Royal
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1   Abstract

2   Objective: To describe the prevalence of systemic hypertension in cats with community acquired acute

3   kidney injury (AKI) and investigate its relationship with disease severity.

4   Methods: Retrospective study of cats presenting to the Queen Mother Hospital for Animals, Royal

5   Veterinary College with AKI between 2007 and 2015. Systolic blood pressure (SBP) was measured

6   using Doppler sphygmomanometry and systemic hypertension was defined as a SBP ≥150mmHg.

7   Median SBP measurement (on admission and during hospitalisation),,IRIS (International Renal Interest

8   Society) grade of AKI, serum creatinine on admission, the presence of anuria or oliguria, length of

9   hospitalisation, survival to discharge and 6-month survival were all recorded.

10   Results: Forty-six cats were eligible for inclusion. The prevalence of systemic hypertension on

11   admission was 48.8% (21/43) and this was severe (≥180mmHg) in 18.6% (8/43) of cases. During the

12   whole hospitalisation period, systemic hypertension was detected in 27/46 (58.7%) cases and severe in

13   28.2% (13/46) cases. Systemic hypertension did not appear to be statistically associated with grade of

14   kidney injury, serum creatinine on presentation, oliguria or anuria.

15   Clinical significance: Systemic hypertension is common in cats with acute kidney injury but does not

16   appear to be associated with its severity.

19

Introduction

Acute kidney injury (AKI) is defined as an acute and abrupt decrease in kidney function resulting in abnormal glomerular filtration rate, tubular function and urine production (Hoste & Kellum 2007). AKI can be graded to encompass a continuum of functional and parenchymal damage from its least severe Grade I, a progressive increase in serum creatinine or documented oliguria over a six-hour period, to its most severe manifestation Grade V, in which renal replacement therapy may be indicated (IRIS 2013). Progressive increases in serum creatinine have been shown to be predictive of mortality in man, dogs and cats (Harison et al. 2012, Hoste et al. 2006, Uchino et al. 2010). For those patients that survive AKI, reversibility of kidney injury is variable and many patients ultimately develop chronic kidney disease (CKD; Basile 2001, Chertow et al. 2006).

Systemic hypertension (SH) is a potentially serious complication of AKI in dogs and cats (Acierno & Labato 2005, Monaghan et al. 2012). The International Renal Interest Society (2015) defines systolic blood pressure (SBP) in the following categories; patients with a SBP <150 mmHg are considered normotensive, SBP of 150 to 159 mmHg are considered borderline hypertensive with a low risk of target organ damage (TOD), patients with SBP ≥160 to 179 mmHg are considered hypertensive with moderate risk of TOD and patients with SBP ≥180 mmHg have severe hypertension with high risk of TOD (IRIS 2015). The most common pathological changes reported in hypertensive cats are hypertensive choroidopathy, which can result in blindness, and hypertensive cardiac hypertrophy, which can result in a cardiac murmur or gallop rhythm (Maggio et al. 2000, Chetboul et al. 2003). A less common but severe complication of SH is hypertensive encephalopathy resulting in obtundation and seizures (Chetboul et al. 2003, Kletzmayr et al. 2003). The kidney itself has been reported to be a target organ for hypertensive injury and hypertension has been associated with progression of kidney disease in humans and dogs (Klag et al. 1996, Jacob et al. 2003, Ravera et al. 2006).

SH has been documented in cats that have CKD (Syme et al. 2002) and has been described in dogs with AKI (Geigy et al. 2011). However, the prevalence of SH in feline AKI patients is currently unknown. The aims of this retrospective study were to describe the prevalence of SH in cats with AKI and to describe its relationship with disease severity and outcome.

49   Materials and Methods

50   The study was approved by Royal Veterinary College Clinical Research and Ethical Review Board.

51   The study population comprised of client-owned cats diagnosed with community-acquired azotaemic

52   AKI referred to the Queen Mother Hospital for Animals, Royal Veterinary College between 2007 and

53   2015. These patients were identified through a search of the hospital's computerised clinical record

54   system. Patients were eligible for inclusion if they fulfilled the International Renal Interest Society

55   (IRIS) guidelines for diagnosis of Grade II AKI and above, i.e. an initial serum creatinine level

56   ≥ 141 μmol/L and one or more of the following criteria: evidence of renal tubular injury on urinalysis

57   (renal glucosuria, proteinuria with an inactive sediment, or renal casts), diagnostic imaging findings

58   suggestive of AKI, or persistent urine output of less than 1 mL/kg/hour) (IRIS 2013).

59   The files of eligible patients were reviewed and the signalment, cause of AKI, medical history

60   (including prior medications), physical examination findings (including fundic examination), SBP

61   measurements, patient-side electrolyte, metabolite and blood gas analysis, complete blood count,

62   biochemistry, abdominal ultrasound, urinalysis, urine output measurements, length of hospitalisation

63   and survival to discharge were recorded when available. The use of anti-hypertensive therapy was also

64   documented. SBP was measured indirectly using Doppler sphygmomanometry. The standard hospital

65   protocol for SBP measurements, adapted from the American College of Veterinary Internal Medicine

66   guidelines (Brown et al. 2007), is to record the mean of three to five consecutive readings.

67   SH was defined as a SBP ≥ 150 mmHg including cats identified as borderline hypertensive (SBP 150 to

68   159 mmHg), hypertensive (160 to 179 mmHg) and severely hypertensive (≥ 180 mmHg) according to

69   the IRIS guidelines (IRIS 2015). Patients were included if there were two or more SBP measurements

70   within the clinical records during the period of hospitalisation. When there was more than one SBP

71   evaluation in the clinical record on a single day of hospitalisation, a median SBP measurement for that

72   day of hospitalisation was used for analysis. The AKI grade was determined for each patient on

73   admission. Patients were grouped as being oliguric or anuric based on whether furosemide was

74   administered or they underwent continuous renal replacement therapy. Patient records were analysed

76   and follow-up phone calls to primary care practices were performed to identify whether those patients

77   that survived to discharge were still alive at six months.

78   Patients were excluded if they had any history, clinicopathological data or diagnostic imaging findings

79   suggestive of CKD including a longer history of polyuria or polydipsia, poor body condition, non-

80   regenerative anaemia or imaging evidence compatible with chronic nephropathy (small irregular

81   kidneys and loss of corticomedullary definition). Patients were also excluded if they had concurrent

82   diseases that could be associated with SH (including hyperthyroidism and hyperaldosteronism) or if

83   they had a history of pretreatment with antihypertensive drugs such as angiotensin-converting enzyme

84   inhibitors, beta-blockers or calcium channel blockers.

85   Statistical analysis

86   Data were assessed for normality using a Shapiro–Wilk W test and visual inspection of histograms.

87   Normally distributed data were expressed as mean and standard deviation and non-normally distributed

88   data expressed as median and ranges. Statistical analysis was performed using an online statistical

89   calculator (http://www.socscistatistics.com/). Fisher's exact test was used to compare categorical data,

90   one-way analysis of variance for ordinal data and Pearson's correlation coefficient to assess for any

91   correlation between SBP and serum creatinine. The level of statistical significance was set at $P<0\cdot05$.

92   Results

93   One hundred and twenty cats were presented with AKI during the investigation period. Of these, 74

94   were excluded: 44 had incomplete data, 29 had evidence of CKD and 1 had a previous diagnosis of

95   hyperthyroidism. Forty-six cats were eligible for inclusion in the study, of which 44 had multiple blood

96   pressure measurements on a single day and required calculation of a median blood pressure. Of these

97   46 cats, 13 were pedigree breeds, 27 domestic short hairs and 5 domestic long hairs and for in one the

98   breed was not identified. Twenty-four were female neutered, 21 were male neutered and 1 cat was male

99   entire. Ages ranged from 5 months to 13 years with a median of two years. All cats had received fluid

100   therapy before presentation. Aetiology of AKI was determined in 38 cases: non-steroidal anti-

101   inflammatory drugs (12/46), ureteroliths (9/46), trauma (6/46), ethylene glycol (5/46), pyelonephritis

103 (3/46), lily ingestion (2/46) and neoplasia (1/46). In 8 of 46 cases the cause was unknown. Of the cases

104 with ureterolithiasis, all were considered to have bilateral ureteral obstruction on review of

105 ultrasonographic imaging. None of the cats reported to have ureteral obstruction in this study had

106 clinical, historical or ultrasound findings specifically supporting chronic disease before the episode of

107 AKI.

108 Of the 46 cats, 43 had SBP measurements on the first day of hospitalisation. The prevalence of SH

109 (SBP $\geq$150 mmHg) on day 1 of hospitalisation was 21 (48.8%) and in 8 (18.6%) cases this was severe

110 ($\geq$180 mmHg). Four cats normotensive on admission developed hypertension in hospital and did so

111 within the first 48 hours. Out of the three cats that did not have a blood pressure measurement on the

112 first day, two were reported to be hypertensive on their first measurement. The prevalence of SH over

113 the whole hospitalisation period was 58·7% (n=27), which was classified as severe in 28·2% (n=13) of

114 cases. The mean SBP of cats on day 1 was 146·6 ±28·25 mmHg and the mean SBP over the whole

115 hospitalisation period, excluding cats that had been started on antihypertensive therapy), was 145·25

116 ±29·11 mmHg.

117 Three cats were started on amlodipine besylate at a dose of 0·625 mg once daily. Two cats with an

118 initial SBP over 180 mmHg were started on amlodipine on day 1 and one cat was started on amlodipine

119 on day 2 of hospitalisation because of evidence of hypertensive retinopathy accompanying a SBP of

120 160 mmHg. Of these three patients one was euthanased, one became borderline hypertensive and the

121 other remained persistently hypertensive at follow-up.

122 The mean SBP of cats on day 1 was 146.6mmHg+/-28.25 and the mean SBP over the whole

123 hospitalisation period, excluding cats that had been started on anti-hypertensive therapy), was

124 145.25mmHg+/- 29.11.

125 On presentation the mean serum creatinine was 1034 ±526·99 μmol/L. Initial SBP and creatinine value

126 on presentation were not correlated (R^2 = 0.069, P = 0.078). Twenty-six cats were classified as having

127 Grade V AKI, 16 classified as Grade IV and 2 each classified as Grades III and II AKI. There was no

128 association between SH on presentation, IRIS AKI grade and survival (Table 1). When assessing the

130 prevalence of SH over the whole of the hospitalisation period there was not a statistically significant

131 association between SH during hospitalisation, and IRIS AKI grade (Table 2).

132 Sixteen cats were administered furosemide, two cats had peritoneal dialysis and one cat underwent

133 continuous renal replacement therapy. There was no statistically significant association between the

134 presence of SH and the presence of oliguria or anuria (p=0.54).

135 Twenty-five cats were euthanased during hospitalisation of which two were borderline hypertensive,

136 five hypertensive and seven were severely hypertensive during hospitalisation. Twenty-one cats

137 survived to discharge of which three were classified as borderline hypertensive, four as hypertensive

138 and six as severely hypertensive during hospitalisation. Of these, one cat was discharged to be

139 euthanased at home and three cats were euthanased within the following six months with the remaining

140 cats still being alive after six months. Those cats that survived had a median hospitalisation length of

141 seven days, ranging from 1 to 18 days.

142 Of the eight patients classified as severely hypertensive on presentation, three survived and five died.

143 Of those cats that survived, one was persistently hypertensive at re-examination despite treatment with

144 amlodipine, one became non-hypertensive during hospitalisation and one had incomplete records. Of

145 those cats that died, two became borderline hypertensive on day 2 of hospitalisation, one of which was

146 given amlodipine. The other three cats were euthanased between one and four days after admission and

147 no follow-up SBP was recorded. Five cats developed severe SH during hospitalisation of which two

148 were normotensive on presentation. Of these five cats, two died and three survived. Out of the three

149 cats that survived two became less severely hypertensive, of which one became normotensive. The

150 other cat had incomplete records. The other two cats were euthanased within 12 to 48 hours of

151 developing severe hypertension.

152 Discussion

153 This retrospective study shows that SH, including cats with borderline hypertension, is common in cats

154 with AKI with a prevalence of 58·7% over the course of hospitalisation; in 28·2% of cases the

155 hypertension was classified as severe. SH in cats has most extensively been described in patients with

157   CKD where studies report a prevalence varying between 19.8% and 65% (Syme et al. 2002, Stiles et al.

158   1994) depending on criteria used to define hypertension. with a prevalence varying between 19·8 and

159   65% (Stiles et al. 1994, Syme et al. 2002) depending on criteria used to define hypertension. Previous

160   studies of feline AKI have attempted to assess prognosis and severity indices (Lee et al. 2012, Segev et

161   al. 2013) but have not examined the prevalence of SH in detail. Segev et al. (2013) reported a

162   prevalence of SH (defined as SBP >150 mmHg) of 38%, but included cats with "acute-on-chronic"

163   kidney disease. Furthermore, the frequency and technique for SBP measurement was not detailed,

164   therefore direct comparison with the current study is not possible.

165   In our study 54·3% of cats were euthanased in hospital, of which 56% were classified as either

166   borderline or severely hypertensive. SBP did not appear to be correlated with IRIS AKI grade or serum

167   creatinine at presentation. This suggests that SH in cats with AKI is not necessarily related to severity

168   of azotaemia and AKI grade. These findings are in line with the studies of CKD in cats in which

169   hypertension has not been found to be associated with survival or a progressive phenotype of CKD

170   (Syme et al. 2002, Jepson et al. 2007, Bijsmans et al. 2015). In contrast, in humans and dogs,

171   hypertension has been associated with progression of renal disease (Klag et al. 1996, Wehner et al.

172   2008).

173   Due to the small sample size and the various confounding factors of outcome including the variable

174   prognosis of the different causes of AKI, owner finances and other co-morbidities, the association

175   between SH and outcome could not be reliably assessed. Failure to see evidence of an association

176   between SH and severity of AKI in this study may have been due to using creatinine as the sole

177   indicator of kidney function [recognised to be problematic for multiple reasons including variation in

178   hydration status and body condition between patients (Braun et al. 2003)] or the result of a type II

179   statistical error.

180   Although hypertension is common in kidney disease its pathogenesis is poorly understood. Suggested

181   aetiologies in humans have included volume-impaired excretion of sodium, leading to volume

182   overload, excessive activation of the renin-angiotensin-aldosterone system, stimulation of the

183   sympathetic nervous system, reduced bioavailability of the endothelial vasodilator, nitric oxide and

185 increased production of the vasoconstrictor endothelin and increase in reactive oxygen species

186 (Campese et al. 2006, Pouchelon et al. 2015). To date there have been no studies of pathogenesis of SH

187 in AKI in cats and even in CKD the pathogenesis remains poorly explained. In this study there was no

188 association between anuria and SH. As these are the patients that are most susceptible to volume

189 overload it could be hypothesised that volume overload was not an important cause of hypertension in

190 this population of cats. However, improved data in terms of clinical signs relating to volume overload

191 such as baseline body weights and body weight changes and urine output as well as an assessment of

192 volume status (e.g. utilising bioimpedence) would be required to better evaluate this hypothesis.

193 Adverse effects on the ophthalmic, cardiovascular and central nervous systems has been reported in

194 cats with hypertension (Jepson 2011). In this study, one patient had signs consistent with hypertensive

195 retinopathy–choroidopathy. However, this was the only patient that had written documentation of a

196 fundic examination having been performed and so the true prevalence of ocular manifestations of SH

197 in these AKI patients cannot be reported with confidence. Nevertheless, ophthalmic examination

198 should be recommended for patients with AKI if there is suspicion of SH and future studies should

199 include evaluation for hypertensive ocular damage.

200 There are a number of limitations to this study. First, the strict inclusion criteria, particularly the

201 exclusion of those patients with acute on CKD reduced the sample size and overall power of the study.

202 Important records such as fundic examination findings, baseline body weights, daily weight changes

203 and accurate urine output measurements were lacking in some cases, preventing further assessment of

204 fluid overload and its effect on development of SH in the hospital. Urine output measurement is

205 required for early IRIS AKI grading and to accurately diagnose a patient as anuric or oliguric and has

206 been shown to be a prognostic factor in AKI in humans (Behrend & Miller 1996). This study did not

207 enrol patients with IRIS AKI Grade I, and only captured two patients with IRIS AKI Grade II reducing

208 the data distribution and therefore making it possible that the lack of association between IRIS grade

209 and SH results from type II statistical error. The IRIS grading system itself is a useful guide for

210 determining severity of disease but it has not yet been validated for use in AKI.

211

213 In the current study, we attempted to exclude those patients with acute-on-chronic disease based on

214 recorded historical, physical examination and ultrasound findings. Patients with ureteroliths, without

215 documented historical, physical examination and ultrasonographic findings consistent with CKD were

216 included. However, due to the pathophysiology of obstructive disease and retrospective nature of the

217 study we cannot exclude the -possibility that these patients did not have underlying subclinical CKD

218 and therefore inadvertently included some acute-on-chronic causes of AKI.

219 Of particular concern in this study, is the "white coat effect," especially in those patients with fewer

220 blood pressure readings in their clinical records. White-coat hypertension, an increase in blood pressure

221 as a result of adrenergic stimulation during situations of stress, anxiety or excitement, has been

222 documented in healthy cats during simulated visits to a veterinary clinic (Belew et al. 1999). In an

223 attempt to minimise this effect on the findings of this study, when multiple blood pressure recordings

224 were recorded on a single day of hospitalisation a median SBP reading for that day was used for the

225 analysis. However, in some patients only a single blood pressure reading was performed on any

226 particular day and these patients therefore carry greater risk of inappropriate classification. Due to the

227 retrospective nature of the study there was limited information on the circumstances surrounding blood

228 pressure measurement and the clinician's perspective on the accuracy of the single blood pressure

229 readings. Assessment for hypertensive retinopathy–choroidopathy in all patients would have been

230 useful to assess for evidence of TOD as part of exclusion criteria for white-coat hypertension and,

231 ideally, continuous blood pressure monitoring would be required to completely mitigate the white-coat

232 effect.

233 In summary, this study suggests SH is common in cats with AKI. However, it does not appear to be

234 associated with disease severity or magnitude of azotaemia. This study should be considered a

235 preliminary study for future prospective studies assessing hypertension in AKI, its persistence during

236 hospitalisation and the effect of antihypertensive treatment on outcome.

237

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240 Conflict of interest

241 None of the authors of this article has a personal or financial relationship with other persons or

242 organisations that could inappropriately influence or bias the content of the paper.

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324

326 Table 1: Classification of systemic hypertension at presentation and its association with Acute
327 Kidney Injury grade and survival

SBP at presentation (mmHg)	Total percentage (%)	Number of cats				Survival to discharge
		IRIS AKI grade				
		II	III	IV	V	
150 to 159	6/21 (28.57%)	0	0	2	4	2/6 (33.33%)
160 to 179	7/21 (33.33%)	0	0	5	2	3/7 (42.85%)
≥180	8/21 (38.09%)	0	2	1	5	3/8 (37.5%)

328 SBP: Systolic blood pressure; IRIS: International Renal Interest Society AKI: Acute Kidney Injury

329 Table 2: Association between IRIS AKI grade and presence of hypertension (defined as
330 systolic blood pressure ≥ 150mmHg) during hospitalisation.

IRIS AKI grade	Number of patients	Mean SBP (mmHg)
II	2	127 ±9·89
III	2	193·75 ±8·84
IV	16	148·28 ±23·92
V	26	158·48 ±28·28

331 n= 46, p= 0.056

332 IRIS: International Renal Interest Society; AKI: Acute Kidney Injury; SBP: Systolic blood pressure