

Article

Familial Aggregation of Chiari-like Malformation and Syringomyelia in Pomeranians

Annabel Bakker ^{1,2} , Roos Duvekot ², Michelle Hermans ^{2,3} , Erik Noorman ⁴, Stefanie Veraa ⁵ and Paul J. J. Mandigers ^{1,2,*} 

¹ Section Neurology and Neurosurgery, Evidensia Referral Clinic Arnhem, Meander 10, 6825 MB Arnhem, The Netherlands

² Expertise Centre of Genetics, Department of Clinical Sciences, Faculty of Veterinary Medicine, Utrecht University, Yalelaan 108, 3584 CM Utrecht, The Netherlands

³ Neurology Service, Orion Companion Animal Hospital, 2200 Herentals, Belgium

⁴ Dierenkliniek Den Heuvel, 5684 NH Oirschot, The Netherlands

⁵ Division of Diagnostic Imaging, Department Clinical Sciences, Faculty of Veterinary Medicine, Utrecht University, 3584 CM Utrecht, The Netherlands

* Correspondence: p.j.j.mandigers@uu.nl

Abstract

Background: Chiari-like malformation (CM) and syringomyelia (SM) are common neurological disorders in Pomeranian dogs. The familial aggregation of these disorders has not been thoroughly characterised in this breed. **Methods:** A cohort of 999 Pomeranians examined with MRI for CM/SM was retrospectively reviewed. Siblings within litters were identified and compared, and the familial aggregation between offspring and parents screened for these conditions was investigated. **Results:** The mean central canal dilation (CCD) increased statistically significantly with the age of the dog at the time of the MRI scan. Forty-three per cent of all the dogs were free of CM, with 57% had CM. SM was detected in 31% of all the dogs, while 69% were not affected. Only 37% of all the dogs were free of both conditions. SM was observed in 6% of all the dogs without being affected by CM. Thirty-nine litters were identified in which two or more siblings were scanned. Siblings within the same litter had comparable scans in almost all the cases. Although pups from affected parents have a higher risk of SM, and pups bred from unaffected parents have a lower incidence of SM, the statistical significance could only be demonstrated for the >3 years of age category of SM in offspring, compared with the presence of SM in the mother. It could not be demonstrated for CM. **Conclusions:** Both CM and SM are frequently observed in this breed, and SM tends to progress over time. Siblings often show comparable findings. The screening results obtained from mothers aged >3 years indicated that SM-affected mothers are more likely to have affected offspring. CM/SM are most likely heritable traits in this breed, and to reduce the frequency of affected pups, breeders are encouraged to screen their breeding stock and use only unaffected parents.



Academic Editor: Osamu Yamato

Received: 18 May 2026

Revised: 18 June 2026

Accepted: 25 June 2026

Published: 26 June 2026

Copyright: © 2026 by the authors.

Licensee MDPI, Basel, Switzerland.

This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC BY\)](https://creativecommons.org/licenses/by/4.0/) license.

Keywords: small breeds dogs; heritability; neurological; screening; MRI

1. Introduction

Chiari-like malformation (CM, also called canine Chiari) and syringomyelia (SM) are frequently seen disorders in several toy breeds and small dogs [1–6]. CM is characterised by a decreased volume of the caudal fossa and caudal displacement of the caudal cerebellar vermis into or through the foramen magnum [7,8] and increased cerebellar volume [9].

Syringomyelia is defined as the presence of a fluid-filled cavity in the spinal cord (syrinx) and is believed to result from an abnormal CSF flow either related to hindbrain problems (CM) and/or the influence of the systolic/diastolic arterial pulse and CSF pulse in the subarachnoid space [7–12]. It was first reported within the Cavalier King Charles Spaniel (CKCS) [13] and recent studies estimated the prevalence of CM up to 99% and for SM up to 46% [14]. Despite CM/SM being within the CKCS, most likely hereditary [6,14,15], with so many dogs affected, a genetic study can be challenging [16].

To reduce the incidence of CM/SM in the CKCS, the British Veterinary Association (BVA) and the Kennel Club (KC) published guidelines for the evaluation of the MRI scans and breeding advice (<https://www.bva.co.uk/canine-health-schemes/cmsm-scheme/>, accessed on 1 April 2026). Just recently, our group investigated the effect of selective breeding against CM/SM, and concluded that improvement is possible, provided the breeding stock is screened using MRI, and only SM-unaffected dogs are used for breeding [14]. However, only in the Netherlands and Denmark are CKCS breeders required to screen their breeding stock for CM/SM [14]. In all other countries it is only advised. Given that CM/SM is a welfare-diminishing disorder, this is remarkable. In other breeds at risk of developing CM/SM, for instance, the Chihuahua, French Bulldog, Yorkshire terrier, Biewer terrier, Griffon, Pug, etc., screening is either not performed or only advised in rare cases [3,17,18].

CM/SM is also frequently reported within the Pomeranian dog, with lower numbers of dogs being affected compared to the CKCS [5,19], which may enable genetic studies more easily. In a not randomly chosen group of 796 Pomeranian dogs, the prevalence was 54.9% for CM and 23.9% for SM. In this study, the dogs came from 22 different countries, indicating that it is a global, not just a local, problem [5]. In contrast with the CKCS, for which several studies have been published demonstrating the heritability of the two disorders [6,14,18,20], this has not been done in the Pomeranian dog.

Nowadays Pomeranian dog breeders in the Netherlands are advised to screen their breeding stock for CM/SM, but this advice was only implemented around 2020. Dogs bred before this date may or may not have been screened, depending on the breeder's choice. Dogs may only be used if free of SM. Dogs may be used for breeding if they only have a very mild CM (<https://www.keeshondenclub.nl/wp/aanvraag-fokgeschiedenis-keuring/>, accessed on 1 April 2026). Breeders are also instructed to send the MRI scan for evaluation to the last author (PM). However, these rules apply only to breeders who are members of the official breed club. If they are not members, they can use any dog and, whether or not the dog is scanned, can obtain an official pedigree from the Dutch Kennel Club. They are also not obliged to have the MRI scan evaluated by a veterinary radiologist or neurologist.

The goal of this study was to investigate whether CM/SM shows familial aggregation, whether selection towards unaffected dogs has already been done, and, if so, whether it is effective. Several hypotheses were formulated: (1) siblings are equally affected by CM and SM; (2) pups from parents affected with CM and/or SM have a higher risk of being affected with CM and/or SM; and (3) pups bred from unaffected parents have a lower incidence of CM and/or SM.

2. Materials and Methods

2.1. Dogs

All the included Pomeranian dogs were seen between 2015 and 2025 for CM/SM screening as described earlier [5,6]. From each dog the microchip number, sex, date of birth, colour, country of origin, and age of the dog at the moment of screening were retrieved. If a pedigree registration was available, this was registered as well. If not, an attempt was made to retrieve information on siblings and/or parents using the microchip number in existing international pedigree registration databases. The dogs could have been screened

before or after mating, as screening before mating is only advised as of 2020. Owners and dogs could participate only if written informed consent was obtained from the owner prior to inclusion. Before starting this study, approval was sought from the animal welfare body at Utrecht University. Although approval was not required, as all the dogs included were referred for clinical reasons, the complete procedure was evaluated by the animal welfare body and approved and registered under number 16205-20-26.

2.2. MRI Studies

MRI studies were performed under general anaesthesia (with individualised anaesthetic protocols) with a high-field MRI scanner at either the IVC Evidensia Referral Hospital Arnhem, The Netherlands (Canon Vantage Elan 1.5T MRI, Otawara-shi, Japan) or the Department of Clinical Sciences, Utrecht University, The Netherlands (Philips Ingenia 1.5T MRI, Eindhoven, The Netherlands), or with a low-field MRI scanner at Dierenkliniek den Heuvel, Best, The Netherlands (Vet-MR Grande, 0.25 Tesla, ESAOTE, Genoa, Italy) or Orion Clinic, Herentals, Belgium (Vet-MR Grande, 0.25 Tesla, ESAOTE, Genoa, Italy).

Whether it was a low-field or high-field MRI scanner, both T1-weighted (T1W) and T2-weighted (T2W) sagittal and transverse images were obtained using the settings described in Appendix A. The maximum slice thickness was 4 millimetres (mm). To ensure consistency across the MRI centres, the scanning protocol (Appendix A) was thoroughly discussed with all the participating investigators. For positioning, the dog's head and neck were extended so that the skull base aligned approximately with the floor of the vertebral canal at C1 and C2, as described previously [5]. If a dog was scanned twice, only the most recent scan was used.

All the MR images were re-evaluated using the Horos DICOM viewer graphical software version 4.0.1. (<https://horosproject.org/>) by the last author (PJJM), an ECVN board-certified veterinary specialist. All the scans were evaluated using the BVA/KC scheme with two important adaptations.

CM was classified according to the grading system described in previous studies [6,14].

- CM0—No cerebellar herniation or impaction (cerebellar uvula rostral to foramen magnum).
- CM1—Cerebellar impaction (cerebellar uvula on the line of the foramen magnum, no CSF present dorsal to the cervicomedullary junction) and non-rounded shape.
- CM2—Cerebellar herniation (cerebellar uvula caudal to the line of the foramen magnum, no CSF present dorsal to the cervicomedullary junction).
- CM3—Cerebellar herniation resembling CM2, but the posterior part of the cerebellum is herniated and shaped like a tongue.
- CM4—Cerebellar herniation resembling CM2, but the posterior part of the cerebellum is severely herniated and distinctly tongue-shaped.

The presence of SM was established by measurement of the exact width of the largest fluid-filled cavity in mm on the transverse T1-weighted images, as differentiation between true syrinx margins and spinal cord oedema surrounding the syrinx is more difficult on T2W than T1W images [21]. The scans were further subdivided into the three BVA/KC Scheme age groups (a: over 5 years of age, b: 3 to 5 years of age, and c: 1 to 3 years of age).

In this study, SM unaffected Pomeranian dogs were defined as those with a central canal dilation (CCD) of 0 mm, while those with a visible fluid-filled cavity (FFC) (>0 mm) on at least two transverse images were considered affected with SM.

2.3. Exclusion Criteria

Dogs diagnosed with intracranial space-occupying lesions or space-occupying lesions in the vertebral canal were excluded from the study. Additionally, if the MRI exhibited excessive artefacts, these dogs were also excluded. Furthermore, Pomeranians with a

microchip number or pedigree number that did not correspond to that specific dog, or with incomplete data on CM or SM, were also removed.

2.4. Data Management

All the dogs and scanning results were managed using Microsoft® Excel, where they were manually checked for duplicates. If a duplicate existed, this duplicate was removed. The scan results of available siblings and parents were linked to each dog.

2.5. Statistical Analysis

The statistical analysis was performed with R version 4.5.2. [22]. Descriptive statistics was used for CM/SM, country of origin, and sex. Continuous data were tested for normality using a Kolmogorov–Smirnov test. A Wilcoxon signed-rank test was used to compare siblings. A Kruskal–Wallis test was used to assess the possible correlation between the grading of CM/SM across age groups and familial aggregation. A generalised linear model [23] was used to assess whether SM presence in offspring was affected by SM presence in father or mother. A subset was made, including dogs > 3 years old, as SM is a late-onset disease. It was also assessed whether the CM grade in offspring was affected by the CM grade of the mother or the father. The ANOVA function [22] was used on the model output to assess statistical differences. *p*-values of <0.05 were regarded as significant.

3. Results

3.1. Dogs

A total of 1059 Pomeranians were scanned between 2015 and 2025. After removing duplicates and those with exclusion criteria, 999 Pomeranian dogs (494 sires and 505 bitches) remained for analysis. The majority of the dogs came from Russia (*n* = 364). Other countries were the Netherlands (*n* = 282), Belarus (*n* = 43), Germany (*n* = 40), Ukraine (*n* = 14), Belgium (*n* = 10), Bulgaria (*n* = 10), and a variety of other countries (*n* = 29). For 198 Pomeranian dogs, the country of origin could not be retrieved.

3.2. CM/SM Status for All 999 Pomeranian Dogs

The mean CCD ± SD in mm increased with the age of the dog at the time of the MRI scan (Table 1). The increase in CCD was statistically significant (*p* < 0.001; Kruskal–Wallis test), indicating that CCD increases with age.

Table 1. Average age (±SD) and CCD for three age groups (a: >5 years, b: 3–5 years, and c: 1–3 years).

	Age Group c (<i>n</i> = 617)	Age Group b (<i>n</i> = 262)	Age Group a (<i>n</i> = 120)
Age at screening	1.7 ± 0.6 years	3.8 ± 0.7 years	6.3 ± 1.3 years
CCD	0.64 ± 1.24 mm	0.92 ± 1.49 mm	1.12 ± 1.52 mm

The CM status could be established in all the dogs (Table 2). Although the frequency of CM status varied slightly, the difference was not statistically significant (*p* = 0.17; Kruskal–Wallis test).

Table 2. CM grading for each age group (a: >5 years, b: 3–5 years, and c: 1–3 years).

CM Grading	Age Group c (<i>n</i> = 617 and %)	Age Group b (<i>n</i> = 262 and %)	Age Group a (<i>n</i> = 120 and %)
CM0	294 (48%)	96 (37%)	40 (33%)
CM1	269 (43%)	146 (56%)	66 (55%)
CM2	47 (8%)	18 (7%)	13 (11%)
CM3	7 (1%)	2 (-%)	1 (1%)

CM0 was observed in 43% of all the dogs, with 57% having CM1 or higher. SM was detected in 31% of all the dogs, while 69% were not affected. Only 37% of all the dogs were free of both conditions. SM was observed in 6% of all the dogs without being affected by CM (Table 3).

Table 3. CM and SM grading for all dogs.

CM/SM Grading	SM0 (Number)	SM1 (Number)	SM2 (Number)
CM0	372	20	38
CM1	270	70	141
CM2	40	11	27
CM3	6	2	2

3.3. CM/SM Status Between Siblings

Thirty-nine litters, comprising 80 scanned dogs in which two or more siblings were scanned, could be analysed. The results, obtained for the same age groups, showed that, for both CM and SM, siblings within the same litter had comparable scans in almost all the cases. The Wilcoxon signed-rank test revealed no significant differences between siblings (CM: $p = 0.157$, SM: $p = 0.286$).

3.4. CM/SM Status Between Litters

Seventy-seven dogs had one or both parents scanned. All of the scanned parents had a Dutch Kennel Club pedigree registration. Only 23 dogs had both parents scanned; for 22 dogs, only the father was scanned; and for another 32 dogs, only the mother was scanned. Of this group of fathers and mothers, only 12 fathers and 26 mothers were scanned at an age of three years or above.

The average age for these 77 dogs was 2.5 ± 1.6 years. CM0 was observed in 35 dogs, CM1 in 40, CM2 in one and CM3 in another dog. Fifty-nine dogs were free of SM, with 18 being affected with an average CCD of 2.15 ± 1.4 mm.

Of the 35 dogs with CM0, four out of 21 fathers had CM1 or above, and 15 out of 26 mothers had CM1 or above. Of the 42 dogs with CM1 or above, 25 out of 58 fathers had CM1 or above, and 12 out of 29 mothers had CM1 or above. Therefore, parents without CM did produce offspring with CM and vice versa. Logistic regression showed no significant differences between parental CM and the CM status of the offspring.

The average CCD $\pm$ SD for each dog is calculated for dogs with only the father, only the mother, or both parents free of SM, or affected by SM (Table 4).

Table 4. CCD results.

CM/SM Grading	CCD Result Offspring	CCD Result Parent	Number of Dogs
Only father free of SM	0.34 ± 0.77 mm	0	41
Only mother free of SM	0.34 ± 0.95 mm	0	46
Both parents free of SM	0.4 ± 0.88 mm	0	20
Father not free of SM	1 ± 1.4 mm	0.85 ± 0.65 mm	4
Mother not free of SM	1.8 ± 1.9 mm	2.43 ± 0.91 mm	9
Both parents not free of SM	3 mm	sire 0.5 mm and bitch 2 mm	1

When comparing CCD in offspring to CCD in father or mother for all the age categories, no significant difference was found ($p > 0.05$). In the >3 years age category, limited data were available on the CCD of the father ($n = 5$). Therefore, no differences were detected. In

the >3 years of age category of CDD in offspring compared to CCD presence in the mother ($n = 17$), a significant difference was found ($p = 0.02$).

When comparing CM status in offspring compared to CM status in father or mother for all the age categories, no significant difference was found.

4. Discussion

The goal of this study was to investigate whether CM/SM shows familial aggregation, whether selection towards unaffected dogs has already been done, and, if so, whether it is effective. The first observation is that of the 999 examined dogs, only 77 Pomeranians (8%) had one or both parents scanned. This low number can partly be explained by the fact that at least 43% of imported dogs originate from an Eastern European country. CM/SM screening is, to the authors' knowledge, very rarely performed in Eastern European countries. Twenty-eight per cent of the 999 examined dogs had a Dutch registration number, and it was to be expected that more dogs would have scanned parents, but surprisingly, this is not so. Given that CM/SM is a welfare-diminishing disorder, screening parents prior to breeding should be a more common practice in the Netherlands.

SM is, within the CKCS, also called a late-onset disease, as it may be absent in a young CKCS, but becomes visible at an older age [6,14,24]. For this reason, CKCS breeders are warned that scan results obtained at a young age may differ as the dog matures [14]. Whether this is also seen in the Pomeranian dog has been the subject of two earlier studies. In 2023 Santifort et al. observed that the number of dogs affected with SM was higher in older dogs [5]. And in 2024 Jansma et al. conducted a longitudinal study with 49 Pomeranian dogs and demonstrated that SM, in a number of dogs, progressed over time [15]. The results obtained in this study strengthen the conclusion of the two earlier observations of Santifort et al. [5] and Jansma et al. [15], as it is now clearly demonstrated that SM, as it is in the CKCS, is a so-called late-onset disease. The older the dog gets, the higher the frequency of affected dogs. In this study, 51% of the parents had a screening result obtained before the age of three. Screening prior to the age of three helps to prevent breeding with an affected parent if abnormalities are found. However, if such a parent becomes affected at a later age, the breeder has still used an affected dog. Only 49% of the parents had a screening result after reaching the age of three. Given that SM in this breed is also a late-onset disease, Pomeranian breeders who want to avoid using affected dogs are advised to screen their breeding stock both before and after reaching the age of three.

An interesting finding, earlier also observed by Santifort et al. [5], is that 6% of all 999 dogs suffered from SM whilst not affected by CM. This has also been observed earlier in several other breeds, such as the Maltese dog and the French Bulldog [3]. This observation questions the need for brachycephalism to develop SM. CM and/or brachycephalism may play a role in the development of SM, but, at least in this breed, it is not the only factor.

The first hypothesis was that siblings are equally affected by CM and SM. The CM/SM status was similar among siblings for both conditions. The hypothesis could not be rejected, and this is a first indication that the two disorders are most likely heritable.

The second and third hypotheses were that pups from parents affected with CM and/or SM have a higher risk of being affected with CM and/or SM, and that pups bred from unaffected parents have a lower incidence of CM and/or SM. These hypotheses have been applied to the CKCS and could not be rejected in this breed, strengthening the need for screening and selection based on screening. The effect of screening for SM within the CKCS has been the subject of two different studies. In 2007, based on presumed heritability, breeding advice was formulated to reduce the presence of SM [25]. Five years later, in 2011, Knowler et al. [18] demonstrated that selection against SM does reduce the percentage of affected CKCS. They showed that if both parents were affected, 92% of offspring would

be at risk of being affected, and if one parent was affected, the risk for CKCS would be 77%. The second study to demonstrate this was performed by Limpens et al. [14], who demonstrated in their study investigating the effect of selection against SM that, if CKCS breeders use only unaffected dogs, the odds of producing affected offspring would be significantly lower than when using one or two affected parents. In the current study, this effect could only be demonstrated for mothers above the age of three. However, it must be noted that the number of screened parents is very small. It is possible that, if a larger group were examined, the effect would be more pronounced. Nevertheless, the findings that siblings are equally affected by CM and SM, and that offspring from affected mothers, who are screened above the age of three, have a statistically significantly higher risk of being affected, are the first to demonstrate that CM/SM are also heritable conditions in the Pomeranian breed.

This study, which investigates the familial aggregation of these two disorders within this breed, has limitations. First, a large number of the examined dogs originate from countries where screening for CM/SM is not common practice. Hence, it is impossible to draw any conclusions from this group. The affected pups could have affected parents, and vice versa, but this information is unknown. Second, the sample size is small. Information on two or three siblings was available from only 39 litters. However, it still clearly demonstrated that siblings have an equal chance of being affected. This limitation also applies to the parental effect, but again, the findings hopefully encourage breeders to screen their breeding stock to reduce the number of affected pups, as CM/SM are welfare-diminishing disorders [5,26].

5. Conclusions

CM and SM are, within Pomeranian dogs, most likely heritable disorders warranting further genetic research. Siblings are equally affected by CM and SM; although the effect was statistically significant only for mothers, pups from affected parents have a higher risk of SM, whereas pups from unaffected parents have a lower incidence of SM. This effect could not be demonstrated for CM. To reduce the incidence of affected pups, breeders are encouraged to screen their breeding stock and use only unaffected parents. The effect of selection will likely be greater if only screening results from higher ages are taken into account.

Author Contributions: Conceptualisation, A.B. and P.J.J.M.; methodology, P.J.J.M.; software, A.B. and P.J.J.M.; validation, R.D. and P.J.J.M.; formal analysis, R.D. and P.J.J.M.; investigation, A.B., R.D., and P.J.J.M.; resources, M.H., E.N., S.V., and P.J.J.M.; data curation, P.J.J.M.; writing—original draft preparation, A.B. and P.J.J.M.; writing—review and editing, A.B., R.D., M.H., E.N., S.V., and P.J.J.M.; visualisation, P.J.J.M.; supervision, P.J.J.M.; project administration, P.J.J.M.; funding acquisition, P.J.J.M. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Before starting this study, approval was sought from the animal welfare body at Utrecht University. Although approval was not required, as all the dogs included were referred for clinical reasons, the complete procedure was evaluated by the animal welfare body and approved and registered under number 16205-20-26.

Informed Consent Statement: Owners and dogs could participate only if written informed consent was obtained from the owner prior to inclusion.

Data Availability Statement: The data used for this study are available upon request.

Acknowledgments: The authors are grateful to all participating owners and breeders.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

BVA	British Veterinary Association
CCD	Central canal dilatation
CM	Chiari-like Malformation
CKCS	Cavalier King Charles Spaniel
ECVN	European College of Veterinary Neurology
FCI	Federation Cynologique Internationale
KC	Kennel Club
MRI	Magnetic Resonance Imaging
SM	Syringomyelia

Appendix A

The following sequences were minimally acquired to ensure diagnostic-quality MRI for assessing Chiari-like malformation (CM) and syringomyelia (SM) in this study:

Sagittal T1-weighted (T1W) and T2-weighted (T2W) images must extend from the interthalamic adhesion to at least the T4/T5 intervertebral disc space, with a maximum slice thickness of 4 mm. The head and neck should be aligned in the sagittal plane. If complete alignment is not feasible, separate sagittal images of the brain and cervical spinal cord must be acquired to ensure midline sagittal representation of each structure. Images are considered non-diagnostic if the central nervous system (CNS), from the cisterna magna to the T4/T5 intervertebral disc space, is not visible in a single continuous sagittal image. An exception applies in cases of scoliosis, where dorsal plane images of the affected region must also be provided. Scoliosis associated with SM typically co-occurs with a marked syrinx. In the absence of syringomyelia, the presence of scoliosis may be questioned, and the images deemed insufficient.

Transverse T1W or T2W images should be obtained perpendicular to the spinal cord at the level of maximum syrinx width if syringomyelia (SM) is present. Alternatively, in the absence of a syrinx, a transverse image block should be centred on the C3 vertebra, extending from at least the midpoint of the vertebral body of C2 to the midpoint of the vertebral body of C4. The maximum slice thickness was 4 mm.

Positioning requirements: The head and neck should be positioned in extension, with the skull base approximately aligned with the floor of the vertebral canal through C1 and C2.

References

1. Rusbridge, C.; Knowler, S.P.; Pieterse, L.; McFadyen, A.K. Chiari-like malformation in the Griffon Bruxellois. *J. Small Anim. Pract.* **2009**, *50*, 386–393. [[PubMed](#)]
2. Kiviranta, A.M.; Rusbridge, C.; Lappalainen, A.K.; Junnila, J.J.T.; Jokinen, T.S. Persistent fontanelles in Chihuahuas. Part II: Association with craniocervical junction abnormalities, syringomyelia, and ventricular volume. *J. Vet. Intern. Med. Am. Coll. Vet. Intern. Med.* **2021**, *35*, 1848–1856. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
3. Hoholm, F.; Bruining-Staal, K.; Mandigers, P.J.J. Incidence of Chiari-like Malformation/Syringomyelia in a Cohort of Small Dog Breeds Scanned Using MRI over a Period of 8 Years in The Netherlands. *Pets* **2024**, *1*, 16. [[CrossRef](#)]
4. Knowler, S.P.; Kiviranta, A.M.; McFadyen, A.K.; Jokinen, T.S.; La Ragione, R.M.; Rusbridge, C. Craniometric Analysis of the Hindbrain and Craniocervical Junction of Chihuahua, Affenpinscher and Cavalier King Charles Spaniel Dogs with and Without Syringomyelia Secondary to Chiari-Like Malformation. *PLoS ONE* **2017**, *12*, e0169898. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
5. Santifort, K.M.; Carrera, I.; Bossens, K.; Mandigers, P.J.J. Phenotypic characterization of Pomeranians with or without Chiari-like malformation and syringomyelia. *Front. Vet. Med.* **2023**, *10*, 13. [[CrossRef](#)] [[PubMed](#)]
6. Wijnrocx, K.; Van Bruggen, L.W.L.; Eggelmeijer, W.; Noorman, E.; Jacques, A.; Buys, N.; Janssens, S.; Mandigers, P.J.J. Twelve years of chiari-like malformation and syringomyelia scanning in Cavalier King Charles Spaniels in The Netherlands: Towards a more precise phenotype. *PLoS ONE* **2017**, *12*, e0184893. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]

7. Rusbridge, C.; Greitz, D.; Iskandar, B.J. Syringomyelia: Current concepts in pathogenesis, diagnosis, and treatment. *J. Vet. Intern. Med.* **2006**, *20*, 469–479. [[CrossRef](#)] [[PubMed](#)]
8. Driver, C.J.; Volk, H.A.; Rusbridge, C.; Van Ham, L.M. An update on the pathogenesis of syringomyelia secondary to Chiari-like malformations in dogs. *Vet. J.* **2013**, *198*, 551–559. [[CrossRef](#)] [[PubMed](#)]
9. Shaw, T.A.; McGonnell, I.M.; Driver, C.J.; Rusbridge, C.; Volk, H.A. Increase in cerebellar volume in Cavalier King Charles Spaniels with Chiari-like malformation and its role in the development of syringomyelia. *PLoS ONE* **2012**, *7*, e33660. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
10. Driver, C.J.; Watts, V.; Bunck, A.C.; Van Ham, L.M.; Volk, H.A. Assessment of cerebellar pulsation in dogs with and without Chiari-like malformation and syringomyelia using cardiac-gated cine magnetic resonance imaging. *Vet. J.* **2013**, *198*, 88–91. [[CrossRef](#)] [[PubMed](#)]
11. Rusbridge, C.; Knowler, P. The Need for Head Space: Brachycephaly and Cerebrospinal Fluid Disorders. *Life* **2021**, *11*, 139. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
12. Cirovic, S.; Rusbridge, C. Slosh Simulation in a Computer Model of Canine Syringomyelia. *Life* **2021**, *11*, 1083. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
13. Rusbridge, C. Persistent scratching in Cavalier King Charles spaniels. *Vet. Rec.* **1997**, *141*, 179. [[PubMed](#)]
14. Limpens, C.; Smits, V.T.M.; Fieten, H.; Mandigers, P.J.J. The effect of MRI-based screening and selection on the prevalence of syringomyelia in the Dutch and Danish Cavalier King Charles Spaniels. *Front. Vet. Sci.* **2024**, *11*, 1326621. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
15. Jansma, M.R.; Heuvel, M.V.D.; Bossens, K.; Noorman, E.; Hermans, M.; Mandigers, P.J.J. Chiari-like Malformation and Syringomyelia in Pomeranians: A Longitudinal Study. *Vet. Sci.* **2025**, *12*, 677. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
16. Donner, J.; Freyer, J.; Davison, S.; Anderson, H.; Blades, M.; Honkanen, L.; Inman, L.; Brookhart-Knox, C.A.; Louviere, A.; Forman, O.P.; et al. Genetic prevalence and clinical relevance of canine Mendelian disease variants in over one million dogs. *PLoS Genet.* **2023**, *19*, e1010651. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
17. Kiviranta, A.M.; Rusbridge, C.; Lappalainen, A.K.; Junnila, J.J.T.; Jokinen, T.S. Persistent fontanelles in Chihuahuas. Part I. Distribution and clinical relevance. *J. Vet. Intern. Med. Am. Coll. Vet. Intern. Med.* **2021**, *35*, 1834–1847. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
18. Knowler, S.P.; McFadyen, A.K.; Rusbridge, C. Effectiveness of breeding guidelines for reducing the prevalence of syringomyelia. *Vet. Rec.* **2011**, *169*, 681. [[CrossRef](#)] [[PubMed](#)]
19. Santifort, K.M.; Carrera, I.; Mandigers, P.J.J. Longitudinal assessment of syringomyelia in Pomeranians. *Front. Vet. Sci.* **2024**, *11*, 1364464. [[CrossRef](#)] [[PubMed](#)]
20. Lewis, T.; Rusbridge, C.; Knowler, P.; Blott, S.; Woolliams, J.A. Heritability of syringomyelia in Cavalier King Charles spaniels. *Vet. J.* **2010**, *183*, 345–347. [[CrossRef](#)] [[PubMed](#)]
21. Akiyama, Y.; Koyanagi, I.; Yoshifuji, K.; Murakami, T.; Baba, T.; Minamida, Y.; Nonaka, T.; Houkin, K. Interstitial spinal-cord oedema in syringomyelia associated with Chiari type 1 malformations. *J. Neurol. Neurosurg. Psychiatry* **2008**, *79*, 1153–1158. [[CrossRef](#)] [[PubMed](#)]
22. R Foundation for Statistical Computing V, Austria. A Language and Environment for Statistical Computing. 2026. Available online: <https://www.R-project.org/> (accessed on 1 April 2026).
23. Venables, W.N.; Ripley, B.D. *Modern Applied Statistics with S*, 4th ed.; Springer: New York, NY, USA, 2010.
24. Parker, J.E.; Knowler, S.P.; Rusbridge, C.; Noorman, E.; Jeffery, N.D. Prevalence of asymptomatic syringomyelia in Cavalier King Charles spaniels. *Vet. Rec.* **2011**, *168*, 667. [[CrossRef](#)] [[PubMed](#)]
25. Cappello, R.; Rusbridge, C. Report from the Chiari-Like Malformation and Syringomyelia Working Group round table. *Vet. Surg.* **2007**, *36*, 509–512. [[PubMed](#)]
26. Maat, R.T.; Heusden, K.V.; Hoogervorst-Spek, L.; Plonek, M.; Mandigers, P.J.J. The Effectiveness of Amitriptyline and Gabapentin in Treating Pomeranians with Chiari-like Malformation and/or Syringomyelia. *Animals* **2025**, *15*, 992. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]

Disclaimer/Publisher’s Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.