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Atypical myopathy in grazing horses: A first exploratory data analysis

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Abstract

Over the last decade, atypical myopathy (AM) in grazing horses has emerged in several European countries. An exploratory analysis was conducted to determine horse- and pasture-level indicators or factors associated with AM in Belgium. Belgian cases of AM confirmed by histology ($n = 57$) were compared to their healthy co-grazing horses ($n = 77$) and to pastured horses not involved with AM as controls ($n = 386$). The pastures where confirmed cases were grazing (42 pastures; 38 sites; 44 incidences of AM) were compared with those of the controls (216 pastures; 96 sites; no incidence of AM).

Statistically significant ($P \leqslant 0.05$) exploratory variables, identified by means of adjusted odds ratios, suggested that indicators or factors associated with individual horses (young age, inactivity, body condition poor to normal), management practices (permanent pasturing, spreading of manure) and pasture characteristics (humid, sloping pastures, accumulated dead leaves, presence of waterway) may increase the risk of AM. Specific interventions based on these factors might help to reduce the incidence of AM.

Keywords: Pasture; Epidemiology; Rhabdomyolysis; Management; Prevention; Atypical myopathy

Introduction

Atypical myopathy (AM) is an acute rhabdomyolysis that appears in an epizootic or sporadic form in grazing horses. Since the disease was recognised in 1984 in Scotland (Anonymous, 1985), several outbreaks of AM have been reported in the United Kingdom (Hosie et al., 1986; Whitwell et al., 1988; Harris and Whitwell, 1990; Hillam, 1991; Robinson, 1991), and over the last 10 years it has emerged in other European countries where the syndrome had not been diagnosed previously (Brandt et al., 1997; Delguste et al., 2002; Moussu et al., 2003; Votion et al., 2003). Personal communications from European veterinary practitioners and clinicians at various Veterinary Colleges via the Atypical Myopathy Alert Group[1] (AMAG; a network initiated by the authors of this study), has indicated that AM was encountered during the autumn of 2004 in the following European countries: Austria, Belgium, England, France, Germany, Italy, Luxembourg, Scotland, The Netherlands and Switzerland. There may also be a seasonal pasture myopathy in the United States similar to AM as seen among horses in Europe (Finno et al., 2006).

Since the first recognised Belgian outbreak in November 2000 (Delguste et al., 2002), 196 clinical cases, all highly

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compatible with a diagnosis of AM, have been reported to the University of Liège. The mortality rate was around 85% (95% confidence interval [CI]: 79–90%) and 57 horses were confirmed with AM on the basis of histopathology performed on samples taken at necropsy (Cassart et al., 2007; Votion et al., 2007). Information on these confirmed cases (CC) and their co-grazing Equidae (Co-G) was collected and analysed to define the history and clinical features of AM (Votion et al., 2007).

The results of this retrospective case series study suggests that an association of AM with specific individual characteristics and environmental conditions. In particular, it indicated that young horses in poor or normal condition may be at more risk of AM than overweight horses. Analysis of natural drainage and hydromorphic characteristics of the pastures where the CC were gazing revealed that most of the pastures were sparse and contained humid areas or had pedological characteristics that favoured humidity. In addition, specific weather conditions (especially wind) seem to predispose to the onset of AM.

AM is usually fatal with no specific treatment or prophylaxis available. The identification of factors associated with the disease, and interventions based on these factors, currently offer the only hope of disease prevention. However, the apparent association of AM with a specific equine population and/or different types of pasture must be interpreted in the light of our knowledge of the equine population and the pasture types commonly found in those regions where AM is encountered.

In Belgium, little is known about the demographic characteristics of the Equidae, mainly because systematic identification of horses only started in January 2006. Similarly, there are currently no data available about horses at pasture and management practices in the country. Yet knowledge of pasture characteristics is probably of paramount importance since repetition of AM cases on certain premises suggests that horses on certain pastures might be particularly at risk of contracting AM (Votion et al., 2003).

This first exploratory data analysis was conducted to determine horse- and pasture-level indicators or factors associated with AM in Belgium. The CC were compared to their Co-G as well as to control horses (CT). The CT were matched with CC and Co-G for the region where AM predominantly broke out. The characteristics of the pastures where CC (and Co-G) were grazing at the time of AM onset (P_{CC}) were compared to the ones where the CT pastured (P_{CT}).

Materials and methods

Case and non-case selection

Our data on confirmed cases comprised those horses whose history and clinical signs suggestive of AM had been communicated to the AMAG after the first recognised Belgian outbreak up to the end of May 2005. Clinical signs included the sudden onset of indicators of acute rhabdomyolysis (e.g. myoglobinuria, stiffness, trembling, sweating), weakness, recumbency and depression, with rapid aggravation of signs or even unexpected death (Votion et al., 2007).

The diagnosis of AM was confirmed histologically by the observation of severe fibre degeneration in postural (e.g. subscapularis) and respiratory (e.g. intercostal) muscle samples taken post mortem (Cassart et al., 2007). The equine co-grazers of CC that remained free of any abnormal clinical signs constituted the group Co-G. Despite the lack of signs, this group included at least five presupposed sub-clinical cases of AM (i.e. among 38 Co-G sampled for creatine kinase; five had enzyme activity >2000 U/L; Votion et al., 2007).

Owners of horses of the CT group were located by contacting practitioners following a random search of a list of equine veterinarians working in the Wallon region where the majority of CCs were recorded. To be recruited into the CT group, horses needed over the year to have had permanent or temporary access to pasture and not to have been managed in equestrian centres (where the condition has not been reported; Votion et al., 2007). Access to pasture was a sine qua non, as AM has only been described in grazing horses (Votion et al., 2004).

Data collection

The group of CC included 57 Equidae, which were pasturing with 77 Co-G on a total of 42 P_{CC} representing 38 sites and 44 incidences. Questionnaires and in-depth interviews of the owners of CC and Co-G enabled us to acquire data regarding animals, management practices, health status and characteristics of the pastures. Twenty-five premises from the 42 P_{CC} were visited by two authors of this study.

A standardised epidemiological inquiry (Table 1) was developed, and included questions on herd characteristics, health and disease prevention, housing and pasturing conditions, management systems and feeding practices. Data concerning the characteristics of the premises were also collected. Parameters that had to be defined subjectively by the owners of horses (i.e. temperament category and body condition as well as the quality of the grassland, the slope and the humidity of the pastures), required the owner's appraisal to fit into predefined categories (see tables for details).

Before its distribution to CT owners, the questionnaire was tested with 20 owners of horses to detect and correct any ambiguous or unclear questions. The questionnaire currently used for the recording of new AM cases by AMAG is currently available via the website[2] (Votion, 2004) and provides details of the questions asked in the inquiry. Four hundred and 75 questionnaires were distributed and 102 were returned (22%). Six were rejected for one of the following reasons: (1) a case of AM was declared after receiving the questionnaire ($n = 1$); (2) a horse died from a myopathic syndrome with no conclusive diagnosis ($n = 1$), or (3) the questionnaires were negligently completed ($n = 4$). Of the 96 valid questionnaires, 386 horses and 216 pastures located on 96 sites were included in the CT and P_{CT} categories, respectively.

Statistical analysis

The means of the quantitative parameters in each group with unequal variance were compared using Welch's test (Dagnelie, 1998). An exploratory study was conducted to begin characterising the prevalence and indicators or risk factors for AM. The trend for a parameter toward risk and preventive factors was evaluated by mean of odds ratios (OR) as defined by Grenier (1990). Because of the confounding factors, adjusted OR on age were calculated for sex, vaccination, deworming and use at the horse level, according to the Mantel–Haenszel procedure (Dohoo et al., 2003). The limit of statistical significance of the conducted tests was defined as $P \leqslant 0.05$.

[2] See: http://www.myopathieatypique.be/.

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Table 1
Surveyed factors that were included in the inquiry to determine the risk factors associated with atypical myopathy (AM) in grazing horses

Subject	Factors
Herd characteristics	– Type and number of animals with access to pastures – Demographic data (e.g. age, sex, type, breed) – Use of the horse (e.g. riding, sport, breeding)
Health and disease prevention	– Health status and previous illness – Deworming and vaccination schemes
Pastures type/history	– Size of pasture – Quality of pasture – Nature of pasture – Accommodation (including water access) – Environmental characteristics (e.g. presence of trees, slope of pasture, humidity) – Previous deaths on the pasture (horses and other species)
Management of pastures	– Number of animals per pasture – Manure removal, use of fertilizers – Mixed species grazing – Seasonal pasturing
Feeding practice	– Seasonal distribution frequency – Type of nutrients fed (e.g. hay, silage, complete-mix) over the four seasons – Characteristics of feeds (e.g. locally produced or imported hay, purchased or home-mixed feed)

Results

Quantitative data at horse and pasture level

The frequency of horse distribution within age categories for each group is shown in Fig. 1. The age of CC was significantly lower than the age of Co-G (Welch's test; $df = 102$; $P = 0.0075$) both being lower than the age of CT (Welch's test; $df = 242$; $P < 0.0001$ and Welch's test; $df = 123$; $P < 0.0001$, respectively) (Table 2). The lowest frequency of deworming was found in CT and was significantly less than in CC (Welch's test; $df = 400$; $P = 0.0068$) and in Co-G (Welch's test; $df = 406$; $P = 0.0001$). The frequency of deworming in Co-G was higher than in CC (Welch's test; $df = 89$; $P < 0.0001$).

Qualitative data at horse and pasture level

Demographic and health data

OR (Table 3) and adjusted OR on age (Table 4) between groups were calculated at horse and pasture level. At first

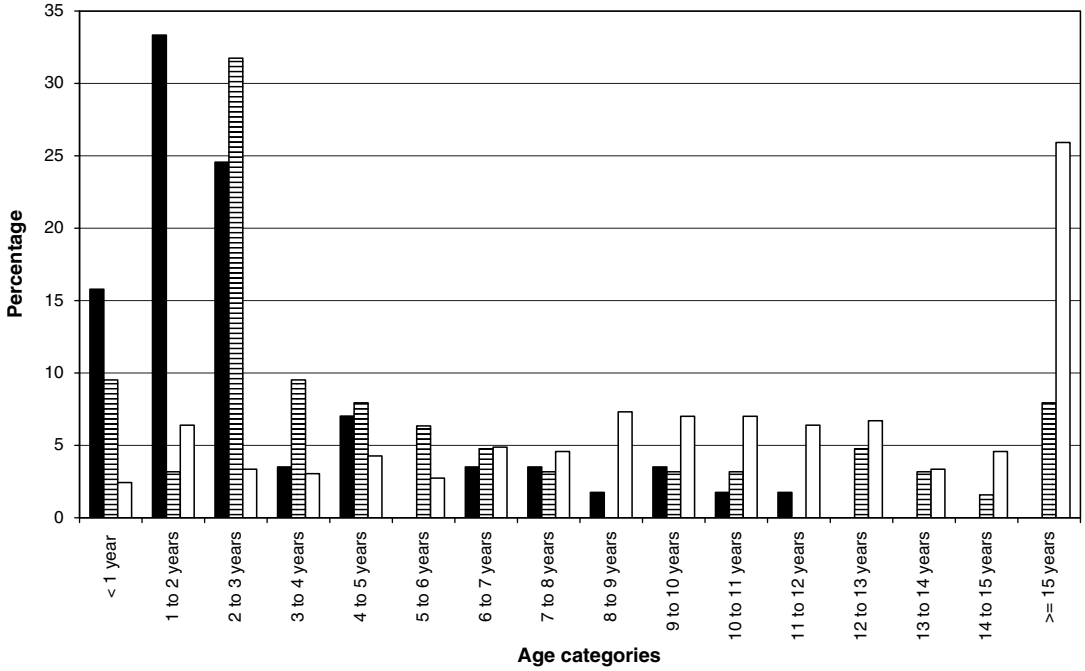

Fig. 1. Frequency distribution within age categories for confirmed cases (■; $n = 57$) of atypical myopathy, co-grazing Equidae (▤; $n = 63$), and control horses (□; $n = 328$).

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Table 2
Quantitative data for AM at the horse and pasture level

Variables	Group[A]	Number	Mean	SE
Age (years)	CC	57	2.81	2.60
	Co-G	63	5.78[a]	5.86
	CT	328	11.13[c,e]	7.29
Frequency of deworming (year^{-1})	CC	26	3.00	0.05
	Co-G	55	3.07[c]	0.07
	CT	376	2.88[b,d]	0.93
Total number of Equidae on premises (Equidae)	P_{CC}	38	3.50	1.94
	P_{CT}	207	3.27	2.30
Size of the pasture (hectare)	P_{CC}	34	0.99	0.78
	P_{CT}	167	1.33	2.24
Live livestock unit per hectare	P_{CC}	31	5.14	4.72
Live livestock unit per hectare	P_{CT}	155	5.09	7.09

[A] SE, standard error of the mean; CC, histologically confirmed cases of atypical myopathy; Co-G, clinically healthy co-grazing Equidae; CT, control horses; P_{CC}, pasture where CC and Co-G were grazing; P_{CT}, pastures where CT were grazing.
[a] Significantly higher than CC (Welch's test; $P \leqslant 0.01$).
[b] Significantly less than CC (Welch's test; $P \leqslant 0.0001$).
[c] Significantly higher than CC (Welch's test; $P \leqslant 0.0001$).
[d] Significantly less than Co-G (Welch's test; $P = 0.0001$).
[e] Significantly higher than Co-G (Welch's test; $P \leqslant 0.0001$).

analysis, it was found that the risk of being affected with AM was significantly increased in entires and decreased in geldings. However, the age was found to be a confounding factor for the sex, and the additional analysis performed demonstrated that there was no sex predisposition for AM.

Animals in poor bodily condition or with a normal weight (as defined by the owners) were more at risk than the ones that were overweight, which were at reduced risk. The analysis identified a history of previous illness as a protective factor. Regular vaccination and deworming reduced the risk of AM whatever the age. Inactive horses were particularly at risk whereas working horses present a reduced risk of AM. The risk of AM was exacerbated in the Province of Liège.

Management practices data

OR between groups were calculated at pasture and horse level (Tables 5 and 6, respectively). Pastures with a history of previous death of horse(s) (regardless of the suspected cause) were at risk of AM. Furnished grassland in winter was a factor of risk for AM. Sloping (in particular pastures with a steep slope), and/or humid pastures, and/or pastures surrounded by or containing a stream/river were of particularly risk for AM. The risk was also significantly increased when manure was spread on the premises. Access to dead leaves piled up in autumn increased the risk of AM, but the presence of trees in the pasture or surrounding was not a risk factor. Providing a salt block was found to be a protective factor against AM. Giving water from the distribution network and providing complementary feeds all the year round were also found to be protective measures.

Full time pasturing over all seasons was a management practice that increased the risk of AM. This risk declined when the owners of horses took into account the climatic conditions to give pasture access to their horses during the risky periods (i.e. autumn and spring, the only seasons where cases of AM were confirmed).

Feeding practices data

OR between groups were calculated at horse and pasture level (Table 7). Giving supplementary feeds throughout all seasons significantly decreased the risk of AM. Hay supplementation during the autumn was a risk factor.

Discussion

Presupposed sub-clinical cases of AM have been reported including in the group of Co-G included in this study (Delguste et al., 2002; Votion et al., 2007). We cannot exclude the possibility that the CT category also contained Equidae with inapparent AM. This might have obscured true differences in OR for the three groups of horses, and observed differences in OR may reflect larger actual differences. However, this case control study indicated that statistically significant exploratory variables for AM were associated with individual and pasture characteristics as well as with their management.

Our report is an exploratory analysis of data compiled from all Belgian CC recorded over 5 years. The information is combined with demographic and pastures characteristics of Co-G and CT horses. The analysis is limited by the small number of CC and Co-G which may not be extended for the period considered. In addition, there are no available statistics about the equine population in Belgium that would have been of great help in validating our data of the CT group. Another problem resulted from the difficulty in linking a specific individual horse to a specific pasture in the CT group (indeed, a single horse may pasture on several premises during the year). Furthermore, pasturing of CT on a specific pasture should have been linked to a referential 'time' for AM. All of these factors have impeded a valid and solid statistical inference regarding risk factors. Nevertheless, the analysis has highlighted several parameters associated with AM, and these may lead to a better understanding of the conditions necessary for AM to occur. It is hoped that the identified exploratory variables discussed below will help to prevent the condition.

The results have demonstrated that young horses, especially those <3 years of age (mode: 18 months), were the primary targets of the disease. This age predisposition may reflect an increased exposure of young horses to the aetiological agent (e.g. food-plasticity) or a dynamic relationship between exposure to the agent and age-related resistance to the disease (e.g. immunity). Foals <4 months old were not affected by AM and, as younger foals are still

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Table 3
Odds ratios according to Grenier (1990) calculated for different demographic data and health data at the horse and pasture level for AM

Parameter	Number										Odds ratio (95% CI)			
	Animals						Pastures				CC vs. Co-G	CC vs. CT	Co-G vs. CT	P_{CC} vs. P_{CT}
	CC[a]		Co-G[b]		CT[c]		P_{CC}[d]		P_{CT}[e]					
	n[f]	N[g]	n	N	n	N	n	N	n	N				
Province of origin														
Hainaut	6	55	9	77	65	386	6	42	29	216	0.94 (0.32–2.73)	0.64 (0.27–1.52)	0.68 (0.32–1.41)	1.13 (0.45–2.84)
Liège	25	55	21	77	139	386	14	42	70	216	2.19 (1.06–4.52) +	1.48 (0.84–2.61)	0.67 (0.39–1.15)	1.05 (0.52–2.11)
Luxembourg	14	55	14	77	98	386	12	42	59	216	1.53 (0.66–3.49)	1.03 (0.54–1.96)	0.67 (0.36–1.25)	1.08 (0.52–2.23)
Namur	7	55	15	77	84	386	6	42	58	216	0.62 (0.24–1.61)	0.54 (0.24–1.22)	0.87 (0.47–1.60)	0.48 (0.19–1.17)
Other	3	55	18	77	0	386	4	42	0	216	0.21 (0.06–0.71) −	n.d. (n.d.–n.d.)	n.d. (n.d.–n.d.)	n.d. (n.d.–n.d.)
Type														
Saddle horses	30	57	39	71	204	386	69	128	204	386	0.91 (0.45–1.82)	0.98 (0.56–1.72)	1.08 (0.65–1.79)	1.04 (0.69–1.55)
Draught horses	4	57	8	71	40	386	12	128	40	386	0.62 (0.18–2.08)	0.71 (0.26–1.98)	1.14 (0.52–2.51)	0.91 (0.47–1.79)
Ponies	23	57	20	71	125	386	43	128	125	386	1.71 (0.82–3.56)	1.41 (0.80–2.49)	0.82 (0.47–1.44)	1.06 (0.69–1.61)
Donkeys	0	57	4	71	17	386	4	128	17	386	0.13 (0.00–2.47)	0.18 (0.01–3.09)	1.40 (0.48–4.09)	0.76 (0.26–2.19)
Sex														
Females	42	56	35	55	239	364	77	111	239	364	1.69 (0.75–3.79)	1.53 (0.81–2.89)	0.90 (0.50–1.62)	1.17 (0.74–1.85)
Colts and stallions	12	56	12	55	37	364	24	111	37	364	0.97 (0.40–2.37)	2.45 (1.20–5.00) +	2.50 (1.22–5.12) +	2.44 (1.39–4.28) +
Geldings	2	56	8	55	88	364	10	111	88	364	0.25 (0.05–1.10)	0.14 (0.03–0.52) −	0.55 (0.25–1.20)	0.32 (0.16–0.63) −
Colts, stallions or geldings	14	56	20	55	125	364	34	111	125	364	0.59 (0.26–1.32)	0.65 (0.35–1.23)	1.10 (0.61–1.98)	0.85 (0.54–1.34)
Temperament category														
Quiet	15	43	14	55	104	350	29	99	104	350	1.59 (0.67–3.76)	1.28 (0.66–2.48)	0.80 (0.42–1.52)	0.98 (0.60–1.60)
Alert/normal	25	43	34	55	194	350	59	99	194	350	0.89 (0.40–2.00)	1.10 (0.58–2.09)	1.23 (0.69–2.18)	1.18 (0.75–1.85)
Nervous	3	43	8	55	52	350	11	99	52	350	0.49 (0.13–1.83)	0.49 (0.15–1.52)	0.99 (0.45–2.18)	0.73 (0.37–1.45)
Body condition														
Thin	4	43	0	55	13	377	4	98	13	377	12.65 (0.66–241.00)	3.08 (1.01–9.39) +	0.2 (0.01–4.15)	1.3 (0.43–3.83)
Normal weight	35	43	41	55	247	377	76	98	247	377	1.46 (0.56–3.80)	2.20 (1.01–4.79) +	1.5 (0.80–2.85)	1.8 (1.07–3.00) +
Overweight	4	43	14	55	117	377	18	98	117	377	0.33 (0.1–1.02)	0.25 (0.09–0.69) −	0.8 (0.41–1.46)	0.5 (0.29–0.88) −
Regularly vaccinated	14	39	27	53	317	374	41	92	317	374	0.55 (0.24–1.27)	0.10 (0.05–0.21) −	0.2 (0.10–0.34) −	0.1 (0.09–0.24) −
Influenza	14	14	27	27	293	305	41	41	293	305	0.53 (0.01–27.97)	1.24 (0.07–21.90)	2.3 (0.14–40.64)	3.5 (0.21–60.83)
Tetanus	11	14	25	27	282	305	36	41	282	305	0.32 (0.06–1.88)	0.27 (0.08–0.97) −	0.8 (0.22–3.32)	0.6 (0.21–1.49)
Rhinopneumonia	6	14	12	27	145	301	18	41	145	301	0.95 (0.27–3.36)	0.82 (0.29–2.34)	0.9 (0.40–1.89)	0.8 (0.44–1.62)
Deworming	35	43	55	56	381	385	90	99	381	385	0.11 (0.02–0.67) −	0.05 (0.01–0.16) −	0.4 (0.07–2.83)	0.1 (0.04–0.35) −
Previous illness	2	30	2	50	79	338	4	80	79	338	1.70 (0.28–10.43)	0.29 (0.08–1.09)	0.2 (0.05–0.63) −	0.2 (0.07–0.52) −
Use for work														
Yes	6	45	7	57	240	363	13	102	240	363	1.10 (0.35–3.42)	0.08 (0.03–0.19) −	0.07 (0.03–0.16) −	0.07 (0.04–0.14) −
No	39	45	50	57	123	363	89	102	123	363	0.90 (0.29–2.79)	11.8 (5.02–27.8) +	13.1 (5.90–29.0) +	12.9 (7.00–23.8) +

+, risk factor; −, protection factor, both statistically significant at $P = 0.05$; n.d.: not determined because a selection bias (all control coming from the Wallon region).
[a] CC, histologically confirmed cases of atypical myopathy.
[b] Co-G, clinically healthy co-grazing Equidae.
[c] CT, control horses.
[d] P_{CC}, pasture where CC and Co-G were grazing.
[e] P_{CT}, pastures where CT were grazing.
[f] n = number of positive responses for each category.
[g] N = number of total responses.

suckling, this finding supports the hypothesis that the aetiological agent of AM is ingested. It might also imply the importance of protection by maternal antibodies in the young foal. On the other hand, it might mean that foals (that start rapidly to eat food other than their mother's milk) do not consume the minimum amount of 'toxins' required for the onset of clinical signs of AM. However, age is linked to the foaling season so it is predictable that the mortality of foals <5 months old is low in the autumn, the season when most of the cases occur.

Age was identified as a confounding variable regarding the risk factors associated with the animal's sex. Sex is linked to age, as 'gelding' is a status that follows castration, a procedure usually performed in males >18 months old (i.e. the most risky age for AM). Because females are more frequently kept at pasture than males (66% vs. 34% in CT), a higher percentage of CC were female (69% vs. 31% of males in CC). The use of the horse is also linked to age, since owners of horses usually do not start to work them when they are still growing, i.e. before the age of 3 years.

Independent of age, the study found a protective factor associated to the use that could be induced by supplementary feeds given to working horses (Harris, 1999). A history of previous illness was also more likely to be reported in

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Table 4
Age adjusted odds ratios (Mantel–Haenszel OR) calculated for sex, vaccination, deworming and use for work at the horse level for AM

Parameter		Number of horses												Adjusted odds ratio on age (95% CI)		
		⩽36 months						>36 months						CC vs. Co-G	CC vs. CT	Co-G vs. CT
		CC[a]		Co-G[b]		CT[c]		CC[a]		Co-G[b]		CT[c]				
		n[e]	N[f]	n[e]	N[f]	n[e]	N[f]	n[e]	N[f]	n[e]	N[f]	n[e]	N[f]			
Sex[d]	F	28	37	10	20	31	46	6	8	19	26	183	277	2.28 (0.88–5.92)	1.44 (0.63–3.33)	0.89 (0.46–1.75)
	M or G	9	37	10	20	15	46	2	8	7	26	94	277			
Use for work	Yes	2	37	0	20	12	48	4	8	7	26	197	277	3.57 (0.72–17.56)	0.26 (0.09–0.69) −	0.11 (0.05–0.26) −
	No	35	37	20	20	36	48	4	8	19	26	80	277			
Deworming	Yes	27	35	28	29	47	48	8	8	26	26	277	279	0.12 (0.02–0.79) −	0.88 (0.02–0.45) −	0.88 (0.06–13.00)
Minimum of once a year	No	8	35	1	29	1	48	0	8	0	26	2	279			
Vaccination	Yes	9	31	9	27	32	48	5	8	18	24	240	276	0.76 (0.30–1.95)	0.21 (0.10–0.48) −	0.31 (0.16–0.60) −
Minimum of once a year	No	22	31	18	27	16	48	3	8	7	24	36	276			

−, protection factor statistically significant at $P = 0.05$.
[a] CC, histologically confirmed cases of atypical myopathy.
[b] Co-G, clinically healthy co-grazing Equidae.
[c] CT, control horses.
[d] F: female, M: male, G: gelding.
[e] n = number of positive responses for each category.
[f] N = number of total responses.

older than in younger horses and may explain the reduced risk found in Co-G vs. CT and P_{CC} vs. P_{CT}. It might be hypothesised that previously ill horses were already affected by AM and might therefore have acquired resistance to the disease.

Except for those animals that had been affected with exercise-induced myopathy (6/79 horses with a previous history of illness in their lifetime in CT, 0/2 in CC and 0/2 in Co-G), the reported previous illnesses were various with no clinical signs resembling AM (e.g. dermatological, respiratory and abdominal diseases, laminitis; 73/79 in CT, 1/2 in CC and 2/2 in Co-G). One CC animal had shown clinical signs highly suggestive of AM the year before it died with a recurrence of signs of AM. It seems likely that this horse was affected twice by AM as, during the first episode, two horses (companions at pasture) presented identical clinical signs before dying and were confirmed as having AM. If this CC horse had suffered from a non-lethal episode of AM, the event did not protect the horse from a further fatal 'attack'. So, the possibility of acquired resistance remains questionable.

No confounding effect due to age was found with regard to deworming or vaccination. Thus, deworming and regular vaccination would appear to reduce the risk of AM.

As opposed to recurrent exertional rhabdomyolysis in racehorses (McLeay et al., 1999; McGowan et al., 2002), having a nervous, excitable temperament does not favour AM. This finding may reflect the different pathological process involved. In exercise-induced rhabdomyolysis, type II fibres of the locomotor muscles are selectively, but not exclusively, degenerated (McEwen and Hulland, 1986), whereas in AM, type I fibres of the postural muscles are predominantly affected (Brandt et al., 1997; Cassart et al., 2007).

Saddle horses, draft horses and ponies of various breeds can be affected with AM and there is no highlighted protective factor associated with breed rusticity. There was no donkey in our group of CC as opposed to the Co-G and CT groups; nevertheless the OR calculated for CC vs. Co-G and CC vs. CT were not statistically significant. It is likely that donkeys are clinically affected by AM since subclinical cases have been reported in the literature (Delguste et al., 2002).

When considering the geographic map of Belgium, AM distribution within the country is rather surprising: AM occurred predominantly in an area located under a line drawn by the Meuse River and one of its tributaries, the Sambre (Delguste et al., 2002). Pedological characteristics known to influence the vegetation to which the aetiological agent may be associated (and the delineation of pedological features between the North and South of the country at the level of the Sambre–Meuse line) might explain why AM is limited to the South of the country (Maréchal and Tavernier, 1970; Van Rompaey and Delvosalle, 1979). The Province of Liège, where heavy rainfall occurs (Mérienne, 2005), was more frequently affected by AM than the other provinces, and has been shown to be at high risk. However, from the 14 P_{CC} and 25 CC of the Province of Liège, 5 P_{CC} and 13 CC belonged to the same stud and this might have introduced a source of bias in interpreting the risks associated with the location. In fact, the stud had most of the risk factors associated with management practices at the horse and pasture level as identified in this study.

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Table 5
Odds ratios according to Grenier (1990) calculated for different management practices at the pasture level for AM

Characteristics and management practice	Pastures				Odds ratio (95% CI)
	P_{CC}[a]		P_{CT}[b]		P_{CC} vs. P_{CT}
	n[e]	N[f]	n	N	
Grazing of the pasture by other animals	2	30	43	213	0.34 (0.1–1.31)
Dead horses on the pasture (apart from CC and whatever the date)	9	24	11	94	4.45 (1.61–12.29) +
Salt block	14	33	283	357	0.20 (0.09–0.40) −
Grassland					
Lush in spring	15	25	154	204	0.48 (0.21–1.12)
Lush in summer	13	26	111	207	0.87 (0.39–1.93)
Lush in autumn	11	26	61	204	1.73 (0.76–3.93)
Lush in winter	7	24	19	198	3.95 (1.49–10.46) +
Nature of the pasture					
Natural	17	21	149	200	1.34 (0.45–3.96)
Obtained by sowing	4	21	51	200	0.75 (0.25–2.21)
Facilities					
Shelter	9	33	88	212	0.55 (0.25–1.21)
Bedding[c]	3	9	53	81	0.29 (0.07–1.14)
Drinking water[d] *provided by*					
Automatic drinker	5	26	47	210	0.75 (0.28–1.99)
Bucket	1	26	15	210	1.12 (0.28–4.49)
Tank	14	26	127	210	0.88 (0.42–1.85)
Watercourse	7	26	30	210	1.90 (0.77–4.69)
Nature of the water[d]					
Distribution	10	24	135	204	0.39 (0.17–0.88) −
Watercourse	8	24	36	204	2.36 (0.97–5.74)
Spring water	5	24	24	204	2.76 (0.96–7.89)
Rain water	4	24	22	204	0.95 (0.29–3.12)
Sloping pasture	27	34	103	207	3.70 (1.58–8.68) +
Yes, 100 %	11	25	16	61	2.19 (0.84–5.70)
Yes, partially	14	25	45	61	0.46 (0.18–1.19)
Steep slope ($\geqslant$10%)	12	25	22	93	2.94 (1.19–7.26) +
Gentle slope (<10%)	13	25	71	93	0.34 (0.14–0.84) −
Surrounded by or containing trees	34	35	175	214	5.18 (0.97–27.52)
Access to dead leaves piled up in autumn	21	23	96	216	10.74 (2.82–40.88) +
Humid pasture	21	36	74	215	2.63 (1.29–5.36) +
Surrounded by or containing a stream/river	13	27	54	216	2.78 (1.24–6.19) +
Management of the pastures					
Fertilizers	11	25	121	203	0.54 (0.24–1.23)
Spreading of manure	12	24	31	211	5.73 (2.40–13.69) +
Removal of faeces	0	24	5	211	0.77 (0.04–14.28)

+, risk factor; −, protection factor, both statistically significant at $P = 0.05$.
[a] P_{CC}, pasture where histologically confirmed cases of atypical myopathy and clinically healthy co-grazing Equidae were grazing.
[b] P_{CT}, pastures where control horses were grazing.
[c] Among those with a shelter.
[d] More than one answer may have been given.
[e] n = number of positive responses for each category.
[f] N = number of total responses.

When examined by the authors at the onset of AM, all visited P_{CC} were found to be particularly bare (21 pastures were surveyed at the time of the AM outbreak) and 84% of the owners of CC reported sparse grassland when AM broke out. On the other hand, when the owners of CC were questioned about the grassland over the four seasons, only 40% and 58% of P_{CC} were evaluated (by the same persons) as being bare during spring and autumn, respectively. This discrepancy over the evaluation of grassland quality may reflect the difficulty of self appraisal by owners of CC: when visiting the P_{CC}, all owners admitted the poor quality of their P_{CC}, but a significant percentage of them maintained,

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Table 6
Odds ratios according to Grenier (1990) calculated for different management practices at the horse level for AM

Characteristics and management practice	Animals						Odds ratio (95% CI)		
	CC[a]		Co-G[b]		CT[c]		CC vs. Co-G	CC vs. CT	Co-G vs. CT
	n[d]	*N*[e]	n	*N*	*n*	*N*			
Pasturing time over seasons									
Winter									
Less than 6 h	1	30	1	52	42	373	1.74 (0.17–17.5)	0.39 (0.07–2.10)	0.22 (0.04–1.18)
Between 6 and 12 h	1	30	6	52	67	373	0.36 (0.05–2.27)	0.23 (0.04–1.21)	0.63 (0.26–1.50)
24 h a day	18	30	22	52	121	373	2.00 (0.81–4.93)	3.07 (1.45–6.50) +	1.53 (0.85–2.75)
Depending on the weather	5	30	4	52	94	373	2.32 (0.61–8.83)	0.63 (0.24–1.65)	0.27 (0.10–0.74) −
Never at pasture	5	30	19	52	49	373	0.37 (0.12–1.08)	1.41 (0.53–3.72)	3.81 (2.02–7.18) +
Spring									
Less than 6 h	2	35	8	56	40	380	0.42 (0.09–1.86)	0.62 (0.16–2.36)	1.47 (0.66–3.27)
Between 6 and 12 h	1	35	0	56	25	380	4.91 (0.19–124)	0.60 (0.11–3.26)	0.12 (0.00–2.05)
24 h a day	30	35	43	56	226	380	1.72 (0.57–5.14)	3.78 (1.49–9.59) +	2.19 (1.15–4.18) +
Depending on the weather	2	35	3	56	89	380	1.14 (0.21–6.11)	0.24 (0.06–0.89) −	0.21 (0.07–0.64) −
Never at pasture	0	35	2	56	0	380	0.30 (0.01–6.58)	10.7 (0.20–548)	34.9 (1.65–736) +
Summer									
Less than 6 h	0	35	0	56	36	374	1.59 (0.03–82.0)	0.13 (0.00–2.17)	0.08 (0.00–1.35)
Between 6 and 12 h	0	35	0	56	17	374	1.59 (0.03–82.0)	0.28 (0.01–4.88)	0.18 (0.01–3.04)
24 h a day	35	35	56	56	282	374	0.62 (0.01–32.3)	23.2 (1.41–382) +	37.0 (2.26–604) +
Depending on the weather	0	35	0	56	39	374	1.59 (0.03–82.0)	0.11 (0.00–1.98)	0.07 (0.00–1.24)
Never at pasture	0	35	0	56	0	374	1.59 (0.03–82.0)	10.5 (0.20–539)	6.62 (0.13–337)
Autumn									
Less than 6 h	0	36	0	58	42	375	1.60 (0.03–82.5)	0.10 (0.00–1.78)	0.06 (0.00–1.10)
Between 6 and 12 h	2	36	0	58	52	375	8.47 (0.39–181)	0.44 (0.11–1.66)	0.05 (0.00–0.86) −
24 h a day	33	36	54	58	175	375	0.79 (0.18–3.40)	10.9 (3.56–33.4) +	13.8 (5.18–36.9) +
Depending on the weather	1	36	4	58	105	375	0.51 (0.07–3.40)	0.10 (0.02–0.56) −	0.21 (0.07–0.56) −
Never at pasture	0	36	0	58	1	375	1.60 (0.03–82.5)	3.42 (0.13–85.4)	2.13 (0.08–53.0)

+, risk factor, −, protection factor, both statistically significant at $P = 0.05$.
[a] CC, histologically confirmed cases of atypical myopathy.
[b] Co-G, clinically healthy co-grazing Equidae.
[c] CT, control horses.
[d] n = number of positive responses for each category.
[e] N = number of total responses.

nevertheless, that, generally speaking, their P_{CC} was lush during the high risk periods. In consequence, the risk factors associated with abundant grassland in winter must be interpreted with great caution.

The live livestock unit per hectare did not significantly differ between groups; nonetheless the time spent at pasture over the year was highest for the CC group and lowest for the CT group. This might explain why the P_{CC} were particularly bare. Permanent pasturing is an important risk factor for AM, whereas weather-dependent pasturing in risky seasons reduces the risk of AM. It may therefore be hypothesised (1) that permanent pasturing would decrease grassland quality, thus favouring the fact that horses would eat plants that would normally not be consumed (and were possibly carrying and/or containing the aetiological agent), and (2) that adverse climatic conditions would favour toxin production by the unknown agent (i.e. it has been long suspected that certain weather conditions predispose to the onset of clinical signs; Hosie et al., 1986; Hillam, 1991; Delguste et al., 2002; Votion et al., 2007). Overgrazing could also result in feed ingestion (e.g. grass or hay placed on the floor, soiled by earth and possibly containing pathogenic micro-organisms).

The spreading of horse manure was found to be an important risk factor for AM. This practice results in long term faecal contamination of soil with microbial contaminants such as spores of *Clostridia* spp., which are known to produce a wide variety of toxins. Equine manure is a significant source of bacteria and fungal spores (Lee et al., 2004) and persistent faecal contamination of soil might explain recurrence of AM on some pastures. It is notable that one series of cases (three CC which were pasture-companions died within 48 h) occurred the day after the mechanical harrowing of a pasture that was particularly bare. This harrowing may have led to the ingestion of grass 'contaminated' by earth. In further AM series, it might be of interest to study the relationship between the time of harrowing and the incidence of cases.

Our data suggest that receiving supplementary feed throughout the year may protect against AM. Horses receiving supplementary feed in their daily diet would be less prone to nutritional deviant behaviour (i.e. eating substances that would normally not be consumed when sufficient nutrients are available). On the other hand, horses fed with a complete mix might be more selective in their

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Table 7
Odds ratios according to Grenier (1990) calculated for different feeding practices at the horse level for AM

Feeding practice	Number of animals						Odds ratio (95% CI)		
Supplementary feeds	CC[a]		Co-G[b]		CT[c]		CC vs. Co-G	CC vs. CT	Co-G vs. CT
	n[e]	N[f]	n	N	n	N			
In winter	27	31	52	53	350	364	0.17 (0.02–1.17)	0.25 (0.08–0.77) −	1.44 (0.26–7.97)
Hay[d]	23	26	37	52	325	350	2.77 (0.77–9.87)	0.52 (0.15–1.73)	0.18 (0.09–0.38) −
Straw	11	26	28	52	234	350	0.63 (0.25–1.62)	0.36 (0.16–0.81) −	0.57 (0.32–1.03)
Silage	3	26	23	52	65	350	0.18 (0.05–0.65) −	0.64 (0.20–2.05)	3.47 (1.89–6.35) +
Complete mix	16	26	43	52	269	350	0.34 (0.12–0.97) −	0.47 (0.21–1.07)	1.38 (0.65–2.91)
Oats	4	26	9	52	28	350	0.91 (0.26–3.13)	2.26 (0.76–6.67)	2.47 (1.11–5.49) +
Barley	1	26	6	52	32	350	0.42 (0.06–2.64)	0.57 (0.10–3.11)	1.36 (0.55–3.35)
Corn	2	26	16	52	11	350	0.22 (0.05–0.93) −	3.01 (0.72–12.5)	13.3 (5.83–30.5) +
In spring	6	30	10	53	243	355	1.09 (0.36–3.28)	0.12 (0.05–0.29) −	0.11 (0.05–0.22) −
Hay[d]	3	6	4	10	185	243	1.44 (0.21–9.73)	0.31 (0.06–1.42)	0.21 (0.06–0.75) −
Straw	2	6	3	10	123	243	1.19 (0.16–8.79)	0.54 (0.11–2.59)	0.45 (0.12–1.65)
Silage	0	6	3	10	17	243	0.16 (0.00–3.82)	0.99 (0.05–18.4)	6.04 (1.55–23.5) +
Complete mix	5	6	10	10	184	243	0.17 (0.00–5.04)	1.18 (0.18–7.36)	6.77 (0.39–117)
Oats	0	6	0	10	9	243	1.61 (0.02–91.8)	1.89 (0.09–36.2)	1.17 (0.06–21.5)
Barley	0	6	0	10	18	243	1.61 (0.02–91.8)	0.93 (0.05–17.3)	0.58 (0.03–10.3)
Corn	0	6	0	10	0	243	1.61 (0.02–91.8)	37.4 (0.68–2038)	23.1 (0.43–1226)
In summer	3	34	6	55	182	365	0.84 (0.21–3.34)	0.11 (0.03–0.34) −	0.13 (0.05–0.30) −
Hay[d]	0	3	0	6	108	182	1.85 (0.02–115)	0.09 (0.00–1.92)	0.05 (0.00–0.95) −
Straw	0	3	0	6	75	182	1.85 (0.02–115)	0.20 (0.01–3.99)	0.10 (0.00–1.97)
Silage	0	3	0	6	2	182	1.85 (0.02–115)	10.3 (0.41–256)	5.55 (0.24–127)
Complete mix	3	3	6	6	132	182	0.53 (0.00–33.4)	2.66 (0.13–52.5)	4.95 (0.27–89.5)
Oats	0	3	0	6	6	182	1.85 (0.02–115)	3.87 (0.18–83.1)	2.08 (0.10–41.1)
Barley	0	3	0	6	16	182	1.85 (0.02–115)	1.44 (0.07–29.1)	0.77 (0.04–14.4)
Corn	0	3	0	6	0	182	1.85 (0.02–115)	52.1 (0.89–3023)	28.0 (0.51–1528)
In autumn	16	35	33	57	295	369	0.61 (0.26–1.42)	0.21 (0.10–0.43) −	0.34 (0.19–0.61) −
Hay[d]	11	16	11	33	240	295	4.09 (1.18–14.1) +	0.48 (0.16–1.38)	0.11 (0.05–0.25) −
Straw	6	16	6	31	151	295	2.42 (0.65–8.94)	0.59 (0.21–1.61)	0.24 (0.09–0.59) −
Silage	2	16	14	31	39	295	0.20 (0.04–0.94) −	1.11 (0.28–4.46)	5.38 (2.48–11.6) +
Complete mix	10	16	28	31	229	295	0.19 (0.04–0.87) −	0.46 (0.16–1.29)	2.35 (0.75–7.40)
Oats	3	16	3	31	23	295	2.11 (0.41–10.6)	3.00 (0.86–10.4)	1.42 (0.43–4.66)
Barley	0	16	0	31	23	295	1.90 (0.03–100)	0.35 (0.02–6.04)	0.18 (0.01–3.10)
Corn	1	16	10	31	6	295	0.19 (0.03–1.23)	4.31 (0.67–27.3)	21.7 (7.44–63.5) +

+, risk factor, −, protection factor, both statistically significant at $P = 0.05$.
[a] CC, histologically confirmed cases of atypical myopathy.
[b] Co-G, clinically healthy co-grazing Equidae.
[c] CT, control horses.
[d] Those owners whose horses were receiving supplementary feeds and who provided an answer.
[e] n = number of positive responses for each category.
[f] N = number of total responses.

grazing. In addition, horses receiving a complete mix might be better protected against rhabdomyolysis because they would have more protective factors such as antioxidants and salts. The fact that giving supplementary salt by means of a salt block was found to be a protective factor for AM supports this hypothesis. Indeed, it is worth noting that nutritional myopathy (also called nutritional myodegeneration or white muscle disease) in horses, associated with dietary deficiency of selenium and/or vitamin E (Löfstedt, 1997; Saito et al., 2003), mimics AM in many respects (Votion et al., 2004). A large majority of CC in our study received hay, which does not provide a large quantity of selenium and/or vitamin E.

Providing hay in the autumn was found to be a factor that favoured AM in CC but it appears nevertheless as a protective factor (according to the OR calculated for Co-G vs. CT). The discrepancy between the positive and negative effect of giving hay in autumn in CC vs. CT might be due to the environment when hay is placed on the ground. In fact, hay may be an important source of bacteria and fungal spores (Lee et al., 2004) that in a humid environment then find more appropriate conditions for exerting potential toxicity. This might explain why hay favours AM in CC that were pastured more frequently in a humid pasture than CT.

Overweight contributed to a reduced risk of AM. Where AM would result in muscle energetics imbalance, it is reasonable to assume that fat animals would better withstand the condition. Also, the relationship between feeding, pasture-management practices and risk of AM reinforces the hypothesis that the condition may be induced or predisposed to by a factor associated with the herbage.

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Most of the P_{CC} presented humid areas or pedological characteristics (e.g. a steep slope; Votion et al., 2007) that favoured humidity. Accumulation of dead leaves in a humid environment encourages mould development that might produce mycotoxins. Moreover, silage supplementation in winter, spring, and autumn increases the risk of AM. Silage can be a source of mycotoxins after thermal, humid and physical stresses that can take place before or after harvest or during storage (Saegerman et al., 2006). In many P_{CC} surrounded or including a river, the water provided to the horses came from the watercourse (7/13 P_{CC} with a river). Providing water from the distribution network reduces the risk of AM. It should be noted that at present there is no evidence that watercourses are the source of the aetiological agent of AM.

Management practices that favour AM are commonly seen in horse husbandry. Many stud farms fed hay in autumn, spread manure, harrowed pastures and provided access to humid areas for water and an increasing number of AM outbreaks has been observed in recent years. Some management practices that are centuries old may have to be revisited to counteract the progress of AM. The factors identified in this study may be of help in improving management practices while looking realistically at the situation. For example, pasture rotation could be organised so only dry and flat pastures are available to young horses in autumn and spring and access to rivers and areas with accumulated dead leaves are avoided at high risk periods.

Conclusions

This case control study is the first major epidemiological study of AM and has identified key demographic, management and environmental exploratory variables that should be considered in developing evidence-based disease avoidance strategies. Specifically, our findings suggest that horses <3 years old should not be pastured on sloping or humid pastures (or those containing a waterway) during autumn and spring, especially if the premises have already had one or more cases of AM. The study also suggests that certain management practices at the horse level (i.e. deworming and regular vaccination, stabling horses during bad weather in autumn and spring, providing a salt block, supplementary feeding all year round, and drinking water via the distribution network) and the pasture level (i.e. removal of dead leaves and proscribing spreading of manure) may help reduce the incidence of AM. Other factors (e.g. co-grazing with other animals) appear to have no influence on the risk of AM. Our findings have indicated some hypotheses about the possible aetiology of the disease, including bacteria, plants and fungi as potential producers of muscle toxins.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

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