Addison's Disease (Hypoadrenocorticism)

(full seminar notes Sept 2015)

by Jo Tucker

What is Addison's Disease?

Addison's disease occurs when the adrenal glands fail to produce sufficient amounts of mineralocorticoid and/or glucocorticoid hormones. These hormones are essential to life, and failure to diagnose and treat Addison's disease will result in an acute, life threatening situation.

The Purpose of the Adrenal Glands

There are two adrenal glands positioned just before the kidneys - hence the name adrenal (ad-renal), and they are responsible for producing several essential hormones.

The adrenal glands are fully developed by the time the dog is 4 months old and they are an important part of the body's endocrine system which consists of several different hormone producing glands that regulate growth and development, tissue function, reproduction, metabolism, sleep, mood etc…..

Both glands are identical. They are made up of an inner core (the medulla – not significant in Addison's disease) and encased by three outer layers (or zones) called the cortex.

The outer zone of the cortex is the glomerulosa, and this produces aldosterone, the principal and vital, mineralocorticoid hormone. This hormone conserves sodium and chloride, and excretes potassium, maintaining an essential and healthy electrolyte balance that assists kidney regulation. It is also fundamental to the regulation of water balance in the body (hydration), and blood pressure.

The middle zone of the cortex is the fasciculata. This part of the adrenal gland constantly produces sufficient glucocorticoid hormone (cortisol – a stress hormone) to maintain day-to-day wellbeing, blood pressure, appetite, immune system function, intestinal function etc., but it also has the ability to increase or adjust production of this hormone in response to stress (both physical and psychological) for example: physical exercise, injury, pain; and stressful situations such as, thunderstorms, change in circumstances etc. This is often referred to as the 'flight/fight' hormone.

The third zone is the reticularis which produces sex hormones and some cortisol, but it is not clinically significant in adult dogs.

Approximately 90% of dogs with Addison's disease have typical primary Addison's disease, whereby all layers of the adrenal cortex have been destroyed. These dogs are deficient in both mineralocorticoid and glucocorticoid hormones.

The other 10% have atypical Addison's disease, and only the middle zone, the fasciculata, is destroyed. Dogs with atypical Addison's disease are only deficient in glucocorticoid hormone.

Cause

Primary Addison's disease is caused by an autoimmune destruction of the adrenal glands whereby the body's own immune system spontaneously produces antibodies that are specifically targeted to destroy the adrenal glands.

It is an uncommon disease within the dog population but certain breeds such as Standard Poodles and Bearded Collies are known to be genetically predisposed to Addison's disease. (BSAVA Manual of Canine and Feline Endocrinology).

The age range reported for the onset of primary Addison's disease is 2 months to 16 years; although a newly diagnosed `older' dog may have an underlying cause.

Autoimmune Disease in brief

There are many different autoimmune diseases.

The immune system of genetically predisposed dogs are sometimes unable to distinguish between what is 'self' and what is 'foreign' to their body.

These dogs may develop an autoimmune disease if their immune system mistakes a part (or parts) of its own body as a foreign invader – for example, in Addison's disease it no longer recognises its adrenal glands as `self'. The immune system will aggressively respond and produce antibodies that are programmed to destroy part (as in atypical Addison's disease) or all of the adrenal glands resulting in primary typical Addison's disease.

The Disease Process of Addison's disease

The disease process of Addison's may be chronic or acute. This is supported by vague clinical signs that wax and wane of over a period of months prior to a diagnosis; but it can also present in an acute state of collapse and shock (an adrenocortical crisis).

The speed of onset depends on the rate of destruction of the glands. As the disease process advances, the glands become smaller and smaller until the shrivelled glands are rendered useless and adrenal failure becomes inevitable (this may be evident by ultrasound examination). It is at this stage when clinical signs become persistent and more obvious.

Unfortunately, the disease process is un-detectible until clinical signs start to show, by which time 85-90+% of adrenal function is lost. The length of time it takes to reach this stage is thought to be anything from a few weeks to one year, and this may vary from dog to dog and is reflected in retrospective clinical signs. A chronic condition may be exacerbated and change to acute adrenal failure, as a result of the dog experiencing a stressful situation.

In very rare cases, fungus, cancer or haemorrhage within the adrenal glands can cause adrenal gland failure.

Secondary Addison's disease can occur when a dog is being treated for Cushing's syndrome (a disease that causes excessive production of cortisol). This happens if the medication used to control the disease reduces cortisol production too much resulting in a drug induced Addisonian crisis.

Very rarely, Addison's disease can be the result of a diseased pituitary gland which fails to produce the stimulating hormone ACTH.

Diagnosis

To obtain a definitive diagnosis, an ACTH (adrenocorticotropic hormone) stimulation test is necessary. The test involves first, taking blood from the dog and then administering intravenously a synthetic ACTH. A further blood sample is taken one hour after the ACTH administration. Both samples are sent to the laboratory to measure the blood cortisol of both pre and post ACTH stimulation test blood samples. A 'flat-line' response to an ACTH stimulation test is diagnostic of

Addison's disease and most dogs with Addison's will have a zero measurement, or lowered baseline measurement, of cortisol both before and after the ACTH test.

It sounds simple – *so why is Addison's disease so difficult to diagnose?*

The diagnosis of Addison's disease is not complicated but some vets can be reluctant to even consider it in their differential diagnoses. Addison's disease is known as `*The Great Pretender'*. The reason for this notorious label is because Addisonian dogs generally present with a wide variety of vague, clinical signs and this, unsurprisingly, can lead to a misdiagnosis of: renal failure, heart failure, gastrointestinal disease, acute pancreatitis, liver disease and even autoimmune haemolytic anaemia (AIHA).

Many vets say "It won't be Addison's as we never see it". Unless your vet is looking for Addison's disease, it will not be diagnosed. Most dogs are presented to their vet at least three times in the 6+ months prior to diagnosis, and many are in an Addisonian crisis before it is identified. This is a true emergency and immediate, supportive care is essential if the dog is to survive.

The biggest hurdle for the owner can be to convince the vet to at least consider the possibility that their dog may have Addison's disease, and not to dismiss the notion without proving it and carrying out a thorough investigation and possibly an ACTH test.

Common Clinical Signs:

Adrenal hormone deficiency will have a significant effect on many body systems, causing a variety of vague clinical signs, such as:

Lethargy, depression, nervousness, weight loss, anorexia (no appetite), vomiting, limping, profound weakness (particularly of the back legs), inability or reluctance to walk even a short distance, exhaustion, shaking, twitching or muscle tremors, collapse (particularly whilst out walking or if stressed), diarrhoea (with or without traces of blood), abdominal pain, dehydration (watch out for tacky gums), excessive thirst and urination, with the inability to concentrate urine; weak pulse, slow heart rate and abnormal heart rhythm, anaemia (pale gums), increased pigmentation of the skin, megaoesophagus (enlargement of the oesophagus resulting in difficulty with swallowing) and rarely, seizures (cause by low blood sugar).

The chances of a dog showing all these clinical signs are unlikely, but just a few signs might be enough to raise your suspicions. Common waxing and waning clinical signs can be: recurring stomach upset and/or diarrhoea, with or without blood. Loss of appetite is another common sign; and increased drinking and urinating/incontinence; nervousness/overly sensitive to noise, and lethargy are also typical. Do not expect a rise in body temperature; a normal or low-normal body temperature is most common. Some weight loss and signs of dehydration may be evident, but until this stage physical examination is likely to reveal very little, although there may be some tenderness around the kidneys. The closer adrenal destruction gets to 100% the greater clinical signs increase, and an adrenal crisis becomes almost inevitable.

There can be a temptation for some vets to suggest surgical exploration, or an endoscopy, as being the best course of action if they are unable to achieve a diagnosis, especially when a dog is presenting with varied, intermittent symptoms. If the dog is ultimately diagnosed Addisonian, an exploratory procedure, prior to treatment, could prove to be fatal. Addison's disease should always be ruled out prior to any potentially stressful or invasive technique being performed on a dog with vague, waxing and waning clinical signs, especially if they are of a breed known to have a genetic predisposition to Addison's disease.

Further Considerations:

Is your dog young / middle aged?

Over a period of time, has your dog experienced several of the typical symptoms? Has he/she been dehydrated and responded well to *fluid therapy* - but the improvement didn't last?

Has your young to middle aged dog been diagnosed with kidney disease? Is he/she improving on the special treatment/diet provided by your vet? If the answer is no, then consider Addison's disease. Kidney disease is the most frequent misdiagnosis of Addison's disease. True, primary kidney failure occurs more often in older dogs and not usually young, previously healthy ones. Are the blood results typical for kidney failure? Check the sodium and potassium levels and ratio, and the leucocyte count. Are these consistent with kidney failure?

If your dog has persistent or intermittent gastrointestinal problems then consider other subtle, clinical signs and look for possible clues in the blood results.

Do you have a dog whose breed is known to be genetically predisposed to Addison's disease?

Does your dog already have an autoimmune endocrine disease, e.g. Hypothyroidism (an underactive thyroid)?

Do you know of any relatives of your dog who may have been diagnosed with Addison's disease or another autoimmune disease, or may have died unexpectedly of suspected kidney failure at a young age? Don't be afraid to speak to your dog's breeder; they may have valuable data about your dog's family history.

If You Suspect Typical Primary Addison's Disease

Inform your vet of the diseases your dog's breed is predisposed to. Have a *Full Serum Biochemistry panel* and a *Complete Blood Count* test done and ask your vet for a copy of the results to keep for your own record. Study the results yourself and note any abnormalities, especially the sodium and potassium values/ratio. If symptoms persist, have a further blood test done to see if there are any changes, but don't leave it too long in between (a week or less) as deterioration seems to quicken in the final stages of the disease process. Keep all laboratory reports for future reference and comparison. Blood testing is never a waste of money (in the long term it can often save you money), and will provide a 'bench mark' on which to compare subsequent tests. Addison's disease is progressive, so a blood test is only valid at the time it was taken. As the disease process advances, the values will change.

The Tell-tale Signs When Looking at Blood Test Results

Routine Laboratory Abnormalities - Haematology & Biochemistry

Sodium/Potassium Ratio

Greater than 90% of Primary Addisonian cases will have **low sodium (Na)** and **high potassium (K)** values, with a **ratio of less than 27:1 (Na:K)**

Prior to diagnosis, `typical' Addisonian dogs often show a ratio of less than 23:1. A low ratio is **very** suggestive, but not diagnostic, of Addison's disease. Individual electrolyte concentrations can be a more reliable indicator.

A diagnosis of Addison's disease must not be presumed just because a dog has a low sodium/potassium ratio. An ACTH test must be performed to confirm Addison's disease.

As the disease progresses, the sodium and potassium ratio will drop even further and the dog may collapse and become critically ill, especially if the dog becomes stressed or excited. Anxiety or excitement, in a dog with reduce adrenal function (even in a dog who has not exhibited typical, clinical signs of Addison's disease) can cause the dog to collapse or even die suddenly.

The mild, clinical signs of *`chronic'* adrenal disease do not differ greatly to the grave clinical signs of *`acute'* adrenal disease. The existing, or previous, clinical signs appear increasingly more severe and urgent when an adrenal crisis is imminent.

Prompt diagnosis and treatment, or supportive treatment, is crucial for a favourable outcome. An Addisonian crisis and must be treated as a true emergency if the dog is to survive.

When comparing laboratory results look for the following as these will give a strong indication of Addison's disease:

INCREASED:

High Potassium (K)
High Creatinine,
High Urea, (BUN – blood urea nitrogen; or SUN - serum urea nitrogen)
High Urea/creatinine ratio (Azotaemia)
Increased Eosinophils
Increased Lymphocytes
High Bilirubin - in some cases
High Calcium (mild to moderate) – in some cases
ALT- ALP - AST (Mild to moderate increase of liver enzymes) – in some cases

DECREASED:
Low Sodium (Na)
Low Sodium/potassium ratio (Na:K) ratio - less than 27:1)
Note: Addisonian dogs often have a ratio of <23:1
Low Chloride (80% of Addisonian dogs will have low chloride values)
Low Glucose – in some patients
Low Albumin (moderate to severe) – in some cases
Total white blood cell count (WBC) – in some cases
Red blood cell count (RBC or HCT)

Note: Whipworms also cause low sodium and high potassium concentrations.

Another possible difference between kidney disease and Addison's may be seen in the white blood cells (eg., neutrophils, eosinophils, lymphocytes).

When a dog is very ill (*but does **not** have Addison's disease*) stress levels rise and this is often reflected in the white cells. The neutrophil numbers in a very poorly dog are expected to be high-normal to increased, and the eosinophils and lymphocytes numbers are low-normal to decreased. This is called a 'stress leucogram' and is seen in both chronic and acute renal failure, **but not in Addison's disease.**

A dog with Addison's disease will show a different white cell reading than would be expected in such an ill dog. In fact there may even be reverse of what would normally be expected, known as 'reverse stress leucogram', caused by the lack of glucocorticoid hormone.

Reverse Stress Leucogram – An important indicator, and is seen in Addisonian dogs

Low-normal numbers of neutrophils

Increased numbers lymphocytes and eosinophils

Just remember, a very poorly dog showing a normal or elevated lymphocyte count is suggestive of Addison's disease.

Electrocardiogram

- Electrocardiogram (ECG) is a very useful tool to detect various abnormalities of the heart resulting from high potassium levels in the blood.

Acutely affected dogs may be in hypovolaemic (reduced blood volume) shock, with severe dehydration and bradycardia (slow heart beat). A collapsed dog with a slow heart beat is unlikely to survive for long, so supportive treatment is both essential and urgent.

Note: Usually, a dog with hypovolaemia will have a fast heart beat (tachycardia). In a hypovolaemic dog with Addison's disease the heart beat will be slow (bradycardia). This anomaly is very suggestive of Addison's disease.

Another misleading differential is non-regenerative autoimmune haemolytic anaemia (AIHA). Decreased glucocorticoid hormone (as a result of adrenal insufficiency) suppresses production of red blood cells in the bone marrow resulting in the dog becoming anaemic. Regenerative anaemia (anaemia within the circulation of the blood) may be present due to gastrointestinal bleeding.

Note: The severity of anaemia can be masked if the dog is dehydrated.

I have known a number of dogs to be diagnosed with AIHA prior to being diagnosed with Addison's disease. The usual treatment for AIHA had been implemented but the clinical signs did not improve as expected, and vague clinical signs of Addison's disease, remained.

If this happens you should check for signs of Addison's on previous blood test results to see if there were any undetected tell-tale results such as high potassium, low sodium and a reverse stress leucogram. These cases are further complicated because the dog will be receiving steroid treatment (prednisolone) for AIHA. A diagnostic ACTH test for Addison's disease cannot be performed whilst a dog is on prednisolone, as it interferes with the test and results, so the dog's steroid medication must be changed to a different steroid called dexamethasone for at least 24 hours before an ACTH test is performed.

Atypical Addison's Disease

Atypical Addison's disease occurs when the part of the adrenal gland that produces the glucocorticoid hormone only is affected.

Dogs with atypical Addison's disease will have normal renal (kidney) blood values and, unlike dogs with primary typical Addison's disease, they will not usually show classic changes in their sodium and potassium levels.

Note: *Some atypical Addisonian dogs may still be deficient in mineralocorticoid hormones without it showing on the blood results. (Catharine Scott-Moncrieff)*

Clinical signs may include: anorexia, vomiting, diarrhoea, weight loss, non-regenerative anaemia, exercise intolerance.

Atypical Addisonian dogs may have been misdiagnosed with inflammatory bowel disease (IBD), non-specific gastrointestinal disease (e.g. colitis), liver disease or non-regenerative haemolytic anaemia. These cases can be challenging to the vet because dogs will often respond more favourably to fluid therapy. The disease process of Atypical Addison's disease may appear slower leading up to a diagnosis, and it may develop at a later age than typical, primary Addison's disease; the mean age being 7 years.

Blood results should show a reverse stress leucogram.

Life Saving Support

An acutely ill Addisonian dog may be collapsed and in a state of shock, with severe dehydration, a slow heart beat and life threatening electrolyte imbalance. Life-saving support treatment must be given immediately.

From BSAVA Manual of Small Animal Endocrinology, Chapter 19 – Endocrine Emergencies

"Whenever a diagnosis of Addisonian crisis is likely, treatment should be initiated without delay."

"A tentative diagnosis of acute adrenocortical insufficiency can be made on the basis of the history and results of physical examination."

"Since death from acute adrenocortical insufficiency is usually attributed to vascular collapse and shock, rapid correction of hypovolaemia is the first priority in treating this condition."

1. ***Restoring blood volume and***
2. ***Correcting imbalance of sodium and potassium levels and***
3. ***Treatment of life threatening cardiac arrhythmias***

Within 1-2 hours, a saline only intravenous drip can restore correct hydration status, increase sodium levels and lower potassium levels which may be causing hyperkalaemic myocardial toxicity. In addition, other protocols may be used if myocardial toxicity is life threatening.

4. ***Correct *glucocorticoid deficiency***

*Dexamethasone is usually given as this has little or no effect on the measurement of endogenous cortisol concentrations and therefore does not interfere with the ACTH test.

Personal note: *If clinical signs and blood results are highly suggestive of Addison's disease and/or the dog is in a suspected adrenal crisis, as long as the ACTH test has been performed, it may be prudent to speculatively treat for Addison's disease. Sadly, I have known several untreated dogs to die whilst waiting for the ACTH test results. Better to be safe than sorry!*

Zycortal Treatment

Once diagnosis is achieved, the prognosis for an Addisonian dog is excellent. The dog should feel much better in a few days but it may take up to 6-8 weeks after the start of treatment for the clinical signs and blood results to return to normal.

Since April 2016, the treatment for Addison's disease in the UK has changed from a daily mineralocorticoid tablet (with some glucocorticoid hormone included), to an injectable mineralocorticoid called Zycortal, ideally given approximately once every month, depending on clinical signs and monitoring of sodium and potassium values.

However, during the first year following the introduction of Zycortal, it is clear from individual case reports that the duration of time between Zycortal injections varies from one individual to another. There are many anecdotal reports suggesting that the manufacturer's recommended starting dose of 2.2mg/kg is much higher than needed, and as a result many dogs have taken a long time to stabilise. These dogs require frequent, sometimes weekly blood tests, to ensure that the sodium and potassium levels are within the optimum range to indicate when the next dose of Zycortal is needed. This is usually followed by a reduction in Zycortal dose, and the same method is repeated at the end of another 28 days and continues to be repeated until the optimum dose for the optimum duration of time is achieved. The duration between injections of Zycortal, in some dogs, has been as long as 80+ days.

Care must be taken not to overdose.

Some dogs have not been successfully stabilised on Zycortal and have reverted back to Florinef to control their Addison's disease.

Vets and owners may like to listen to this very informative podcast by the RVC (Royal Veterinary College). Prof David Church, endocrinologist at the RVC, where he discusses the high starting dose of Zycortal. See link below:

60 Hypoadrenocorticism treatment[https://www.rvc.ac.uk/small-animal-referrals/news-events/clinical-podcasts/60-hypoadrenocorticism-treatment]

Fri, 10 Nov 2017
Author: Dominic Barfield

Duration: 00:40:50

Download 74.8Mb[https://www.podtrac.com/pts/redirect.mp3/www.rvc.ac.uk/small-animal-referrals/Media/SmallAnimalReferrals/podcasts/clinical/60%20HypoA.mp3]

For the 60 th Pod we have a treat, not one but two Professors join Brian and myself in the studio. This week we talk to Hattie Syme, Professor of internal medicine and David Church, Deputy Principal here

at the RVC about treating patients with hypoadrenocorticism. The initial therapy and the transitional therapy. It is all about supplementing with mineralocorticoids and glucocorticoids. Now there might be differing views on which medications to use. This all came about from a conversation with them both on the clinic floor and thought that a slight contrast in views would be good for you to hear.
To screen for HypoA
https://www.ncbi.nlm.nih.gov/pubmed/22092627[https://www.ncbi.nlm.nih.gov/pubmed/22092627]

Please see these links also:

https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=2c9b766a-c36b-44aa-bbd8-24fab24ca97c

http://www.endocrinevet.info/2011/11/q-prolonged-action-of-percorten-in-dog.html

www.addisondogs.com/addisons/articles/julia_bates_interview_2012.pdf

http://www.ncbi.nlm.nih.gov/pubmed/23438457

A daily dose of prednisolone, to replace glucocorticoid deficiency, will also be necessary when a dog is being treated with Zycortal.

'Oral replacement dose of glucocorticoid in an Addisonian dog is prednisolone 0.1– 0.22mg/kg once a day, then taper to the lowest dose to control clinical signs'. (Scott Moncrieff, 2010, Ref: Plumb's Veterinary Drug Handbook, Eighth Edition).

This dose may need to be increased at times of stress because an Addisonian dog no longer has the 'supply and demand' facility for the 'fight/flight' response at times of stress. It might be prudent for the owner to anticipate added stress such as a brewing thunderstorm or kennelling etc.

Life-long, daily glucocorticoid hormone replacement therapy is essential alongside regular subcutaneous injections of Zycortal.

Zycortal is expected to suit approximately 80% of Addisonian dogs, but those who fail to stabilise on Zycortal will have to revert to treatment with Florinef. See information below.

Florinef:

Florinef is a daily replacement mineralocorticoid hormone tablet (with some glucocorticoid activity) called fludrocortisone (Florinef). Tablets should be given regularly, around the same time/times every day. Initially, Florinef will be given with the addition of another hormone tablet called prednisolone. Prednisolone is a glucocorticoid hormone.

Note: Although Addison's disease is easy to treat with Florinef, daily medication is essential. Missing one or two doses of Florinef is not an option and it could prove to be fatal.

Florinef Treatment - Drugs & Dosage:

Extract from BSAVA Small Animal Formulary, 6th Edition

For mineralocorticoid supplementation in chronic or subacute adrenal insufficiency:

Fludrocortisone (Florinef) Oral: 0.1mg tablet

Initial dose:

Start at 0.01mg/kg 24 hrs depending on size of animal. Monitor sodium and potassium levels every 1-2 weeks and adjust dose by 0.05 – 0.1mg accordingly. Most patients once stabilised will require approx. 0.1mg/5kg 24hrs.

For glucocorticoid supplementation in chronic or subacute adrenal insufficiency:

Prednisolone 0.2-0.3mg/kg given once daily with fludrocortisone. The use of prednisolone may be discontinued in most cases once the animal is stable.

Useful References:

"When the disease is first diagnosed, a higher dose of prednisolone is often necessary (up to 0.5mg/kg every 12 hours) but should be tapered rapidly to the lowest amount". Ref: Unmasking the Great Pretender': how to recognise and manage canine Addison's disease by Audrey Cook BVM&S MRCVS Dip ACVIM-SAIM Dip ECVIM-CA'

'Most dogs can be successfully treated on once daily therapy but some will require twice daily treatment to maintain electrolyte concentrations within reference ranges. Fludrocortisone possesses a small degree of glucocorticoid activity and therefore also assists in weaning affected dogs off prednisolone therapy. The dosage of Fludrocortisone increases with time for reasons that are yet unclear and many dogs subsequently require doses as high as 0.03 mg/kg/day'. Ref: FECAVA Lecture Addison's Disease (Hypoadrenocorticism) in Dogs – Carmel T Mooney

Note: Florinef should be kept cool and stored in a fridge.

A gastroprotectant may be given if gastrointestinal bleeding is present.

Following replacement hormone treatment with Florinef and prednisolone, the dog should feel much better in a few days, but it may take up to 6-8 weeks for the clinical signs and blood results to return to normal. The optimum dose of Florinef is established during this time and several blood tests, to check that the sodium and potassium ratio is back within normal range, are necessary and should be performed weekly from the start of treatment.

The oldest dog with Addison's that I have known was over 16 years old when she died, so getting that diagnosis is worth fighting for.

For more information about Addison's Disease and treatment, please see the following link:

http://www.addisondogs.com/support/

Just because the sodium and potassium ratio has increased to 27:1 doesn't mean the optimum dose of Florinef has been reached. The ratio should be greater than 27:1. If clinical signs are still obvious then an increase in Florinef dosage is probably needed. Subsequent, small increases are expected until the dog appears `normal' and the improvement in clinical signs is reflected in normal blood results.

If a poor appetite persists, an increase in the dose of prednisolone may be required. If the dog has an excessive thirst, the prednisolone may need to be reduced. The aim is to reach the `*perfect*' hormone replacement dose for the individual, and this will vary from one dog to another. Once

stabilised, it is hoped that the dog can be maintained on Florinef only, needing an occasional low, replacement dose of prednisolone at times of stress (the automatic `supply and demand' facility for the f*ight/flight' response*, naturally provided by the glucocorticoid hormone, cortisol, is no longer available to Addisonian dogs). It might be prudent for the owner to anticipate potential stress such as an impending thunderstorm or noise from fireworks, building works etc., and give a low, one-off dose of prednisolone for that day.

Once the optimum dose of medication has been achieved, it is anticipated that the dog will live to a normal life expectancy however, there is a chance that an Addisonian dog may be predisposed to other endocrine diseases.

Healthy dogs become unwell from time to time and Addisonian dogs are no different so don't assume that every future health blip is because their Addison's is not being controlled. The problem could be totally unrelated.

If your Addisonian dog has to have a general anaesthetic then depending on how serious the predicted stress will be, the vet should increase prednisolone to above the normal dose just before, during and after the procedure. It is also important to maintain fluid support whilst an Addisonian dog is under an anaesthetic.

Percorten-V (Desoxycorticosterone pivalate – DOCP): Dogs with primary typical Addison's disease living in the USA are generally treated with an injectable mineralocorticoid replacement called Percorten-V. This injectable mineralocorticoid lasts for approximately 25-30 days and is given with a low, daily dose of prednisolone. Percorten-V is not licensed in the UK.

Treatment of Atypical Addison's Disease:

Extract from BSAVA Small Animal Formulary, 6th Edition

<u>Prednisolone:</u> 0.2-0.3mg/kg given once daily.

"When the disease is first diagnosed, a higher dose of prednisolone is often necessary (up to 0.5mg/kg every 12 hours) but should be tapered rapidly to the lowest amount". **Ref: *Unmasking the Great Pretender': how to recognise and manage canine Addison's disease by Audrey Cook BVM&S MRCVS Dip ACVIM-SAIM Dip ECVIM-CA.***

Atypical Addisonian dogs may ultimately become 'typical` and require replacement mineralocorticoid hormone. Sodium, and potassium levels should be monitored every few months for the first year following diagnosis and the owner should look out for any clinical signs of primary typical Addison's disease. Checking aldosterone levels might be prudent as atypical Addisonian dogs are less likely to develop typical Addison's disease if they have normal aldosterone levels.

After All This, Is It Worth It?

YES, absolutely worth it! Just ask all the people who have shared their life with an Addisonian dog and still have the pleasure of seeing them enjoying a normal life, running and playing just like all the other dogs. These dogs are often young and very healthy and once replacement therapy is underway and the dog is stable they are, to all intents and purposes, normal dogs again. They may have a few ups and downs along the way, but on the whole they will live life to the full.

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All information is provided for assistance and reference purposes only and is not meant in any way to substitute advice or treatment from your veterinary surgeon.

References:

BSAVA Manual of Canine and Feline Clinical Pathology
BSAVA Manual of Small Animal Endocrinology
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`Unmasking the Great Pretender': How to recognise and manage canine Addison's disease by Audrey Cook BVM&S MRCVS Dip ACVIM-SAIM Dip ECVIM-CA.