Incontinence in Boxer

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HISTORY AND PHYSICAL EXAM

Samson was a 4-year old MC boxer presented with a 1 month history of incontinence and abnormal urination. Samson’s owners had noted that he was leaving small puddles of urine on his beds when he rested or slept, and that they occasionally found dribbles of urine on the floor as well. When taken outside, Samson assumed a normal leg-lifted posture to urinate and initiated a urine stream that was swiftly attenuated. He often stood in the same position without straining but without producing a urine stream for several minutes, and if permitted, would repeat this behavior several times. With the exception of the incontinence and abnormal urination behavior, Samson’s behavior, appetite, activity, and general well-being were normal and unchanging according to the owners.

Physical examination was normal with the exception of a large bladder which could not be expressed with firm pressure. Neurological examination was normal. When taken out to urinate, Samson postured normally and produced a small urine stream that was attenuated after about 5 seconds. He held the urination posture without straining for a minute, and then repeated this process twice more. After urination attempts, the bladder was still large.

PROBLEM LIST AND INITIAL DIFFERENTIALS

1. Urine retention: inadequate detrusor contraction (neurogenic or myogenic), inadequate sphincter relaxation, mechanical obstruction of outlet
2. Incontinence: In the face of pathological urine retention, incontinence is de facto due to overflow (bladder hypoaccommodation, urethral irritation, polyuria with overflow, anatomical anomaly at outflow, lower motor neuron lesion are other differentials that could possibly contribute, but are unlikely to be primarily responsible).

INITIAL PLAN

1. CBC/chemistry panel: WNL
2. Urinalysis: color = light yellow/brown, USG = 1.026, 3+ blood, 0-3 WBC/hpf, 5-10 RBC/hpf, 3_ amorphous debris
3. Urine culture: no growth in 72 hours
4. Urethral catheterization: no mechanical obstruction detected; after urination attempts, 800 ml urine removed from bladder
5. Abdominal radiographs: large bladder, normal shape and positioning, no evidence of urinary stone disease
6. Abdominal ultrasound exam: bladder is small to medium-sized with normal wall and no apparent prostatic or urethral abnormality
7. Retrograde and voiding urethrogram: normal urethral distension, and no intraluminal space-occupying lesion seen with retrograde study; marked bladder compression resulted in intermittent and incomplete voiding, with inadequate distension of the proximal urethral lumen/ proximal urethral narrowing
ASSESSMENT

When incontinence occurs in the face of significant urine retention, it is generally (and by definition) overflow incontinence. This does not rule out other contributing factors such as anatomical abnormality of the outflow tract (e.g., a trigonal tumor could both cause outflow obstruction and interfere with normal closure function). In this case, mechanical obstruction was ruled out with catheterization, contrast study, and ultrasound, leaving functional obstruction (inadequate outlet opening/relaxation) as the most likely differential for this dog’s urine retention. In normal voiding, the stimulus for urethral contraction is inhibited, permitting urethral relaxation and urine outflow. Our observations of this dog’s voiding pattern suggest dyssynergic voiding, a condition in which the urethra contracts instead of relaxes in response to detrusor contraction (in theory, usually due to a subtle supra-sacral spinal lesion). The end result of this is inadequate bladder emptying, with eventual development of detrusor atony. In general, no more than 0.5-1.0 ml urine should remain in the bladder following full voiding attempts, so the 800 ml removed from this dog is radically abnormal. This is a disease seen almost exclusively in intact and castrated male dogs, usually large to giant breeds, and often in the young. In intact dogs, exacerbation of signs is often seen with psychological or sexual excitation.

Definitive documentation of detrusor-sphincter dyssynergia requires urethral pressure profilometry performed simultaneously with cystometrography to show simultaneous urethral and detrusor contraction. Practically speaking, we diagnose it presumptively based on observation of micturition and exclusion of other differentials, as above.

TREATMENT

Therapy for dyssynergia consists of relaxation of the proximal urethra and bladder emptying. Most dogs with dyssynergia have some degree of bladder atony at the time of diagnosis, and initial therapy should include hospitalization with a urinary catheter and closed collection system for 3-5 days to help start detrusor recovery. Bladder expression is not an adequate or appropriate means of urinary care in these dogs, and owners often will have to cleanly catheterize and empty the dog’s bladder at home 2-3 times daily during initial therapy.

Contraction of the proximal urethra occurs via stimulation of alpha-1 adrenergic-innervated smooth muscle, so relaxation of the proximal urethra is achieved via alpha-1 inhibition. Several medications are available for this purpose:

- **Phenoxybenzamine:** Non-selective alpha 1-alpha 2 antagonist; less potent than alternatives, with greater risk of side-effects; obsolete in human medicine for urinary applications. There is no reason at this point to use phenoxybenzamine over alternatives for urinary purposes.
- **Prazosin:** Selective alpha-1 antagonist; very inexpensive; useful in both cats and dogs. Can measurably decrease blood pressure, so not recommended in hypovolemic or hypotensive animals.
- **Tamsulosin (Flomax):** Potent, uroselective alpha-1 antagonist; does not decrease blood pressure at normal doses; not shown to be safe in cats yet; more expensive than prazosin.

Samson’s owners could not afford hospitalization, but agreed to catheterize him at home 3-4 times daily initially, then 2-3 times daily as needed. For financial reasons, they elected to initially try prazosin at 1 mg/15 kg q 8 hrs. This improved Samson’s voiding, but did not normalize it, and his residual urine volume stayed >300 ml. After 2 weeks the owners switched Samson to tamsulosin at 0.125 mg/10 kg q day. Samson’s residual urine volume steadily decreased and the owners were able to stop catheterizing him after 2 additional weeks. Eighteen months later he is still voiding normally on daily tamsulosin, but relapses after a few weeks if the medication is discontinued.