Primary lens luxation in the Chinese Shar Pei: clinical and hereditary characteristics

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INTRODUCTION

Lens luxation in the dog may be classified as primary or secondary. Primary luxations, in which prior ocular disease cannot be identified, are commonly considered to be hereditary, and are thought to be due to weakened or aberrant lens zonules that rupture relatively early in life.1 This form of lens luxation tends to be more common than secondary luxations, which can occur from damage to the suspensory lenticular zonules subsequent to glaucoma, cataracts, uveitis, intraocular tumors or other ocular disease.1–6 Traumatic and congenital luxations are relatively rare in the dog, although trauma may precipitate luxation in a dog that is predisposed to the condition.1–5

Primary lens luxation in the adult dog has been reported in many breeds; predominantly the terrier breeds.1–3,5 Primary lens luxations have been reported in the Sealyham, Boston, Cairn, Jack Russell, Wheaten, Wire- and Smooth-haired Fox Terrier breeds.1,2,3,5,7,8 Additionally, primary luxations have been documented in the Tibetan Terrier9,10 and the Border Collie.4 The condition is often bilateral,1,2,4 has no sex predilection,1,4,7 and most commonly arises between 3 and 7 years of age.2,4,7

Primary lens luxations have been observed in young adult Chinese Shar Peis examined at the Virginia–Maryland Regional College of Veterinary Medicine Teaching Hospital (VMRCVM). The syndrome observed in these dogs follows a progressive clinical pattern, initially presenting with a unilateral lens luxation/subluxation in an otherwise disease-free, normotensive eye. Subsequent ocular hypertension ensues in the affected eye with attempts made to control the pressure rise using lens extraction (in the case of anterior luxation), varied regimens of antiglaucoma medications (miotics, topical beta blockers, systemic carbonic anhydrase

Abstract

A pedigree analysis of a family of 15 related Chinese Shar Peis was conducted. This pedigree analysis, including affected and nonaffected dams, sires and offspring, was compiled to document and characterize the occurrence, common clinical signs, and age of onset of primary lens luxation while suggesting a possible mode of inheritance in this breed. Of the five offspring from the mating of an affected dam to two unrelated affected males, 100% of offspring were affected with bilateral primary lens luxations. Of the four viable offspring from the mating of the same affected dam to an unrelated, unaffected male, two dogs (50%) were affected. The average age of onset of affected animals (seven) in this first generation was 4.9 years (range 3–6 years). The six dogs in the second generation of the same pedigree line were 2-years-old at examination with none of these animals affected at the time of this study. The most common presenting complaints were a unilateral change in ocular appearance (5 of 7 dogs) and subjective vision impairment (4 of 7 dogs). The most common clinical sign upon ophthalmic examination was iridodonesis (unilateral 4 of 7 dogs; bilateral 3 of 7 dogs) and the presence of an aphakic crescent (3 of 7 dogs). Gonioscopy and tonometry of severely affected eyes revealed a narrow or closed iridocorneal angle and ocular hypertension. This study suggests that primary lens luxation does occur in the Chinese Shar Pei, resembling the clinical condition (age of onset, clinical signs) previously described in the terrier breeds, the Border Collie, and the Tibetan Terrier. Application of the phenotypic findings in this study to a Mendelian genetic model of inheritance suggests that primary lens luxation in the Chinese Shar Pei is inherited as a simple autosomal recessive trait.

Key Words: primary lens luxation, Shar Pei, genetics, hereditary, dog
inhibitors), and/or cyclocryotherapy/Nd:YAG laser cycloablation. End results of glaucoma treatment varied from maintenance of normal to slightly elevated intraocular pressures with or without vision, to phthisical nonvisual eyes. In most cases, within 8–12 months, the previously normal, contralateral eye becomes affected, following a similar course and end result. Moreover, this clinical entity subjectively appeared to manifest itself at a high frequency within families of related Chinese Shar Pei dogs with affected dogs in their ancestral lineage. This consistent clinical syndrome involving primary lens luxation and subsequent glaucoma suggests a hereditary pattern, warranting an investigation into its etiology, characterization and mode of inheritance as has been conducted in other dog breeds showing a similar predisposition towards primary lens luxation.\(^3\),\(^4\),\(^8\),\(^10\)

The etiology of primary lens luxation in the dog is an issue of some debate. Many feel that an inherent weakness of the suspensory lens zonules predisposes the dog to the condition. It has also been suggested that the pathogenesis of primary lens luxation in the dog is associated with an inflammatory process of the lens zonule.\(^6\),\(^12\) However, it remains unclear as to whether a zonular degeneration (of noninflammatory origin) or primary structural weakness stimulates a subsequent inflammatory response, or whether inflammation itself results in zonular degeneration. Additional investigation into the etiology using scanning and transmission electron microscopic studies in the Tibetan Terrier revealed an abnormal arrangement of zonular fibrillar insertion onto the posterior lens capsule, representing a mechanically inferior suspensory apparatus, and suggested this mechanical anomaly as a direct cause for primary lens luxations in that breed.\(^9\) In the Tibetan Terrier, primary lens luxations appear to be inherited via a Mendelian simple autosomal recessive mode of inheritance.\(^9\),\(^10\) There is currently scarce evidence to prove an identical mode of transmission in other breeds. Genetic studies generally require examination of a large population of related animals in order to characterize the disorder (age of onset, characteristic appearance, rate of progression) and to define the mode of inheritance.\(^11\) However, in a clinical setting, related animals are frequently unavailable for examination once a heritable disorder is suspected or identified in an individual animal. Maintaining a large population of dogs for controlled breeding trials through several generations is a laborious and expensive process which is exacerbated by the fact that many ocular conditions do not develop until adulthood. Alternatively, pedigree analysis of a related line of animals could simplify the investigational process, shedding light on the inheritance pattern without the time and financial expense. The purpose of this study was to perform a pedigree analysis on a related family of Chinese Shar Peis to document and characterize the occurrence, common clinical signs and age of onset of primary lens luxation while suggesting a possible mode of inheritance in this breed.

**MATERIALS AND METHODS**

**Record examination**

The records of 55 Chinese Shar Peis examined by the ophthalmology service at the Virginia–Maryland Regional College of Veterinary Medicine Teaching Hospital (VTVMTH) between 1990 and 1996 were reviewed. Eleven of the 55 Chinese Shar Peis were recorded as affected with primary lens luxations. All initial and follow-up ophthalmic examinations were performed by one of two board-certified veterinary ophthalmologists (J.P.P., E.S.C.), although most dogs were examined by both ophthalmologists at some time during return visits to the VMRCVM. Diagnosis of lens luxation was based on the presence of clinical signs including: iridotonia, altered anterior chamber depth, presence of an aphakic crescent, the presence of an anteriorly or posteriorly displaced lens and/or focal corneal opacification (due to anterior lens luxation and endothelial contact). The primary nature of the luxation was suspected by clinical examination, combined with the absence of physical evidence or history supporting prior ocular disease. Animals seen in the early stages of the syndrome were normotensive and not buphthalmic, but subsequently developed ocular hypertension presumably as the iridocorneal angle became obstructed due to inflammation, the anteriorly displaced lens, or due to primary progressive filtration angle narrowing/closure.

Of the 11 affected Shar Peis, two were related as dam (F\(_{A1}\)) and offspring (generations S\(_0\) and S\(_1\)) (see Fig. 1), whereas the remaining nine animals were unrelated and hence not included in the pedigree analysis. By employing official breeding pedigree documentation supplied by the owners and breeders, the lineage of the affected dam (F\(_{A1}\)) was traced to include all related and unrelated dogs (sires and offspring) for examination and inclusion in the pedigree analysis. This pedigree analysis (see Fig. 1) of the affected dam (F\(_{A1}\)), was traced to matings with three unrelated sires (no common ancestry for four or more generations), yielding 10 offspring (litters 1, 2 and 3). One of the female offspring (generation S\(_1\)) was mated to yet another unrelated male, yielding six offspring (generation S\(_2\), litter no. 4). All offspring were given a complete physical and ophthalmic examination by the author(s) with the permission of the owners. If applicable, the subject’s local veterinarian was contacted with questions regarding prior ocular disease and previous pertinent medical/surgical problems, if any. Parents of the affected dam were not available for ophthalmic examination by the authors.

**Clinical examination**

The eyes of all dogs were examined by slit-lamp biomicroscopy and indirect ophthalmoscopy. Pupillary dilation, if necessary, was achieved with 1% tropicamide (Mydriacyl, Alcon) to facilitate fundic examination. Schirmer tear test and fluorescein stainings were performed in all dogs, as was tonometry with an electronic applanation tonometer (Tono-Pen XL, Bio-Rad). Gonioscopy, using an 18-mm Koeppe lens (Ocular Instruments, Inc.), was also performed. Animals were
considered to be affected with primary lens luxations if they:
(i) exhibited unilateral or bilateral lens luxation or iridodonesis, with no signs of current or prior ocular hypertension (i.e. buphthalmos or fundus/cornea/iris changes) or other predisposing ocular disease; (ii) exhibited unilateral or bilateral lens luxations or iridodonesis, with ocular hypertension but no buphthalmia; (iii) exhibited bilateral lens luxations or iridodonesis, with unilateral ocular hypertension.

Scanning electron microscopy
The lenses of the original dam (F A1) and offspring (M A1) were fixed in neutral buffered formalin fixative, washed with 0.1 M sodium phosphate buffer at pH 7.4, postfixed in 1% osmium tetroxide in 0.1 M phosphate buffer for 1 h, and washed again in 0.1 M phosphate buffer. The tissues were dehydrated in increasing ethanol concentrations, critically point dried, mounted on stubs with silver paint, sputter coated with \( \approx 1000 \text{ Å gold and viewed in a JEOL JSM-35C scanning electron microscope.} \) Sections were viewed at 15 KV at 1000 \( \times \), 7200 \( \times \), and 15 000 \( \times \). The lenses were examined over the full 360° circumference of the equator.

RESULTS
Of the five offspring from the mating of the affected dam (F A1) to two unrelated affected males (M A1 and M A2), all offspring were found to be affected with primary lens luxations. Of the five offspring from the mating of the affected dam (F A1) to an unrelated, unaffected male (M NA1), one male dog died at 3 years of age with no evidence of vision problems at the time of death, two dogs were affected at the time of this study and two dogs were unaffected. The average age of onset of affected animals in the S1 generation was 4.9 years (range 3–6 years; see Table 1). The age of dogs in the S2 generation was 2 years and none of these animals were affected at the time of the study. The most common complaint presented by the owner in affected animals was a unilateral change in ocular appearance (5 of 7 dogs) and subjective vision impairment (4 of 7 dogs). Those owners reporting bilateral change in ocular appearance reported an approximate 12–18-month lag period in ocular changes between eyes. The most common clinical sign upon ophthalmic examination was iridodonesis (unilateral 4 of 7 dogs; bilateral 3 of 7 dogs) and the presence of an aphakic crescent (3 of 7 dogs). Of the 10 affected eyes (in seven dogs), eight eyes were visual and two eyes were clinically blind as evidenced by lack of a dazzle response, menace response, and direct/consensual pupillary light responses. Optic nervehead degeneration and peripapillary retinal degeneration was noted in both blind eyes and in four of the eight visual eyes.
Scanning electron microscopy (SEM) of the affected lenses revealed reduced numbers of zonular fragment attachments to the lens equator as well as several ruptured zonular fragments. Moderate inflammation with multiple lymphocytes and activated macrophages was found on the surface of the lenticular zonules (see Figs 2, 3, 4, and 5).

Clinically, the syndrome observed in the FÃ1-related and the nine nonrelated Chinese Shar Peis varied somewhat between animals but, in general, it exhibited similar chronological progression and clinical signs. The chronological progression of the condition in the affected dam’s (FÃ1) offspring was difficult to assess in two cases as the owners did not seek veterinary counsel until the condition had advanced to severe clinical disease, exhibiting buphthalmos, severe ocular hypertension and loss of vision. In these cases, diagnosis of primary lens luxation was made upon examination of the contralateral eye, previously thought to be unaffected by the owner. All affected animals in this pedigree exhibited iridodonesis, while only three exhibited an actual aphakic crescent on presentation. All affected eyes in the early stages of the disease had normal intraocular pressures, with two dogs exhibiting bilateral iridodonesis with unilateral ocular hypertension.

In the nine Chinese Shar Peis examined at the VMRCVM that were not included in the pedigree analysis (i.e. not FÃ1, affected dam of 1st generation; FÃNA, female, not affected; MÃ, male, affected; MÃNA, male, not affected; ID, iridodonesis; n-IOP, normal intraocular pressure; B, blind; AC, aphakic crescent; OH, ocular hypertension; V, visual; OU, both eyes; OD, right eye; OS, left eye; FCO, focal corneal opacification; LAC, lens in anterior chamber; Age aff., age at which owners recognized a problem with their dog’s vision (i.e. bumping into walls, ‘marble’ in eye, cloudiness; years); Age TOS, Age at the time of the study (years). Affected, affected with primary lens luxation.

Table 1 Offspring of affected dam (FÃ1) over two successive generations recording owner’s reporting of age affected, age at the time of study, status (affected vs. not affected) and clinical signs

<table>
<thead>
<tr>
<th>Offspring</th>
<th>Mating</th>
<th>Age aff.</th>
<th>Age TOS</th>
<th>Status</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>FÃ1 × MÃA1</td>
<td>5.5</td>
<td>7</td>
<td>Affected</td>
<td>ID OU, AC OS, n-IOP OU, V OU</td>
</tr>
<tr>
<td>2</td>
<td>FÃ1 × MÃA1</td>
<td>6</td>
<td>7</td>
<td>Affected</td>
<td>ID OU, OH OD, B OD, V OS</td>
</tr>
<tr>
<td>3</td>
<td>FÃ1 × MÃA2</td>
<td>4</td>
<td>5</td>
<td>Affected</td>
<td>ID OS, n-IOP OU, V OU</td>
</tr>
<tr>
<td>4</td>
<td>FÃ1 × MÃA2</td>
<td>5</td>
<td>5</td>
<td>Affected</td>
<td>ID OU, n-IOP OU, V OU</td>
</tr>
<tr>
<td>5</td>
<td>FÃ1 × MÃA2</td>
<td>3</td>
<td>5</td>
<td>Affected</td>
<td>ID OU, n-IOP OS, B OD</td>
</tr>
<tr>
<td>6</td>
<td>FÃ1 × MÃNA1</td>
<td>5</td>
<td>6</td>
<td>Affected</td>
<td>ID OS, AC OS, LAC OD, OH OD, B OD</td>
</tr>
<tr>
<td>7</td>
<td>FÃ1 × MÃNA1</td>
<td>6</td>
<td>6</td>
<td>Affected</td>
<td>ID OD, OH OS, V OU</td>
</tr>
<tr>
<td>8</td>
<td>FÃ1 × MÃNA1</td>
<td>N/A</td>
<td>6</td>
<td>Not affected</td>
<td>N/A</td>
</tr>
<tr>
<td>9</td>
<td>FÃ1 × MÃNA1</td>
<td>N/A</td>
<td>6</td>
<td>Not affected</td>
<td>N/A</td>
</tr>
<tr>
<td>10</td>
<td>FÃ1 × MÃNA1</td>
<td>N/A</td>
<td>N/A</td>
<td>Died (3 years old)</td>
<td>N/A</td>
</tr>
<tr>
<td>11</td>
<td>FÃNA × MÃA2</td>
<td>N/A</td>
<td>2</td>
<td>Not affected</td>
<td>N/A</td>
</tr>
<tr>
<td>12</td>
<td>FÃNA × MÃA2</td>
<td>N/A</td>
<td>2</td>
<td>Not affected</td>
<td>N/A</td>
</tr>
<tr>
<td>13</td>
<td>FÃNA × MÃA2</td>
<td>2</td>
<td>2</td>
<td>Not affected</td>
<td>N/A</td>
</tr>
<tr>
<td>14</td>
<td>FÃNA × MÃA2</td>
<td>N/A</td>
<td>2</td>
<td>Not affected</td>
<td>N/A</td>
</tr>
<tr>
<td>15</td>
<td>FÃNA × MÃA2</td>
<td>N/A</td>
<td>2</td>
<td>Not affected</td>
<td>N/A</td>
</tr>
<tr>
<td>16</td>
<td>FÃNA × MÃA2</td>
<td>N/A</td>
<td>2</td>
<td>Not affected</td>
<td>N/A</td>
</tr>
</tbody>
</table>

FA1, affected dam of 1st generation; FÃNA, female, not affected; MÃ, male, affected; MÃNA, male, not affected; ID, iridodonesis; n-IOP, normal intraocular pressure; B, blind; AC, aphakic crescent; OH, ocular hypertension; V, visual; OU, both eyes; OD, right eye; OS, left eye; FCO, focal corneal opacification; LAC, lens in anterior chamber; Age aff., age at which owners recognized a problem with their dog’s vision (i.e. bumping into walls, ‘marble’ in eye, cloudiness; years); Age TOS, Age at the time of the study (years). Affected, affected with primary lens luxation.

Figure 2. Scanning electron micrograph of the equator of a luxated lens showing disrupted zonular attachments (× 220).
related to the $F_A$ dam), the condition typically followed a similar clinical progression: unilateral to bilateral lens subluxation/luxation, subsequent ocular hypertension, antiglaucoma therapy (lens extraction, antiglaucoma medications and/or cyclocryotherapy/Nd:YAG laser cycloablation) with resultant maintenance of normal to slightly elevated intraocular pressures (with or without vision), or phthisical nonvisual eyes. The time lag between demonstration of affected eyes in each dog varied between 1 to 8 months. Of the 11 eyes (nine Shar Peis) in this group of dogs not included in the pedigree analysis, all 11 demonstrated ocular hypertension at some point prior to antiglaucoma therapy. Seven eyes were recorded as being blind at the time of this study. The most common clinical signs at the time of presentation to the VMRCVM were iridodonesis (11 of 11 eyes) and the presence of an aphakic crescent (six of 11 eyes). The most common owner complaint on presentation was subjective vision impairment and ocular cloudiness.

**DISCUSSION**

This study suggests that primary lens luxation does occur in the Chinese Shar Pei, appearing identical to and consistent with the simple autosomal recessive condition previously characterized in the Tibetan Terrier.$^9,10$ Following from this study, as well as anecdotal reports of lens luxations and

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**Figure 3.** Scanning electron micrograph of lenticular zonules illustrating inflammatory cell adherence to individual zonular fibers (× 1000).

**Figure 4.** Scanning electron micrograph of lenticular zonules illustrating multiple inflammatory cell adherence to individual zonular fibers (× 7200).
sudden blindness in young adult Shar Peis from veterinarians polled in the Virginia, Maryland (N. M. Bromberg, personal communication), and Chicago areas, it is the authors’ opinion that this condition is not uncommon. Of the total population of Shar Peis presented to the ophthalmology service at VMRCVM, ≈ 20% exhibited primary lens luxations. The high frequency of primary lens luxations observed when affected animals were bred supports a heritable component to the condition. Indeed, when two dogs affected with primary luxations were bred, as was the case with two distinct matings in the examined pedigree (see Fig. 1, mating nos 1 and 2), 100% of the resulting offspring were affected. Fifty per cent of the offspring resulting from an affected x presumed carrier mating were also affected with the syndrome. Application of these findings to a Mendelian genetic model for inheritance suggests that primary lens luxation in the Chinese Shar Pei is inherited as an autosomal recessive trait. This suggestion is limited by the assumption that the phenotypically normal male (mating no. 3) was indeed a heterozygote for the ‘luxation gene’. However, due to obvious limitations inherent in studying a group of privately owned animals, it was not feasible, or desired by the owners or authors, to evaluate this assumption by a second test crossing of the phenotypically normal, presumed heterozygote to a phenotypically affected animal (presumed homozygote recessive) to assess its carrier status. Such a project would require a group of animals bred solely for genetic study and was beyond the scope of this study. Another possibility is that the trait is of X-linked inheritance. The results of this pedigree analysis could also support a sex-linked inheritance pattern. It is difficult to speculate on the genotype of animals in the S2 generation (from mating no. 4). Phenotypically they are unaffected, yet this observation is somewhat expected as the average age of onset in affected animals was 4.9 years and the S2 generation offspring were 2 years of age at the time of the study. Yet, if they are to develop primary luxations in early adulthood, the probability of doing so would depend heavily upon the genotype of the paternal male, as the maternal parent would be a carrier for the ‘luxation gene’, having inherited the recessive allele from her own maternal parent (generation S0). If the sire (mating no. 4) was a carrier, one would expect 25% of the generation S2 offspring to be homozygous recessive. However, if the male was unaffected and homozygous dominant, all offspring would be phenotypically normal, but approximately 50% would be heterozygous for the recessive gene.

The genetic aspects of primary lens luxation in the Tibetan Terrier have been extensively investigated, with strong evidence to confirm a Mendelian simple autosomal recessive mode of inheritance. Results obtained in this study would strongly suggest that the condition in the Chinese Shar Pei may exhibit a simple autosomal mode of inheritance as well, with the homozygous recessive genotype conferring primary lens luxation. Recessive sex linkage as a mode of inheritance for primary lens luxations in the Shar Pei is unlikely, as linkage would not allow a homozygous recessive (affected) female bred with a phenotypically normal male (as seen in mating no. 3) to produce, under normal circumstances, a phenotypically affected female homozygous for the luxation, as was produced in this mating. Previous studies have shown, however, a predilection for primary lens luxation in male dogs. A preponderance of males affected in this study may indicate a greater propensity for luxations in males or, more probably, a reflection of a small sample size.

The presenting and subsequent clinical signs of lens luxation in the Chinese Shar Pei reported here are consistent with published descriptions of primary lens luxation in the terrier breeds, the Tibetan Terrier, and the Border Collie. The most common presenting complaints by owners of the subject animals were vision impairment and
focal corneal edema. Other observable changes observed by the owners included redness, pain, photophobia, and a ‘marble-like’ structure appearing in the eye. The age of onset of such changes occurred between 3 and 6 years of age (average 4.9 years), which is consistent with previous reports of such changes occurring in the Tibetan Terrier \(^9,10\) and Border Collie. \(^4\) Although primary luxations are usually unilateral, \(^1,2,8,9,15\) the majority of Shar Pei owners reported only unilateral ocular changes on presentation. However, upon ophthalmic examination of the reportedly normal eye, subtle evidence of subluxation, including iridodonesis and/or mild anterior vitreous prolapse, was noted. In addition, some owners reported an approximate 12–18-month lag time in observable changes between eyes, suggesting that zonular breakdown was a process that occurred over a prolonged period of time. This is similar to observations noted in previous studies of the Tibetan Terrier \(^9,10\) in which clinical evidence (mild iris tremor and lens mobility signaling impending luxation), was observed at least 12 months before zonular rupture and true luxation occurred.

In most cases, gonioscopy and tonometry of severely affected eyes revealed a narrow or closed iridocorneal angle and ocular hypertension (precluding cases where the eye was phthisical, presumably having suffered from ciliary body damage secondary to prolonged and severe ocular hypertension). These changes could be secondary to degenerative changes from the primary lens luxation, but may also be due to a concurrent primary angle closure disorder. Some ophthalmologists have proposed an entity of primary angle closure glaucoma in this breed, but it is beyond the scope of this article to address that clinical entity.

The tendency to develop secondary glaucoma in dogs suffering from lens luxation is well documented in other breeds. \(^2,3,9,10\) however, the normal intraocular pressure findings in eight eyes affected with lens luxations (two having anterior displacement of vitreous) in this study provides additional testimony that these luxations were indeed primary as opposed to secondary to ocular hypertension. Presumably, if allowed to proceed, inflammation and vitreous obstruction of the iridocorneal angle could lead to ocular hypertension and subsequent blindness. Aside from published reports documenting the existence of primary lens luxation in the Tibetan Terrier and the Border Collie, there has been scarce evidence to suggest that this condition occurs regularly in nonterrier breeds. The documentation of primary lens luxation in Chinese Shar Peis, a nonterrier breed not related to the Tibetan Terrier or the Border Collie, is of particular concern to practicing veterinarians in that this blinding condition may be evident in other canine populations.

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