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8 **Severe hypophosphataemia associated with the management of hyperlipaemia in a**
9 **miniature pony**

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17 **Keywords:** horse; glucose; insulin; phosphorus; triglycerides

18

19 **Summary**

20 This report describes a case of severe hypophosphataemia associated with the
21 management of hyperlipaemia in a miniature pony following colic surgery. Clinical signs
22 attributed to hypophosphataemia included obtundation, anorexia, tachycardia, tachypnoea
23 and generalised muscle fasciculations. Hyperlipaemia was managed with enteral and partial
24 parenteral nutrition; insulin was also administered to control hyperglycaemia after the
25 initiation of caloric support. Specific therapy for hypophosphataemia consisted of parenteral
26 potassium phosphate at 0.03 mmol/kg bwt/h (i.v.). The pony made a full recovery without
27 further complications. Hypophosphataemia may be an under-recognised clinical problem in
28 certain populations of critically ill equids, such as those with hyperlipaemia and receiving

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29 insulin as part of their management. The routine measurement of phosphate concentration in
30 these cases is recommended.

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31 **Introduction**

32 Hyperlipaemia is a well-described condition of miniature equine breeds that enter a
33 negative energy balance, due to the high hepatic efficiency of this species in the synthesis and
34 release of triglycerides (TG) into the circulation (Hughes *et al.* 2004; McKenzie 2011).

35 Conditions that increase energy demand or limit energy intake, including recent colic surgery,
36 are risk factors for hyperlipaemia (Jeffcott and Field 1985; Hughes *et al.* 2003). Differences
37 in glucose, insulin and lipid dynamics, and the high incidence of obesity, may also explain
38 the especially frequent occurrence of hyperlipaemia in miniature ponies and donkeys (Gay *et*
39 *al.* 1978; Burden *et al.* 2011).

40 Various terminologies have been used to describe the degree of hypertriglyceridaemia
41 in equids, including hyperlipidaemia, severe hypertriglyceridaemia and hyperlipaemia
42 (McKenzie 2011). Hyperlipaemia is used when TG concentrations exceed 5.7 mmol/l (500
43 mg/dl), the plasma becomes grossly lipaemic and clinical signs of disease are present.
44 Conversely, hyperlipidaemia is used when TG concentrations are between 1.0 and 5.7
45 mmol/l, the plasma remains clear and clinical signs of disease are absent. Severe
46 hypertriglyceridaemia is usually reserved to describe TG concentrations greater than 5.7
47 mmol/l in large-breed horses that are typically asymptomatic (Dunkel and McKenzie 2003).

48 Early reports of equids with hyperlipaemia suggested that the prognosis was poor,
49 with survival rates as low as 30% (Watson *et al.* 1992). The routine measurement of plasma
50 TG concentrations in predisposed individuals is now standard practice at many hospitals.
51 Early and aggressive caloric support has now improved the survival of individuals in which
52 hypertriglyceridaemia is promptly identified (Durham 2006; Durham and Thiemann 2015).

53 Hypophosphataemia is a well-recognised comorbidity of critically ill humans that is
54 associated with increased mortality when left untreated (Bugg and Jones 1998).
55 Hypophosphataemia has also been mentioned as a potential sequela to hyperlipaemia,
56 parenteral nutrition and insulin therapy in equids (Magdesian 2010; Toribio 2015), and it may
57 therefore contribute to the morbidity or mortality of affected animals if not recognised and
58 treated appropriately. To the authors' knowledge, there are no clinical case descriptions of
59 acute hypophosphataemia in the equine literature. The purpose of this report was to describe
60 the clinical progression of a miniature pony with severe hypophosphataemia associated with
61 the management of hyperlipaemia, and to highlight the importance of routine phosphate
62 measurement in these cases.

63

64 **Case details**

65 ***History and initial case management***

66 A 10 year old miniature pony gelding (139 kg; BCS 7 out of 9 [Henneke *et al.* 1983])
67 presented to the University of Melbourne's Equine Centre with a 10 hour history of moderate
68 colic that had been unresponsive to medical management. The referring veterinarian had
69 administered flunixin meglumine (1.1 mg/kg bwt i.v.), xylazine hydrochloride (0.2 mg/kg
70 bwt i.v.) and paraffin oil (200 ml via nasogastric tube); however, colic improved only
71 temporarily. On admission the pony was moderately painful. Pertinent physical examination
72 findings included tachycardia (80 beats/min), tachypnoea (30 breaths/min), absent
73 borborygmi and abdominal distension. The oral mucous membranes were of normal colour
74 but tacky to the touch with a prolonged capillary refill time. A limited rectal examination
75 revealed marked gas distension of the large colon. Packed cell volume (33%; reference 25–
76 45%), plasma total solids (56 g/l; reference 52–79 g/l), L-lactate (2.3 mmol/l; reference <1.5
77 mmol/l) and glucose (6.9 mmol/l; reference 4.2–6.7 mmol/l) concentrations were measured.
78 Plasma electrolyte concentrations (sodium, potassium, chloride, ionised calcium) and venous
79 blood gas analyses performed on admission were within normal ranges.

80 Based on the history, clinical findings and increasing level of pain, the pony was
81 administered flunixin meglumine (1.1 mg/kg bwt i.v.), procaine penicillin (22 mg/kg bwt
82 i.m.) and gentamicin (6.6 mg/kg bwt i.v.), and an exploratory laparotomy was performed. At
83 surgery, marked gas distension of the large colon was relieved and a faecalith removed from
84 the transverse colon via an enterotomy at the pelvic flexure. The pony received a buffered
85 and balanced, polyionic crystalloid solution (Hartmann's¹) whilst under anaesthesia (10
86 ml/kg bwt/h i.v.) and for 6 hours post-operatively (5 ml/kg bwt/h i.v.). Recovery from
87 anaesthesia was uneventful.

88
89 ***Post-operative case management***

90 Six hours after surgery the pony was bright and alert. Pertinent physical examination
91 findings included tachycardia (60 beats/min) and reduced borborygmi. Hydration was
92 considered adequate on the basis of physical examination findings, packed cell volume (32
93 %), plasma total solids (55 g/l) and L-lactate (1.2 mmol/l) concentration. Blood glucose
94 concentration was 6.7 mmol/l, whilst plasma electrolyte concentrations (sodium, potassium,
95 chloride, ionised calcium) and venous blood gas analyses were within normal ranges. Mild
96 hyperlipidaemia was identified when plasma TG concentration (2.3 mmol/l; reference 0.1–
97 1.0 mmol/l) was measured as part of the routine monitoring protocol for miniature equids.
98 The pony had been inappetent for approximately 24 hours at this time.

99 A selection of fresh grass, pasture hay, lucerne chaff and pelleted feed was offered to
100 encourage voluntary feed intake. Intravenous fluid therapy was discontinued, with a plan to
101 closely monitor hydration status and water intake during the next 24 hours. Flunixin
102 meglumine (1.1 mg/kg bwt i.v., q12h) was administered to provide pain relief. Post-operative
103 antimicrobial prophylaxis consisted of procaine penicillin (22 mg/kg bwt i.m., q12h) and
104 gentamicin (6.6 mg/kg bwt i.v., q24h) for 3 days. The pony maintained a moderate appetite
105 during the first day, although due to the many feed options made available, actual intake was
106 difficult to quantify. Hyperlipidaemia was found to have progressed 12 hours post-
107 operatively (TG 4.3 mmol/l). Additional caloric support was instituted in an attempt to avert
108 the potential development of clinical hyperlipaemia.

109 A pelleted feed (Gumnuts²; 14 MJ/kg digestible energy, 16% protein, 8% fibre, 7%
110 fat) was blended with water to form a gruel and administered via nasogastric tube every 4
111 hours to meet estimated resting energy requirements of 100 kJ/kg bwt/d (Durham and
112 Thiemann 2015). However, hyperlipidaemia continued to progress and TG concentrations
113 were 5.7 mmol/l when measured 18 hours after surgery. Additional caloric support was
114 subsequently provided with a balanced, polyionic (Hartmann's¹) solution supplemented with
115 dextrose to a concentration of approximately 2.5% and potassium chloride to a concentration
116 of approximately 20 mmol/l. An initial maintenance fluid rate of 2 ml/kg bwt/h (i.v.)
117 delivered dextrose at 1 mg/kg bwt/min, providing approximately 25 kJ/kg bwt/d. Enteral
118 nutrition continued as outlined previously to provide the remainder of the estimated resting
119 energy requirement. Blood glucose concentrations were measured regularly (q1h for the first
120 6 h and q2h for the next 12 h) after commencing the dextrose infusion (Fig 1). Significant
121 hyperglycaemia (13.8 mmol/l; reference 4.6 – 7.9 mmol/l) was encountered after 3 hours of
122 the dextrose infusion, for which a dose of regular insulin was administered (0.1 i.u./kg bwt
123 s.c.). Blood glucose concentrations subsequently decreased and remained within acceptable
124 ranges thereafter (Fig 1).

125 On the second day of hospitalisation and 30 hours after surgery, the pony was
126 obtunded and minimally responsive to stimuli. Rectal temperature (38.0°C) was normal, but
127 the pony was tachycardic (60 beats/min, regular rhythm) and tachypnoeic (40 breaths/min).
128 Prominent generalised muscle fasciculations, most evident in the triceps and quadriceps
129 muscle groups, were present. Triglyceride concentration was again higher (8.3 mmol/l) and
130 the plasma now appeared lipaemic. Packed cell volume (35%), plasma total solids (58 g/l),
131 and L-lactate concentration (1.3 mmol/l) were within normal ranges. A complete plasma
132 biochemistry profile showed electrolyte concentrations (sodium, potassium, chloride, total

133 calcium, total magnesium) and markers of muscle, liver and renal function to be within
134 normal ranges. However, plasma phosphate concentration was extremely low (0.1 mmol/l;
135 reference 0.8–1.8 mmol/l).

136 Specific therapy to correct hypophosphatemia was initiated. Potassium phosphate³ (1
137 mmol/ml phosphate ions; diluted in 0.9% saline solution) was provided at a rate of 0.03
138 mmol/kg bwt/h (i.v.). Enteral nutrition and the dextrose infusion continued as previously
139 described. Phosphate concentration measured 8 hours after commencement of the infusion
140 had only improved marginally (0.2 mmol/l) and clinical signs attributed to
141 hypophosphataemia persisted. The infusion was continued overnight at 0.03 mmol/kg bwt/h.

142 On the third day of hospitalisation and 54 hours after surgery, the pony appeared
143 bright and alert and muscle fasciculations had ceased. Physical examination findings were
144 unremarkable. Phosphate concentration (24 hours after commencing the infusion) had
145 improved to 0.6 mmol/l and hyperlipaemia had resolved (TG 1.4 mmol/l, transparent
146 plasma). The phosphate and dextrose infusions were discontinued later that day, because
147 phosphate and TG concentrations had returned to normal ranges (Fig 1) and the pony was
148 eating well. The pony was walked to grass every 4 hours, small meals of chaff and pellets
149 were provided every 6 hours, and hay was re-introduced using a graduated protocol for
150 feeding post-operative colic patients. The pony was discharged on day 8 of hospitalisation
151 and was reported to be clinically healthy 6 months later.

152

153 Discussion

154 Phosphate is an essential anion that must be readily available to cells for effective
155 energy homeostasis (DiBartola and Willard 2012). Phosphate actively participates in
156 glycolytic processes through the activation of key enzymes and the phosphorylation of many
157 intermediate metabolites. The energy that is ultimately utilised by cells is stored within the
158 phosphate bonds of adenosine triphosphate (ATP). Phosphate also plays important roles in
159 cell membrane structure, acid-base equilibrium, oxygen transport and gene transcription
160 (Toribio 2010). Most phosphorus (85%) is stored in the body as hydroxyapatite in bone, 14 to
161 15% is intracellular and less than 1% is extracellular (Schropp and Kovacic 2007).
162 Approximately two-thirds of circulating phosphate is organic (as phospholipids and
163 phosphoproteins) while the remaining one-third consists of the inorganic form that is
164 measured by chemistry analysers.

165 Hypophosphataemia is likely to have the most profound effect on tissues with a high
166 metabolic requirement such as erythrocytes, skeletal muscle and the central nervous system.

167 The clinical signs of hypophosphataemia in horses are not well characterised, but those
168 described in human patients include muscular weakness, myalgia, obtundation, seizures,
169 respiratory insufficiency and cardiac arrhythmias (Gaasbeek and Meinders 2005). More
170 severe clinical signs include rhabdomyolysis and haemolytic anaemia. In agreement with
171 these observations, the pony in this case report demonstrated obtundation, tachycardia,
172 tachypnoea and generalised muscle fasciculations. Although these clinical signs are relatively
173 non-specific, in the authors' opinion, the severity of obtundation and muscle fasciculations
174 were more pronounced than expected with hyperlipaemia alone.

175 Hypophosphataemia can occur as the result of inadequate intake, excessive losses or
176 intracellular redistribution. Chronic renal failure may be the most common cause of
177 hypophosphataemia in horses, although clinical problems attributed to hypophosphataemia in
178 these cases have not been recognised (Schott 2007). Other causes of hypophosphataemia
179 include prolonged insufficient dietary intake or absorption, starvation and 'refeeding
180 syndrome', parenteral nutrition, insulin therapy, respiratory alkalosis, hyperparathyroidism or
181 the release of parathyroid hormone-related protein by certain tumours (Toribio 2011).
182 Phosphatonins such as fibroblast growth factor-23 may play a role in the development of
183 hypophosphataemia in critically ill humans through the induction of phosphaturia (Berndt
184 and Kumar 2007). Spurious results should also be considered when evaluating phosphate
185 concentrations; however, the presence of lipaemia usually causes a false increase in
186 phosphate concentration (Hughes *et al.* 2004).

187 The development of hypophosphataemia in this pony was attributed to an intracellular
188 redistribution of phosphate associated with the combination of probable insulin resistance,
189 short-term inappetence, hyperlipaemia, the provision of enteral and partial parenteral
190 nutrition, and the administration of exogenous insulin. The intracellular movement of
191 phosphate accompanies insulin-mediated glucose uptake into cells in order to sustain
192 glycolytic processes (Bugg and Jones 1998). Alkalaemia (respiratory alkalosis, bicarbonate
193 administration) and catecholamine release also stimulate glycolysis and promote an
194 intracellular redistribution of phosphate. Hypophosphataemia can be further exacerbated if
195 intracellular phosphate stores become depleted during a period of anorexia before the
196 initiation of caloric support.

197 Insulin therapy might be a precipitating cause in some cases of hypophosphataemia,
198 although it is often difficult to separate this effect from coexisting risk factors. Although
199 hypophosphataemia can occur without the administration of exogenous insulin, in previous
200 cases managed by the authors the appearance of clinical signs attributed to

201 hypophosphataemia only occurred after exogenous insulin was given. Therefore, the routine
202 monitoring of phosphate concentrations may be warranted in equids receiving insulin
203 therapy.

204 Anecdotally, some clinicians have experience with the management of acute
205 hypophosphataemia in equids that are being managed for hyperlipaemia (Toribio 2015).
206 However, hypophosphataemia was not reported as a comorbidity in any of the major clinical
207 case series of equine hyperlipaemia (Watson *et al.* 1992; Moore *et al.* 1994; Mogg and
208 Palmer 1995; Dunkel and McKenzie 2003; Hughes *et al.* 2003; Durham 2006; Burden *et al.*
209 2011). It is not clear if this is because the animals studied were normophosphataemic or
210 because phosphate concentrations were not measured. The absence of phosphate from some
211 biochemistry panels could mean that hypophosphataemia is an under-recognised clinical
212 problem in equids with hyperlipaemia.

213 Hypophosphataemia is a relatively frequently encountered condition in critically ill
214 human patients (Taylor *et al.* 2004). The degree of hypophosphataemia can be defined as
215 mild (0.66–0.80 mmol/l), moderate (0.32–0.65 mmol/l) and severe (<0.32 mmol/l) (Gaasbeek
216 and Meinders 2005). Severe hypophosphataemia is associated with increased morbidity and
217 mortality in hospitalised human patients and there is strong evidence that targeted phosphate
218 replacement improves survival (Bugg and Jones 1998). However, there is conflicting
219 evidence as to whether moderate hypophosphatemia requires specific treatment (Amanzadeh
220 and Reilly 2006).

221 Recommendations for phosphate replacement therapy in horses have been
222 extrapolated from human and small animal guidelines (Toribio 2015). Parenteral (i.v.)
223 phosphorus replacement been suggested at rates of 0.01 to 0.08 mmol/kg bwt/h (Amanzadeh
224 and Reilly 2006; DiBartola and Willard 2012). More aggressive rates, up to 0.3 mmol/kg
225 bwt/h for 1 to 2 hours, have also been used (Geerse *et al.* 2010). One approach that has been
226 suggested for horses is to replace the estimated deficit over a 1-hour period (yielding a rate of
227 up to 0.3 mmol/kg bwt/h) with the treatment repeated every 8 hours as necessary (Toribio
228 2015). The extracellular phosphate deficit (mmol) can be calculated as: bwt (kg) x (desired
229 phosphate concentration [mmol/l] – current phosphate concentration [mmol/l]) x 0.3 (the
230 extracellular fluid coefficient). Care must be taken to avoid iatrogenic hyperphosphataemia,
231 although the threshold rate at which this becomes a concern is not known for horses. In the
232 present case, a cautious rate of parenteral phosphate supplementation was empirically
233 selected (0.03 mmol/kg bwt/h), resulting in the resolution of clinical signs in 24 hours and a
234 gradual return to normophosphataemia in 32 hours.

235 This case report highlights the importance of phosphate measurement in order to
236 identify hypophosphataemia in critically ill equids, such as those with hyperlipaemia. Insulin
237 resistance, enteral or parenteral nutrition, the administration of exogenous insulin and
238 increased substrate phosphorylation may result in an acute intracellular shift of phosphate.
239 Specific phosphate replacement therapy in cases of severe hypophosphataemia is warranted.

240

241 **Authors' declaration of interests**

242 No conflicts of interest have been declared.

243

244 **Ethical animal research**

245 Institutional animal ethics approval was not required for this case report. Owner consent was
246 obtained for all aspects of case management and prior to submission of the manuscript.

247

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249 No funding received.

250

251 **Antimicrobial stewardship policy**

252 The pony was not administered quinolones, extended spectrum beta-lactam antimicrobials or
253 macrolides.

254

255 **Authorship**

256 All authors contributed to clinical case management. The manuscript was drafted by N. J.
257 Bamford and revised by the other authors. All authors approved the final version of the
258 manuscript.

259

260 **Manufacturers' addresses**

261 ¹Baxter Healthcare, Toongabbie, New South Wales, Australia.

262 ²Mitavite, Somersby, New South Wales, Australia.

263 ³Hospira, Melbourne, Victoria, Australia.

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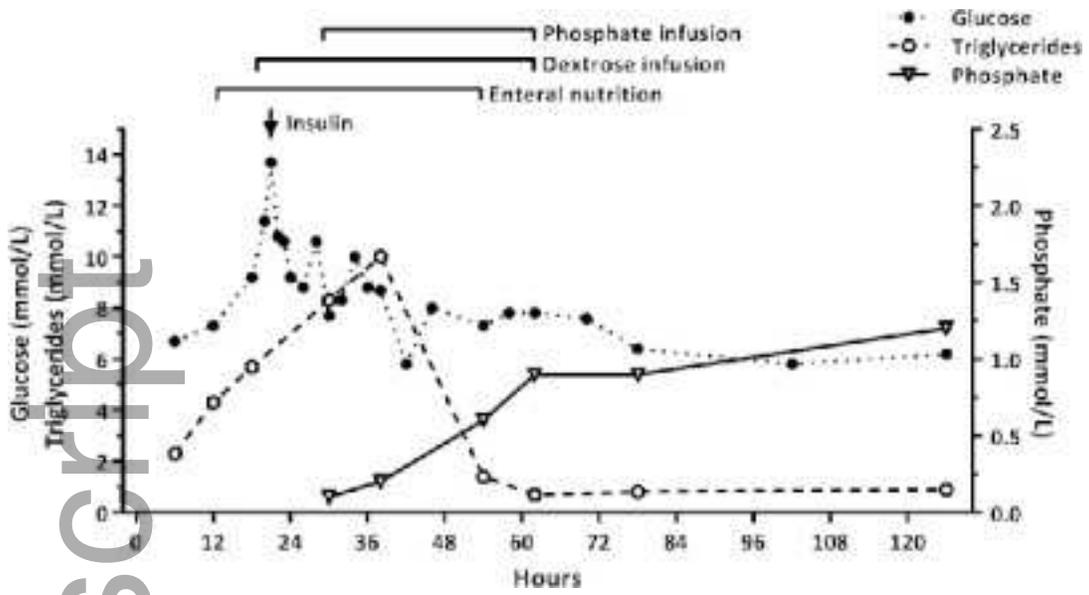
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320 **Figure legend**

321 **Fig 1:** Sequential measurements of glucose, triglyceride and phosphate concentrations in this
322 case. Time 0 indicates the end of surgery.

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