DIC in a Cockatiel

Deborah Monks
Brighton Veterinary Surgery
Brisbane, Queensland, Australia.

Abstract

This report discusses a case of ascaridiasis and air sacculitis in a young cockatiel, which ultimately led to disseminated intravascular coagulation (DIC) and death. It highlights some deficiencies in diagnosing ascarid infections and air sacculitis, and discusses DIC as an under-reported condition in birds.

Case Study

On 18/5/99, a 10 week old cockatiel was presented with a complaint of discoloration of the left foot. It had been purchased 3 weeks previously from a market, and weighed 88g. Physical examination showed a bluish discoloration of the left foot, with decreased circulation (evidenced by decreased heat production compared with the contralateral foot) and minor lameness. There were multiple discolored areas from hip to tarsus, resembling bruises. Microscopic examination of both faeces and a crop wash by wet smear were negative for pathogens. The presumptive diagnosis at this stage was trauma. The bird was started on a treatment regime of twice daily topical DMSO/Tribactril mixture .[1]

Three days later, the discoloration of the left leg had worsened and now resembled cyanosis. The right foot was also discoloured, and the leg had areas of bluish discoloration. The peri-lumbar dermis (near powder down) was erythematous and felt hotter than the surrounding skin. The beak was pale, and both wing tips were oedematous. Repeat examinations of wet faecal smears were negative for pathogens.

The problem list at this point included anaemia and oedema. The differential list included protein-losing nephropathy, protein losing enteropathy, vasculitis, toxin exposure, bacterial nephritis and renal failure. The owner had significant financial constraints, which limited further diagnostic testing.

Blood was collected from an unrelated cockatiel in a lithium heparin syringe for an homologous blood transfusion, while preparations were made for anaesthesia to perform endoscopy. A blood sample was taken from the patient before transfusion. Analysis after the anaesthetic revealed a PCV of 35, with a total protein 32g/L. The endoscopic exam, performed through a left flank incision showed smooth, marginally swollen kidneys. All other organs were grossly normal. One ml of whole blood was transfused slowly into the left jugular vein, and the bird recovered uneventfully

[1] Dimethyl Sulphoxide (Pharmachem) / Tribactril 80mg Trimethoprim: 400mg Sulfadiazine per ml, Jurox.

The bird was maintained in hospital on twice daily lincospectin[2] intra-muscular injections (0.04ml), continued twice daily topical DMSO/Tribactril (12ml:3ml)[1], and twice daily crop feeding with Roudybush[3] handrearing mixture. Over the next 12 hours, the distal wing oedema resolved.

Over the next two days, the cyanosis spread to the tarsometatarsus and all toes became necrotic. The bird lost weight (down to 82g), despite the crop feeding. Faecal examinations continued to be negative. On 23/5/99, the bird began passing frank blood in the stools. No cause was found and the owner elected euthanasia due to poor response to treatment.

Post-mortem showed a severe ascarid burden, causing gross intestinal dilatation. Immature worms were occasionally present in the peritoneal cavity. Both atria were enlarged, and a large clot was within one atrium. The liver was mottled. The air sacs were cloudy. Samples of heart, liver, kidney, airsac, spleen, proventriculus were submitted, along with an entire foot (encompassing both necrotic and viable tissue).

Pathology results showed the right side of the heart entirely occupied by a blood clot (probably an organising thrombus), with mild to moderate multifocal myofibre degeneration accompanied by some leucocytic inflammation. The liver had multiple areas of coagulative necrosis, associated with venous and sinusoidal thromboses. The kidney had early thrombotic occlusion of hilar vessels, associated with widespread multifocal acute tubular epithelium degeneration. The airsacs had severe inflammation, featuring congestion, organising thrombosis, haemorrhage, oedema, fibrinous exudation and diffuse cellular infiltration. There were numerous intracellular gram-positive rods. Extensive epithelial ulceration was present, with hyperplasia of the residual epithelium. The spleen had congestion, depletion of lymphoid tissue and reticuloendothelial transformation of periarteriolar lymphoreticular sheaths. There was subcapsular inflammation with an organising fibrinosuppurative and haemorrhagic process, and gram positive bacteria were detected intracellularly within macrophage cytoplasm. There was coagulative necrosis associated with focally organising thrombi in extremely congested dermal vessels within the foot.

The histological diagnosis was disseminated intravascular coagulation, and gram positive bacterial air sacculitis with serositis.

Discussion

Ascarids are common endoparasites in birds; particularly those kept in dirt-floored aviaries. Predisposing conditions include inadequate hygiene, and ineffective anthelmintic regimes. The life cycle is direct, and, once passed, eggs require 2-3 weeks for embryonation before becoming infective (Greiner & Ritchie, 1994). Clinical signs of infection range from subclinical to weight loss, inappetance, ill thrift and diarrhoea. Severe infections can cause malabsorption, hypoproteinaemia (due to a protein losing enteropathy), intussusception, bowel occlusion and death. Diagnosis is usually via microscopic examination of faeces, by either wet mount, or faecal floatation.

The massive ascarid infection is the most likely cause of this bird's hypoproteinaemia, which in turn led to wing tip oedema. This resolved rapidly after the blood transfusion, which presumably elevated plasma protein. Frank haemorrhage into the gut was presumed to be a combination of mechanical

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[2]2 Lincospectin Injectable Solution, 50mg Lincomycin:100mg Spectinomycin per ml, UpJohn
[1 3]3Roudybush Handrearing Formula 3, Roudybush Australia.

damage by the parasites, and lack of clotting ability due to disseminated intravascular coagulation (DIC).

It was confounding that repeated faecal examinations were negative. Performing faecal flattens could increase the sensitivity of endoparasite diagnosis.

This bird was clinically immunosuppressed with advanced air sacculitis, septicaemia and ascaridiasis. With the limited diagnostic testing permitted, it was impossible to determine if these were the initial causes of disease, or consequences of immunocompromise. Further testing might have included full blood counts, biochemistries and faecal and air sac culture and sensitivity.

Air sac infections may be inapparent on physical examination, and may require endoscopy or radiography for diagnosis. In this case, the histologic severity of the infection was belied by the gross appearance, both on endoscopy and on post-mortem. The peritoneal ascarids probably contributed to air sac pathology. The presence of extra-gastrointestinal ascarids can be due to aberrant migration or intestinal perforation.

Although this bird had a pale beak, which were initially assumed to be due to anaemia, the PCV was within normal limits. The pallor was possibly caused by ischaemia, secondary to capillary thrombosis.

The atrial clot may have been caused by migrating ascarid larva, circulatory changes due to septicaemia, cardiac dysfunction (evidenced by myofibre degeneration), or secondary to disseminated intravascular coagulation (DIC).

DIC is the end result of many disease processes, and involves the concurrent activation of both coagulation and fibrinolytic pathways. The effects can vary, and may depend upon the underlying disease rate and severity. DIC results from blood cell, blood vessel or tissue injury, which release factors that initiate the coagulation cascade, causing an hypercoagulable state. Once initiated, this results in circulating emboli, which may cause vascular occlusion in many organs and consume platelets and coagulation factors (Feldman, 1981). These effects may manifest preferentially in the extremities (N. Sullivan, pers. com). Simultaneously, the fibrinolytic pathway attempts to dissolve the thrombi, which results in circulating fibrin degradation products (FDPs), which have an anticoagulant activity. The combination of platelet depletion and increased FDPs causes an hypocoagulable state, signs of which include petechiae, gastrointestinal bleeding, epistaxis etc (Feldman, 1981; Drazner, 1982).

Diagnosis of DIC in mammals requires evidence of prolongation of partial thromboplastin time (extrinsic pathway) and prothrombin time (intrinsic pathway) and decrease in platelet count below a certain level (Firth, 1995). Activated clotting tubes (ACT) may provide a more immediate, in-house alternative to the PTT and PT (Feldman, 1981).

Shibatani *et al* (1997) demonstrated increased prothrombin and activated partial thromboplastin times in their experimental model of avian DIC induced by *Erysipelothrix* infection. Avian thrombocytopaenia can be demonstrated on a blood film, but PT and PTT require laboratories with avian reference values, or the submission of a normal control. ACT may allow faster in house comparison.

Diagnosis and monitoring of DIC requires multiple assessments of haematology and clinical signs. DIC is a complication of serious systemic disease and treatment is unlikely to be successful unless this can be addressed. Treatment of DIC is aimed at immediately treating the primary aetiology, and reversing the secondary circulatory changes caused by DIC. The latter is most often attempted with transfusions

and fluid therapy attempt. These reduce circulatory stasis, improve hypovolaemia, replace depleted coagulation factors and correct acid-base and electrolyte imbalances (Drazner, 1982).

Conclusions

1. A negative faecal examination does not always indicate a negative parasite burden. Clinicians must be suspicious of occult ascaridiasis in hypoproteinaemic patients.
2. Gross appearance of organs and air sacs at endoscopy may be misleading. Biopsy and cytology should be used in conjunction with gross assessment.
3. DIC is a serious and under-reported complication of systemic disease in avian patients. Diagnosis and monitoring of DIC requires multiple assessments of haematology and clinical signs. Treatment of DIC is aimed at reversing the circulatory changes, coagulation deficits and removing the primary cause.

Acknowledgements

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