History

A 6-month-old sexually intact male Boxer was brought to our facility for castration. The dog had been examined at 2, 3, and 4 months of age and had a palpable right testis located in the scrotum but no palpable left testis during any of the examinations. A presumptive diagnosis of unilateral, abdominal cryptorchidism was made at the examination conducted when the dog was 4 months old.

Another examination was performed prior to administration of preoperative medications on the day of surgery, which again revealed a palpable right testis in the scrotum but no evidence of a left testis. The dog was then prepared for routine exploratory abdominal celiotomy and castration via a prescrotal incision. After anesthesia was induced, a ventral abdominal incision was made to the left of the prepuce that extended toward the midline at the umbilicus. A thorough exploration of the abdominal cavity did not reveal the left testis, ductus deferens, or epididymus. The left kidney, left ureter, and bladder had a normal appearance. The celiotomy was closed by use of a routine 3-layer closure technique. Routine techniques via a prescrotal incision were used to remove the right testis, and it was not submitted for histologic examination. The dog recovered from anesthesia without complications.

Question

What additional diagnostic tests should be performed to support a diagnosis of unilateral anorchidism (ie, monorchidism)? Please turn the page.

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>GnRH</td>
<td>Gonadotropin-releasing hormone</td>
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<tr>
<td>hCG</td>
<td>Human chorionic gonadotropin</td>
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<td>LH</td>
<td>Luteinizing hormone</td>
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Answer
Ultrasoundography or magnetic resonance imaging of the inguinal canal and abdomen, a GnRH or hCG stimulation test, and measurement of serum concentrations of LH and follicle-stimulating hormone.

Results
When the dog was 9 and 12 months old, a GnRH stimulation test was performed by IM administration of 50 µg of GnRH. Blood samples were collected before (baseline) and 60 minutes after injection; serum was harvested and used to measure testosterone concentrations. Testosterone concentrations were < 0.04 ng/mL before and after GnRH administration for tests conducted at both 9 and 12 months of age.

When the dog was 14 months old, a complete abdominal ultrasonographic examination was performed. The examination revealed no evidence of testicular tissue in the abdomen or inguinal region. The prostate was small, with measurements of $14 \times 9 \times 9$ mm ($0.551 \times 0.354 \times 0.354$ inches).

Another GnRH stimulation test (50 µg, IM) was performed when the dog was 16 months old. Blood samples were collected before (baseline) and 60 and 90 minutes and 24 hours after GnRH administration. Serum concentrations of testosterone were < 0.04 ng/mL in all samples.

Serum concentrations of LH were measured when the dog was 16 months old; the test yielded a strong positive result. Magnetic resonance imaging, an hCG stimulation test, and measurement of serum concentrations of follicle-stimulating hormone were not performed.

Discussion
We believe the results of the diagnostic tests performed in the dog reported here strongly supported a diagnosis of unilateral anorchidism (ie, monorchidism). In 1 report, a sexually intact male dog injected with 50 µg of GnRH had an immediate and substantial increase in the LH concentration, which resulted in increases in serum testosterone concentrations that were higher than the concentrations at baseline for several days. Furthermore, serum LH concentrations do not increase above the reference range in patients with a unilateral testis (ie, monorchidism).  

Unilateral anorchidism or monorchidism is defined as the lack of one of the testes. It has been described most commonly in humans, horses, and domestic cats. To our knowledge, it has not been reported in dogs, with most authors simply describing it as an extremely rare condition.

Little research has been devoted to determining the cause of monorchidism, especially in domestic animals. Most reports are in human medical journals. It is believed that monorchidism in humans is most commonly caused by ischemia of the testis as a result of intrauterine testicular torsion, which makes it a syndrome of testicular regression. Monorchidism is often associated with a blind-ended vas deferens or with the vas deferens ending in an ill-defined area of blood vessels (ie, vanishing testis). Results of histologic examination of remnants in these cases are consistent with atrophy secondary to ischemia, which supports testicular regression as the cause. It is also possible to have a complete absence of a testis, epididymus, and vas deferens. These cases may be more appropriately classified as testicular agenesis attributable to an unknown cause.

Monorchidism has been reported in horses and is believed to be a congenital condition. Most of the affected horses had 1 testis (usually the right testis) in the scrotum and a blind-ended vas deferens with no epididymus. It is believed that most of these cases were caused by testicular degeneration, which is similar to the condition in humans. Monorchidism in at least 1 horse is believed to have been attributable to testicular agenesis.

A dog suspected of being a monorchid in the basis of findings during exploratory laparotomy warrants a thorough diagnostic evaluation. Monorchidism is believed to be extremely rare in dogs, and we believe the dog described here is the first reported incidence of this condition. Anecdotally, other dogs with monorchidism (on the basis of findings during exploratory laparotomy) have had positive responses to administration of hCG or GnRH. It is important to remember that any increase in testosterone concentration from the concentration at baseline after administration of hCG or GnRH should be considered a positive response. Additional exploratory surgery or evaluation by magnetic resonance imaging would then be indicated. However, we are unaware of any published reports in which magnetic resonance imaging was used to identify retained testicular tissue in any species.

Outcome
The dog subsequently developed urinary incontinence; cause of the incontinence was unknown. The incontinence was responsive to administration of phenylpropanolamine.

References