of serum sodium concentration is relevant when evaluating foals with severe hyponatraemia, the rapidity of change is also likely to be a very important determinant for the development of hyponatraemic encephalopathy. Because of the retrospective nature of this study, we could not specifically evaluate this factor.

The most common primary diagnoses of foals with severe hyponatraemia were renal disease (26.1%), enterocolitis (23.2%) and uroperitoneum (21.7%). Not only was renal disease found to be the most common primary diagnosis in foals with severe hyponatraemia at admission, it was also the primary diagnosis of all 11 foals with hyponatraemic encephalopathy. This highlights the importance of the kidneys in the delicate balance of electrolyte and water regulation. When the 10 foals in previously reported cases of hyponatraemic encephalopathy were reviewed, [5,6,9,11,13,14] 6 had a primary diagnosis of renal disease,[9,11,13] and 2 had renal disease with a second diagnosis (rhabdomyolysis, enterocolitis).[5] The remaining cases were primary enterocolitis[6] and adrenal insufficiency.[14] The predominance of renal disease as the primary diagnosis in foals with hyponatraemic encephalopathy, both in the present study and in the cases previously reported in the literature, supports a critical examination of renal function in foals presenting with hyponatraemic encephalopathy. This has been recently highlighted by Hardefeldt in a case series of 4 foals with hyponatraemic encephalopathy.[11]

Potential mechanisms for the development of severe hyponatraemia in renal disease would include a reduction in the glomerular filtration rate and resultant water retention, increased sodium loss with renal tubular damage[20] and interruption in the action of aldosterone.[13] Active water excretion by the kidneys is especially important in foals for the maintenance of serum osmolality in the normal range. Foals may ingest a volume of milk in excess of 20% of their body weight daily[20] and mare's milk is low in sodium (9–12 mmol/L).[21] Failure to produce a large volume of hyposthenuric urine in renal disease can result in water retention, decreased serum osmolality and severe hyponatraemia with or without concurrent neurological signs.[20]

Accurate and timely identification of clinically important sodium/water imbalance is an essential part of equine critical care medicine.[4] However, severe hyponatraemia presents a therapeutic challenge for equine clinicians because of the paucity of information available in the literature, necessitating extrapolation of treatment protocols from

other species.[5] Wong et al. suggested basing treatment on several factors, including the primary diagnosis, the volume status of the foal, the duration and severity of the hyponatraemia, and the presence or absence of hyponatraemic encephalopathy.[5] Treatment of severe hyponatraemia in the setting of severe neurological signs requires a small, rapid rise in the serum sodium concentration (1–-2 mmol/L/h) for the first 2–3 h of treatment until neurological signs abate, to reduce cerebral oedema and further deterioration of the neurological status but not to exceed a total increase of 10–12 mmol/L in the first 24 h of treatment.[4,5] Human studies have shown that a relatively small increase in the serum sodium concentration, in the order of 5%, can substantially reduce cerebral oedema in cases of hyponatraemic encephalopathy.[16] More conservative correction is advisable when clinical signs are mild or absent and hyponatraemia is chronic, because of the potential risk of inducing osmotic demyelination syndrome, which can result in severe neurological impairment and/or death.[4] Osmotic demyelination syndrome in humans has been most commonly associated with rapid correction of chronic hyponatraemia.[11] The syndrome has not as yet been reported in horses.[11]

In all 69 foals in this study with severe hyponatraemia, the aim was to increase the serum sodium concentration to a safer level (>120 mmol/L) without rapid restoration of normal serum sodium concentrations. The 11 foals with hyponatraemic encephalopathy were considered at greatest risk from the neurological complications of hyponatraemia itself, while the 58 foals with severe hyponatraemia without hyponatraemic encephalopathy were considered at minimal risk from complications of hyponatraemia itself, but at potential risk of osmotic demyelination syndrome following overly rapid correction. A combination of 3% hypertonic saline administered by an infusion pump, together with 10% dextrose in water to provide the remainder of the foal's fluid requirements, was used in all 11 foals with hyponatraemic encephalopathy and in 6 of the 58 foals with severe hyponatraemia without hyponatraemic encephalopathy. The Androgue formula was used to calculate an appropriate rate of sodium replacement therapy,[16] although the validity of this formula in veterinary species has not been corroborated.[4] The rate of sodium replacement therapy was initially faster in foals with hyponatraemic encephalopathy to prevent the development of further cerebral oedema and worsening of neurological function.

In the remaining 52 foals with severe hyponatraemia without hyponatraemic encephalopathy at admission, isotonic crystalloid fluids were used to replace the foals' effective circulating volume. Most of these foals were considered to be hypovolaemic. In volume depletion, the body will try to restore effective circulating volume to preserve tissue perfusion. Arginine vasopressin is released from the pituitary gland, resulting in resorption of free water at the level of the collecting tubules of the kidneys. This excess water preservation relative to sodium may result in a decrease in plasma osmolality and hyponatraemia. When euvolaemia is restored, the physiological stimulus for vasopressin release and free water retention should be removed, allowing restoration of appropriate water balance and serum sodium concentration.[22] Regular re-assessment of serum electrolyte concentrations was performed for all 52 foals with severe hyponatraemia in this study to assess the rate of correction, aiming not to exceed a total increase of 10–12 mmol/L of sodium per day to decrease the potential risk of osmotic myelination syndrome.

In the 10 previously reported cases of hyponatraemic encephalopathy in the literature,[5,6,9,11,13,14] all the foals were reported to have demonstrated a rapid improvement in neurological status and subsequent complete resolution of their neurological signs within 48 h of initiating treatment to increase their serum sodium concentrations. In the 11 foals with hyponatraemic encephalopathy in this study, response to treatment was excellent in 10 of 11 foals, with partial correction of the severe hyponatraemia. In these 10 foals, the neurological signs had significantly decreased within 12 h and no signs were present within 24 h of initiating treatment. Although more advanced imaging techniques (such as computed tomography) were not available in the ICU to confirm the presence of cerebral oedema, a temporal relationship between correction of the electrolyte abnormality and resolution of seizures and encephalopathic signs has been reported previously to support causation.[11,17] One of the 11 foals with hyponatraemic encephalopathy went into respiratory arrest during the first hour of treatment and attempts to resuscitate it were unsuccessful. This foal had presented to the ICU with severe neurological signs (status epilepticus). Although respiratory arrest has been previously reported in horses with hyponatraemic encephalopathy, because of impairment of central respiratory centres,[3] a post-mortem examination would have been useful to determine the neuropathology present in this foal.

Study limitations

The authors recognise limitations to the definition of foals with hyponatraemic encephalopathy used in this study. The definition was primarily based on the response to treatment of the small number of previously reported cases of hyponatraemic encephalopathy in foals.[5,6,9,11,13,14] The acute onset of cerebral oedema in hyponatraemic encephalopathy can result in a severe increase in intracranial pressure and adverse sequelae, including brain herniation, respiratory arrest and death.[3] The definition used in the study did not account for this. Additionally, although none of the surviving foals in this study were noted to show ongoing neurological deficits, mild neurological deficits might have been difficult to perceive.

In the present study, the short-term survival rate for foals with severe hyponatraemia was good (72.5%). The serum sodium concentration on admission to the ICU did not predict survival in foals with severe hyponatraemia. At least 76% of the short-term survivors in the current study were confirmed to have survived at least 12 months post hospital discharge and 89.5% of the long-term survivors were subsequently raced. One limitation of this study is that the long-term follow-up results may have underestimated the number of horses that survived at least 12 months after hospital discharge if surviving horses were not subsequently registered with the *Australian stud book*. The racing performance of long-term surviving horses was not specifically evaluated in this study; however, at least one race start implied some degree of athletic potential and indicates that the horse achieved its intended use. The favourable outcome in the majority of severely hyponatraemic foals with or without hyponatraemic encephalopathy in the present study should encourage practitioners

to pursue appropriate and timely diagnostic investigation and treatment of foals with severe hyponatraemia.

Conclusion

The prevalence of severe hyponatraemia in foals on presentation as medical emergencies was 4%. The majority of foals with severe hyponatraemia did not demonstrate neurological signs as a result of the low serum sodium concentration. Hyponatraemic encephalopathy was infrequently diagnosed, but still should be considered in the differential diagnoses for foals showing neurological signs such as obtundation, seizures, ataxia and apparent blindness. Renal disease, enterocolitis and uroperitoneum were the most common primary diagnoses in foals with severe hyponatraemia. As long as the primary problem was resolved, the prognosis for foals with severe hyponatraemia with or without hyponatraemic encephalopathy was favourable.

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