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SARD, adrenal activity, and hormone replacement in three dogs — a retrospective study

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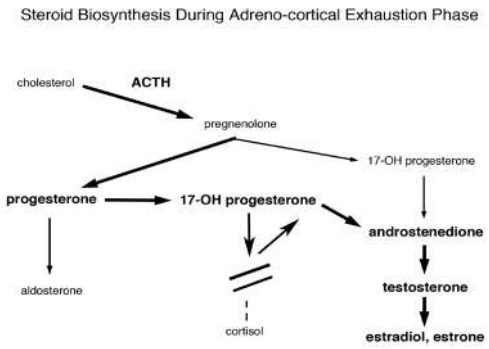
Purpose. To describe the clinical and laboratory findings, hormone replacement therapy and outcome of three dogs with Sudden Acquired Retinal Degeneration (SARD). **Methods.** Data were collected retrospectively from the general practice charts of one female and two male dogs (all castrated) with persistent signs of PU, PP, lethargy, confusion, agitation/panting, aggression and obesity. Mean age = 11 years. An endocrine/immune (E&I) panel (National Veterinary Diagnostic Services, Quail Valley, CA) was performed for all dogs. Mean time interval from SARD onset = 4.58 months. Range = 4.0–5.75 months. Initial E&I panels identified low levels of immunoglobulins (IgA, IgG and IgM), low cortisol and elevated estrogen in all dogs. T3 and T4 fell within the bottom 28% of normal range. General practice veterinarians initiated hormone replacement with triamcinolone acetonide injectable glucocorticoid (Fort Dodge Laboratories, Overland Park, KS or Bristol-Myers Squibb, Princeton, NJ) followed by low-dose oral methylprednisolone (Vintage Pharmaceuticals, Charlotte, NC or Pharmacia, Kalamazoo, MI) and oral levothyroxine (Lloyd, Inc., Shenandoah, IA). E&I panels were repeated between 1.0–5.5 months. Mean interval = 3.5 months. **Results**. All dogs demonstrated a shift toward normal immunoglobulin, estrogen and cortisol levels. T3 and T4 rose toward the mid-normal range in all dogs. Clients reported full resolution in 43% (mean) of clinical signs and “some improvement” in 36% (mean).

Sex	Initial cortisol	Repeat cortisol	Normal cortisol	Initial estrog.	Repeat estrog.	Normal estrogen	Initial IgA / IgG / IgM	Repeat IgA / IgG / IgM	Normal range IgA / IgG / IgM
M	0.52	1.16	1.00-2.50 ug/dL	25.40	24.28	**Male:** 20.00-25.00 pg/ml	43 / 817 / 84	78 / 1353 / 127	**IgA** 70-170 **IgG** 1000-2000 **IgM** 100-200 mg/dL
F	0.54	0.68		35.19	35.12	**Female:** 30.00-35.00 pg/ml	49 / 989 / 96	56 / 1049 / 102	
M	0.48	1.29		25.32	25.11		43 / 781 / 80	63 / 1106 / 114	

Conclusion. These dogs demonstrated concurrent levels of elevated estrogen and low cortisol—a pathological pattern of steroidogenesis described as adrenal exhaustion.
Clinical signs were associated with hyperestrogenism rather than hypercortisolism. Low-dose glucocorticoid and thyroid hormone replacement had a positive effect on both clinical presentation and laboratory findings. Supported by Lantern Publications. **None.**



SARD, adrenal activity, and hormone replacement – retrospective study

Caroline Levin, Lantern Publications, Oregon City, OR

PURPOSE

Describe the clinical and laboratory findings, hormone replacement therapy, and outcome of three dogs with Sudden Acquired Retinal Degeneration (SARD).

ANIMALS STUDIED

- **4–6 months** post SARD-onset
- **Male = 2, female = 1, mean age = 11 years**
- Diagnostic findings:
  - elevated estrogen
  - low cortisol
  - low-normal T3, T4
  - low immunoglobulins

(Endocrine & immunology panel, National Veterinary Diagnostic Services, Good Valley, CA)

METHODS

General practice veterinarians initiated hormone replacement therapy to augment low cortisol and low-normal thyroid levels:

triamcinolone acetonide	IM single dose	0.13 mg–0.27 mg/kg
methylprednisolone	PO sid	0.08 mg–0.17 mg/kg
levothyroxine	PO bid	0.01 mg–0.02 mg/kg

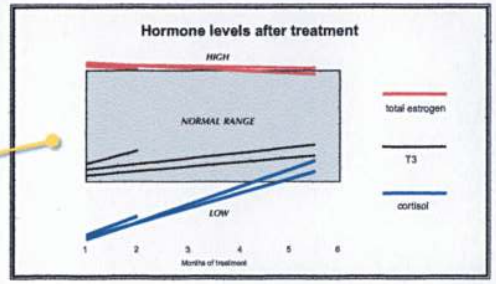
Endocrine & immunology panels were repeated. Range = 1.0–5.5 months.

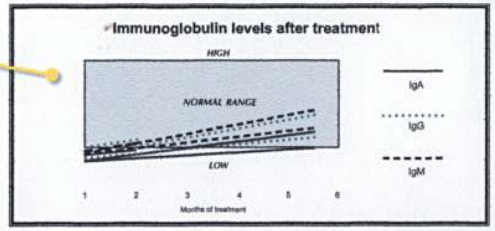
RESULTS

- All dogs demonstrated a shifted toward normal estrogen, cortisol, thyroid and immunoglobulin levels.
- Owners reported complete resolution in 43% (mean) of adrenal signs and "some improvement" in 36% (mean).

DISCUSSION

Clinical signs of hypercortisolism are a common feature of SARD. Early on, researchers speculated that this was the physiological response to stress[1]. Elevated adrenal sex hormones have also been reported within the first year of SARD.[2] One explanation for this pattern is Selye's model of stress adaptation which describes the progression from adrenal gland hyperactivity (hypercortisolism) to adrenal gland exhaustion (hypocortisolism).





Clinical presentation:		lethargy	obesity	confusion	PU/PD	PP	aggression	hirsutism	GI upset	pacing/ panting
	Initial	X	X					X		X
	With treatment	RESOLVED	RESOLVED					RESOLVED		RESOLVED
	Initial	X			X	X	X		X	X
	With treatment	RESOLVED			—	—	RESOLVED		IMPROVED	IMPROVED
	Initial	X		X	X	X				
	With treatment	IMPROVED		IMPROVED	—	IMPROVED				

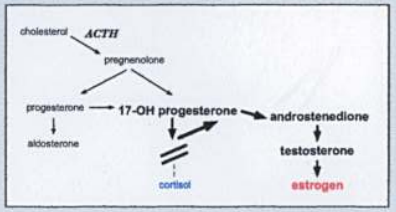
The stress response:

Alarm phase — normal response to stressors — HPA activity / cortisol secretion resolve when the stressor resolves.

Resistance phase — follows a prolonged period of stress. Cortisol production is slightly elevated, desensitizing the HPA feedback loop. Cortisol production continues unabated.[3]

Exhaustion phase — cortisol production fails. Precursor hormones accumulate and shift to sex-hormone production. *The dogs described here developed adrenal exhaustion within 4–6 months of blindness.*

Adrenal sex-hormone production during exhaustion phase



- Elevated estrogen mimics clinical signs of elevated cortisol including: PU/PD, lethargy, depression, confusion, agitation, seizures, bone marrow and immunoglobulin suppression. thyroid binding, histamine release; renal, pancreatic and hepatic disease.[4-15]
- Elevated progesterone and androgens result in: polyphagia, heat intolerance, acne, obesity, and hirsutism.[16-19]
- Severely depleted cortisol causes: anorexia, vomiting, diarrhea, weakness, organ failure and death.[10]

CONCLUSION

These dogs demonstrated concurrent levels of elevated estrogen and low cortisol — a pattern of steroidgenesis described as adrenal exhaustion. Clinical signs were the result of *hyperestrogenism* rather than hypercortisolism. Low-dose glucocorticoid and thyroid hormone replacement had a positive effect on clinical presentation and laboratory findings. Owners should be encouraged to pursue adrenal testing and hormone replacement therapy for signs of elevated adrenal activity.

FUTURE RESEARCH

The author would like to collaborate on SARD cases at the time of onset. Interested parties please contact the author at: caroline@pelicanbooks.com

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