

Case Report

Severe hyperkalaemia associated with renal dysplasia in a 2-day-old foal

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Summary

A 2-day-old filly foal presented with signs of depression, recumbency and inappetence. Blood analyses revealed hypoalbuminaemia, hyperfibrinogenaemia, hyperglycaemia and hyperkalaemia. The foal deteriorated despite intensive treatment and was subjected to euthanasia. At post mortem examination, the urinary bladder, ureters and kidneys appeared normal grossly. Histologically both kidneys showed disorganised development with the presence of structures inappropriate for a foal of this age, including primitive glomeruli, immature renal tubules and persistent metanephric ducts. Based on these findings a diagnosis of bilateral renal dysplasia was made.

Introduction

Renal dysplasia is a collection of disorders in which the kidneys begin to form but then fail to differentiate into normal nephrons and collecting ducts (Woolf *et al.* 2004). Two main theories have been considered in its pathogenesis: a primary failure of ureteric bud activity and a disruption produced by fetal urinary outflow impairment (Gull *et al.* 2001; Woolf *et al.* 2004). In the horse, the kidneys are essentially mature at birth, and renal dysplasia is therefore usually the result of a congenital problem. In other species, such as the dog, a nephrogenic zone persists after birth, and dysplasia can thus be the result of disease occurring during the neonatal period. Although reported rarely, there have been a limited number of case reports of both renal dysplasia and hypoplasia in several breeds of horses (Andrews *et al.* 1986; Zicker *et al.* 1990; Jones *et al.* 1994; Ramirez *et al.* 1998; Gull *et al.* 2001); in most of these cases the affected animal presented with signs of renal failure and biochemical evidence of azotaemia.

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This Case Report describes the clinical and pathological findings in a neonatal foal with bilateral renal dysplasia from which *Enterobacter cloacae* was isolated *post mortem*. The foal did not demonstrate azotaemia, but had severe hyperkalaemia and hyperglycaemia.

Case details

History

A Selle Français filly was referred to the Bell Equine Veterinary Clinic with a primary complaint of being off suck, listless and depressed. The foal had been born at 322 days of gestation. Delivery had been observed and was assisted. Gross examination of the placenta revealed no abnormalities. Initially the foal had been slow to suck, and colostrum had been administered via nasogastric intubation. Following this the foal was bright, alert, responsive and suckling well. The foal was reported to have passed meconium and to urinate normally. Serum IgG levels were measured at 24 h of age, and were greater than 80 g/l.

The referring veterinary surgeon was called to examine the foal at 48 h of age. The filly had become progressively weak, recumbent and less responsive. Having not suckled for 8 h, the foal was referred to the hospital for further evaluation and treatment.

Clinical findings

Physical examination

On arrival the foal was recumbent and had no suck reflex. She was bradycardic (heart rate 56 beats/min; reference range [rr] 80–100 beats/min), and hypothermic (rectal temperature 36.6°C, rr 37.5–38.5°C), with cold extremities and weak peripheral pulses. Auscultation of the heart and lung fields was within normal limits. Audible borborygmi were present in all 4 quadrants of the abdomen. Mucous

membranes were pale with a prolonged capillary refill time of 2–3 s. The foal appeared dysmature with a domed head, silky coat and floppy ears. Bodyweight at admission was 50 kg.

Clinical pathology

Results of haematology revealed a marginally reduced packed cell volume (PCV) of 29.5% (rr 31–40%) with a total leucocyte count of $10.4 \times 10^9/l$ (rr $5\text{--}12 \times 10^9/l$), neutrophils $7.4 \times 10^9/l$ (rr $3.5\text{--}10 \times 10^9/l$), and lymphocytes $1.8 \times 10^9/l$ (rr $1.2\text{--}2.2 \times 10^9/l$). There was no left shift or toxic changes to neutrophils. Serum biochemistry revealed hypoalbuminaemia (serum albumin 23 g/l, rr 25–35 g/l), and normal values for creatinine of 122 $\mu\text{mol/l}$ (rr 106–380 $\mu\text{mol/l}$), and urea of 3.7 mmol/l (rr 3.5–4.0 mmol/l). Plasma fibrinogen was elevated (4.5 g/l, rr 2–4 g/l). Serum glucose concentration was markedly elevated at 22.6 mmol/l (rr 6.0–12.5 mmol/l). Electrolyte analysis revealed marked hyperkalaemia (plasma potassium 7.05 mmol/l; rr 3.5–5.5 mmol/l) with hyponatraemia (plasma sodium 107.7 mmol/l, rr 134–143 mmol/l) and hypocalcaemia (plasma ionised calcium 0.88 mmol/l, rr 2.5–4.0 mmol/l). Blood samples were collected for both aerobic and anaerobic culture, which subsequently yielded no bacterial growth.

Due to poor perfusion, attempts to obtain an arterial blood sample were unsuccessful. Venous blood gas analysis revealed an acidosis (pH 7.341, rr 7.37–7.98). Base excess and bicarbonate levels were within normal limits.

The foal was seen to pass a small volume of discoloured urine during physical examination. A urinary catheter was then placed and a urine sample obtained. This yielded 20 ml of brown-coloured urine. Dipstick analysis identified ++++ protein, +++ blood, ++++ glucose and + bilirubin. The urine had a specific gravity of 1.030 with a pH of 6.0.

Ultrasonography

Transcutaneous abdominal ultrasonography was within normal limits. A moderately filled bladder was identified, and no defects were identifiable in the bladder wall. No free fluid was visible within the abdomen. The small intestine was observed to contract fully and no distension was identified. Ultrasonography of the kidneys identified no obvious abnormalities in either their size or appearance.

Differential diagnosis

At this stage the most likely differentials were considered to include perinatal asphyxia syndrome (resulting in lactic acidosis and hyperkalaemia), acute renal failure (although there was no azotaemia), and ruptured bladder or ureter (although no free abdominal fluid was identified.) The elevated fibrinogen was considered to be suggestive

of an intrauterine or periparturient infection. The cause of the severe hyperglycaemia was not established, although possibilities of septicemia and diabetes mellitus were considered. The hypoalbuminaemia and proteinuria suggested the possibility of a nephritic syndrome, although proteinuria can be a normal finding with ingestion of a large volume of colostrum.

Treatment

In view of the clinical signs indicative of hypovolaemia, immediate i.v. fluid therapy was initiated (prior to the availability of the full clinical pathology results) with Hartmann's solution administered at twice maintenance rate (8 ml/kg bwt/h). Antibiotic therapy consisted of ceftiofur (5 mg/kg bwt i.m. q. 12 h). Oxygen was supplemented via a nasal tube at a flow rate of 200 ml/min and 200 ml of mare's milk was administered via nasogastric tube. The foal was maintained in sternal recumbency, under a heat lamp. Mild seizure activity was noted and controlled as necessary using diazepam (0.1 mg/kg bwt) i.v. boluses.

Vital parameters were measured every hour. Over the first hour of hospitalisation the heart rate continued to fall to 40 beats/min; however, after 4 h this had risen to 65 beats/min. A large volume of dark urine was passed voluntarily as was a small pile of dark, pasty diarrhoea. At this stage the plasma potassium concentration had reduced slightly but remained elevated at 6.85 mmol/l, and the sodium concentration had risen to 109 mmol/l. The serum glucose concentration remained elevated at 22.1 mmol/l. A constant rate insulin infusion was started (0.00125 u/kg bwt/h of regular insulin) in an attempt to reduce the hyperglycaemia and concurrently treat the hyperkalaemia. The i.v. fluids were changed to isotonic sodium chloride supplemented with calcium gluconate (200 mg/l) as soon as the results of blood analysis indicating hyperkalaemia, hyponatraemia and hypocalcaemia became available.

The foal became increasingly agitated and less responsive over the next 6–7 h. It became dyspnoeic and tachypnoeic, developing increased respiratory noise, with expiratory crackles audible on thoracic auscultation. Mucous membranes were now pale pink. The serum glucose concentration had reduced to 5.8 mmol/l, and the insulin infusion was therefore stopped. Haematology confirmed that the PCV had remained stable (29.2%). Ultrasonography of the abdomen and of the chest wall was performed and no abnormalities were detected. Plasma potassium concentration was now 6.32 mmol/l and sodium 117 mmol/l, with ionised calcium concentration remaining low at 0.70 mmol/l.

Outcome

The foal deteriorated acutely after 12 h of hospitalisation. It became unresponsive and started to exhibit

opisthotonus. Breathing became increasingly laboured. Due to the poor prognosis and financial constraints the foal was subjected to euthanasia.

Post mortem findings

Due to the owner's wishes only a limited *post mortem* examination was conducted. This revealed no gross abnormalities within the thoracic or abdominal cavities. The heart and lungs appeared normal, and there was no evidence of pulmonary oedema. The pancreas, liver and gastrointestinal tract appeared grossly normal, but no histological examinations were possible. No free fluid was found within the abdomen. The urinary tract appeared to be normally formed. The bladder was found to be flaccid and empty. The bladder was distended with 150 ml of tap water, and no leakage of fluid could be identified. The ureters and kidneys appeared normal grossly. Both kidneys were removed and retained for histopathology. A swab taken from the renal medulla of both kidneys yielded a heavy growth of *Escherichia coli* and *Enterobacter cloacae*.

Histopathology

Histologically both kidneys showed disorganised development with the presence of structures inappropriate for a foal of this age (Figs 1 and 2). There were primitive glomeruli, some undergoing cystic atrophy, in the cortical zone. Immature renal tubules were lined by cuboidal epithelium. Multiple persistent metanephric ducts, surrounded by primitive mesenchyme, present in the outer cortex and undifferentiated mesenchyme located between primitive collecting ducts were found in the medulla. Based on these findings a diagnosis of bilateral renal dysplasia was made.

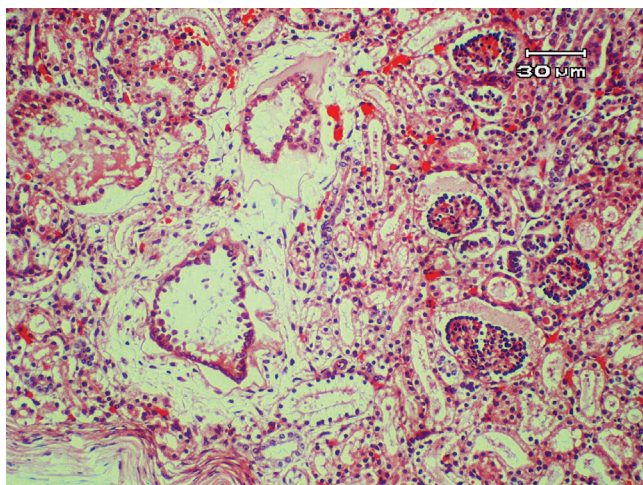


Fig 1: Photomicrograph showing outer cortex with primitive glomeruli, primitive tubules lined by cuboidal to columnar adenomatous type epithelium, persistent metanephric ducts encompassed by primitive mesenchyme.

Discussion

The kidneys are formed from 2 separate structures, the metanephric and the uteric buds (Miyazaki and Ichikawa 2003). Normal kidney development is dependent upon appropriate interaction between the 2. As the 2 structures come together, the ampulla of the uteric bud stimulates differentiation of the metanephric bud, which forms the normal renal parenchyma (Potter 1972; Gull *et al.* 2001; Woolf *et al.* 2004). Renal dysplasia occurs when these events do not occur properly. As a result hypoplastic kidneys containing immature glomeruli and tubules are formed. Persistent mesenchyme and adenomatous epithelium often remain. Affected kidneys are unable to function normally and signs of renal failure develop.

The cause of renal dysplasia is often unidentified. Ureteral obstruction during the development of the renal parenchyma had been suggested (Jones *et al.* 1994), with up to 90% of human cases being associated with a urinary tract obstruction (Taxy 1985; Andreoli 2004). This obstruction predisposes to bacterial pyelonephritis. As renal development is essentially complete by the time of parturition, it is likely that these insults occur *in utero*. The use of aminoglycosides and steroids in the gestational period has been associated with the development of renal dysplasia in man (Hulton and Kaplan 1995). A familial incidence has been identified in some canine breeds (Gull *et al.* 2001).

Although most cases of renal dysplasia in human babies are sporadic, a genetic origin and familial prevalence is recognised in some, many of which are associated with multi-organ syndromes. One such human syndrome is renal cysts and diabetes syndrome (RCAD) (Lindner *et al.* 1999; Kolatsi-Joannou *et al.* 2001). The foal in this Case Report presented with severe hyperglycaemia that required insulin therapy for management. It is interesting to speculate whether this hyperglycaemia may have been due to diabetes mellitus; however, no specific endocrinological

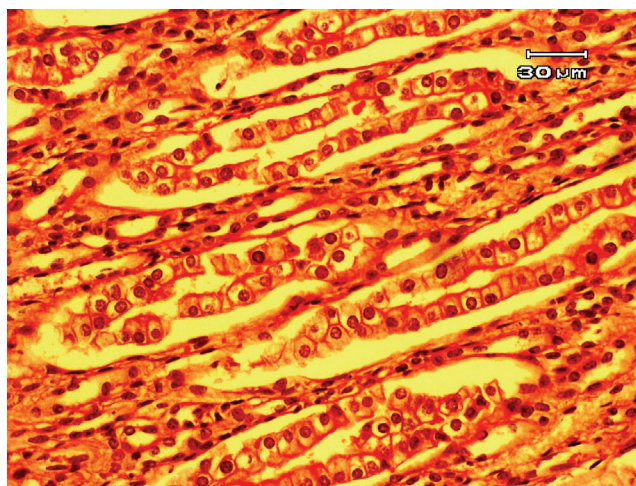


Fig 2: Photomicrograph of medulla showing undifferentiated mesenchyme and primitive collecting ducts.



evaluation was carried out. Hyperglycaemia is common in critically ill human patients, and has been reported in adult horses with colic (Hollis *et al.* 2007). However, most critically ill foals present with hypoglycaemia as a result of not nursing, because the glycogen stores at birth are sufficient only for approximately 2 h of energy requirements in the unfed foal and fat stores are also very low at birth (Corley and Axon 2005). Severe hyperglycaemia (blood glucose >10 mmol/l) has been reported in a small percentage (9.5%) of sick foals (Corley and Axon 2005). There are several potential reasons for the development of hyperglycaemia in critically ill patients, including altered glucose metabolism and insulin resistance, with an increase in gluconeogenesis, despite increased blood glucose and insulin concentrations (Langouche and van den Berghe 2006). Both hypo- and hyperglycaemia are harmful, and there is good evidence from human critical care that keeping serum glucose concentrations within a narrow range is associated with better outcomes (van den Berghe *et al.* 2003).

There are only a small number of case reports describing renal dysplasia in foals and mature horses, presenting with nonspecific signs of weight loss, depression and lethargy due to chronic renal failure (Andrews *et al.* 1986; Zicker *et al.* 1990; Jones *et al.* 1994; Ramirez *et al.* 1998; Gull *et al.* 2001). One case in a 3-month-old foal was diagnosed by the incidental finding of azotaemia (Ramirez *et al.* 1998). Ultrasonography revealed asymmetric kidneys, which were grossly small and misshapen. The kidneys were isoechoic in comparison to the spleen with a poor cortico-medullary demarcation. A renal biopsy confirmed the diagnosis. This case contrasts the described case in the present report as no azotaemia was present and the ultrasonographic appearance of the kidneys was considered to be normal. Gull *et al.* (2001) used contrast computed topography to diagnose renal hypoplasia and dysplasia in a 2-month-old American miniature foal. Changes were also identified in the skeletal muscle, which were consistent with nutritional myodegeneration. Andrews *et al.* (1986) described a one-day-old colt that had been found dead. *Post mortem* examination identified an absence of renal medullary tissue, a dilated renal pelvis, but minimal tubular changes. Unfortunately no clinical pathology values were available. Another case involving a 2-day-old Quarter Horse presenting with depression, diarrhoea and lethargy was described by Zicker *et al.* (1990). The foal in the latter report had increased fractional excretions of sodium and potassium, and was azotaemic. This foal also had grossly normal kidneys, as were identified in the present case report, suggesting that renal size is not related to severity of the renal failure. Renal dysplasia was diagnosed in a premature Trakhener foal at 4 months of age with concurrent ureteropolyps leading to hydronephrosis (Jones *et al.* 1994). The foal in the present case report showed no other developmental abnormalities or urinary tract outflow obstruction.

An elevation in creatinine and blood urea nitrogen has been extensively described as a reliable marker of renal

dysfunction, whether this be caused by pre-renal, renal or post renal disease. Although not specific for renal disease, particularly in neonatal foals, the absence of azotaemia in the present case was considered surprising, particularly in light of such severe electrolyte derangements. Renal failure is characterised by an increase in the blood concentration of creatinine and nitrogenous waste products, a decrease in glomerular filtration rate, and by the inability of the kidney to appropriately regulate fluid and electrolyte homeostasis. Although the fetal kidney produces urine at a relatively early stage of development, the placenta is primarily responsible for fluid and electrolyte homeostasis and for the excretion of nitrogenous waste products (Brewer 1990). Immediately after birth, the serum creatinine and urea concentrations are considered to be a reflection of placental function and maternal renal function, and cannot be used as a measure of renal function in the newborn (Vanpee *et al.* 1992; Gouyon and Guignard 2000). Although elevations in serum creatinine and urea will eventually occur in the neonate as a result of renal failure, these changes are usually delayed (Andreoli 2004), and were not present in this foal until its death at 60 h of age.

The kidney tightly regulates potassium balance and excretes approximately 90% of dietary potassium intake. Hyperkalaemia is a common and potentially life-threatening electrolyte abnormality in newborn human babies with acute renal failure (Rodriguez-Soriano 1995), and the severe hyperkalaemia that was present in this foal was considered to reflect renal dysfunction even though there was no azotaemia. As in this case, hyperkalaemia in neonatal foals is reported to occur most commonly in anuric renal failure (or ruptured bladder), but also occurs with massive tissue damage resulting from severe shock, hypoxia, asphyxia, or muscular diseases and hyperkalaemic periodic paralysis (Magdesian and Wikins 2008). The hyperkalaemia in this foal was accompanied by hyponatraemia, and attempts to correct these electrolyte abnormalities included the administration of i.v. sodium chloride and insulin infusions. Calcium supplementation was also provided in an attempt to correct the hypocalcaemia and to prevent cardiac arrhythmias occurring secondary to the hyperkalaemia. The precise cause of the hyponatraemia was not established, although the passage of pasty diarrhoea suggested the possibility of concurrent enteritis (unfortunately confirmation of this could not be established after death). Neurological dysfunction may be present in foals with low plasma sodium concentrations (Lakritz *et al.* 1992) and it is possible that some of the neurological signs exhibited by the foal may have been due to the severe hyponatraemia. Enteritis might also have contributed to the hypoalbuminaemia.

The isolation of *Escherichia coli* and *Enterobacter cloacae* from the kidneys in this case may have been significant, but in the absence of any inflammatory response in the renal tissue we consider that it was likely to be secondary to the underlying renal dysplasia. It is also



possible that this bacterial growth was a direct result of bladder catheterisation or *post mortem* contamination, although every attempt was made to minimise this possibility. Urinary tract infections (especially bacterial pyelonephritis) are common in human patients affected by renal dysplasia and other congenital anomalies of the urinary tract (Feldenberg and Siegel 1999; Hiraoka 2003), and these may result in further impairment of renal function post nately. However, the major pathophysiological derangement in babies with congenital anomalies of the kidney and urinary tract is a developmental one, and the main renal pathology is renal dysplasia (Woolf *et al.* 2004). *E. coli* and *E. cloacae* are both Gram-negative rod bacteria of the Enterobacteriaceae family. *E. cloacae* has been associated with urinary tract infections, septicaemia, pneumonia and intra-abdominal infections and is frequently isolated in nosocomial infections in human medicine (Cordero *et al.* 2004). Paterson (2006) recently reported an increasing incidence of antibiotic resistance of *E. cloacae*. This organism has been isolated *post mortem* from an adult horse at the authors' hospital that was subjected to euthanasia due to acute renal failure (unpublished observations). *Enterobacter* species, including *E. cloacae*, have also been isolated from blood cultures of a small proportion of neonatal foals with septicaemia (Marsh and Palmer 2001; Corley *et al.* 2007). The present case report supports the view that *E. cloacae* may be a significant pathogen in equine patients. The elevated fibrinogen suggests that this infection could have occurred *in utero* or around the time of birth, and is likely to be secondary to the developmental abnormalities of the kidneys.

The presence of hypoalbuminaemia and proteinuria in this foal are compatible with the presence of primary glomerular dysfunction and congenital nephrotic syndrome. In man, there are several diseases that are known to account for congenital nephrotic syndrome in infancy (Habib 1993; Salomon *et al.* 2000; Khoshnoodi and Tryggvason 2001). These include congenital nephrotic syndrome of Finnish type, diffuse mesangial sclerosis, idiopathic nephrotic syndrome and nephrotic syndrome related to infections during pregnancy. In addition, mitochondrial respiratory chain deficiency associated with congenital nephrotic syndrome and congenital renal disease has recently been identified (Goldenberg *et al.* 2005). The ubiquitous nature of the mitochondrial respiratory chain means that genetic defects of oxidative phosphorylation can account for a diverse range of human clinical problems including renal disease, muscle disease, neurological disturbances, deafness, retinopathy and diabetes mellitus (Goldenberg *et al.* 2005). Although no respiratory chain enzymological studies were undertaken in this foal, the possibility of a defect of oxidative phosphorylation cannot be excluded; such a defect might explain the diverse range of pathophysiological abnormalities demonstrated by the

case, including renal disease and hyperglycaemia. Unfortunately a more extensive *post mortem* examination was precluded at the owners' request. It would have been very interesting to examine the liver and pancreas histologically to rule out further abnormalities, particularly those that might have explained the hyperglycaemia.

In conclusion, renal dysplasia in foals is a rare congenital condition. It has been reported a few times over the past 3 decades. Clinical signs can vary from acute deterioration and collapse to vague lethargy and depression. Azotaemia and electrolyte abnormalities are useful diagnostic aids; however, neonates with acute renal failure due to renal dysplasia may develop electrolyte abnormalities in the absence of azotaemia. Ultrasonography can be a helpful imaging modality, but in some cases the kidneys may appear ultrasonographically normal. Ultimately the only reliable diagnostic technique is histopathology of renal tissue.

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A method of measuring the influence of rider interaction and training on the temporal relationships between limb cycles of horses using inertial motion sensors. *J.M. Walters et al. (UK)*

Seminars on 29 September

Activate the core muscles of horse and rider to improve performance and maintain health. *Christel Auer (Germany), Michael Baxter (Spain)*

Managing race and sport horses to keep them competing. *Philippe Benoit (France), Jeremy Naylor (UK)*

Knowledge on behaviour to better school and train horses. *Martine Hausberger (France), Rachel Murray (UK)*

Training sport horses. *Michael Baxter (Spain), Rachel Murray (UK)*

What about the equipment to compete horses – surcingle, sticking plaster, bridle, saddle etc. *Philippe Benoit, Lars Roepstorff (Sweden)*

Guiding exercise intensity through blood lactate and heart rate. *Arno Lindner (Germany), Pablo Trigo (Spain)*

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