Common Reproductive Disorders in Birds & Exotic Pets

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Keywords: Reproductive, Birds, Exotic Pets

Introduction:

Reproductive disorders are common in captive birds and exotic species including reptiles and mammals. The myriad of diseases of this body system encountered in these species frequently reflect suboptimal husbandry. In many instances the prevalence of such conditions can be reduced with increased client education and awareness, combined with appropriate preventative health measures. Nonetheless, reproductive disorders in birds and exotic pets frequently present in veterinary practice and clinicians should be familiar with their presentation, diagnosis and management.

1. Birds

1.1 Dystocia

Dystocia, commonly referred to in birds as "egg binding," refers to the delayed or failed passage of an egg through the oviduct causing a mechanical obstruction. The average normal rate of egg passage through the oviduct is 24-48 hours[1,2]. Dystocia is frequently seen in domestic birds especially small psittacines such as budgerigars, cockatiels, and lovebirds. It is occasionally seen in backyard poultry, such as bantam chickens.

The reproductive tract of most hens includes a left ovary and left oviduct (salpinx). The oviduct is divided into five functional sections: the infundibulum, magnum, isthmus, uterus or shell gland where calcium is deposited to form the hard shell and the vagina. Each component plays an important role in developing the egg and in oviposition.

Egg laying is driven by both endogenous and exogenous factors[3]. Endogenous factors are less well understood but include gonadotropic hormones follicle stimulating hormone (FSH) and luteinising hormone (LH), other sex hormones as well as age and sexual maturity[3]. Exogenous factors refer to environmental cues for reproduction such as photoperiod, humidity, rainfall and abundance of food[3]. It's important for veterinarians to be aware of the environmental influences on avian reproductive behaviour both as it pertains to client education, husbandry practices and manipulation of reproductive activity to manage reproductive disorders.

Major predisposing factors include hypocalcaemia, obesity, oversized or misshapen eggs, salpingitis/metritis, and systemic illness and weakness. Hypocalcaemia is one of the leading causes of dystocia in birds and in early stages may present as soft-shelled eggs ('slugs' or 'lash eggs') being laid. Some parrot species such as cockatiels and budgerigars are particularly predisposed to dystocia as they are indeterminate layers. This means that rather than laying one to two clutches seasonally, they will continue to lay eggs with no end point, particularly if eggs are removed[3].

Clinical signs of dystocia include dyspnoea, tenesmus, coelomic distension, wide-based stance, lethargy, weakness, inappetence and there may be pelvic limb paresis or paralysis due

to obturator nerve compression and paralysis. In severe cases dystocia can compress and compromise vital organs causing visceral necrosis and post renal disease.

Diagnosis is based on history, signalment and clinical signs, physical exam - which will usually reveal a firm mass on coelomic palpation - and confirmation via radiography and/or ultrasound, which demonstrates the presence and location of the egg as well as other important features such as egg size and bone density. Ultrasound is necessary to identify unshelled or soft-shelled eggs (as a result of hypocalcaemia). On initial presentation many birds are too compromised to perform radiographs safely under general anaesthesia. In these instances a 'bird in a box' radiograph can be performed conscious, with oxygen supplementation if necessary, purely for the purposes of identifying and confirming the presence of an egg.

Management depends heavily on the timeline, severity of dystocia and the physical state of the patient[1]. Medical stabilisation should be the priority but for advanced cases further interventions like ovocentesis and even salpingotomy surgery may be required, once the patient is stabilised. Medical stabilisation involves heat support, vent lubrication, fluid therapy (SC, IV, IO), parenteral calcium supplementation, analgesia, and oxygen therapy.

Inducing oviposition in egg-bound hens is a point of contention and always carries a reasonable risk of uterine rupture and prolapse. Prostaglandins such as Prostaglandin E2 gel can be applied to the cloaca to relax the uterovaginal sphincter and encourage egg expulsion if the egg is in the shell gland or vagina[2,4] but this drug carries a risk of uterine rupture, particularly with large eggs and further it carries occupational health and safety risks for pregnant personnel. Oxytocin is less reliable in birds and carries an even higher risk of uterine rupture and has no effect on relaxing the uterovaginal sphincter in birds. Arginine vasotocin is more biologically appropriate and a more potent hormone for stimulating uterine contractions in birds but is not readily available.

If the egg does not pass in 24-48 hours, the bird is excessively straining, distressed or dyspnoeic then emergency ovocentesis may be required. The bird should be anaesthetised and ideally intubated for this procedure. A 16-18 gauge needle is introduced into the cloaca to pierce the egg. Ideally the egg is collapsed and drained then removed in one piece. However this is not always possible. In these cases the egg shell pieces will usually be expelled within the next few days provided there are no adhesions. If there are adhesions or damage to the oviduct has occurred then salpingohysterectomy is usually required.

Preventing dystocia involves addressing nutrition, ensuring the bird is on a balanced diet, appropriate for the species with appropriate calcium and vitamin D3. Supplementation is often required until diet conversion is achieved. Seed diets contain about 20% of the calcium requirement for non-laying birds and as little as 5% for laying birds. Birds should be fed a formulated diet appropriate for the species (pellets or crumble) supplemented with fresh plant foods.

In addition to nutritional management ongoing prevention involves avoiding chronic reproductive stimulation through provision of an appropriate 10-12 hour photoperiod and environmental management (e.g. removing nest boxes, no petting below the neck). GnRH agonists such as deslorelin (Suprelorin®) or leuprolide acetate can also be helpful adjunctive measures.

1.2 Egg Yolk Peritonitis (Egg Yolk Coelomitis)

Egg yolk peritonitis is a life-threatening condition seen often in high-producing birds such as cockatiels, budgerigars, and backyard chickens. It occurs when yolk material leaks into the coelomic cavity, typically secondary to retrograde ovulation or oviductal rupture. Not uncommonly, there is concurrent reproductive tract infection[3].

Affected birds present with dyspnoea (due to coelomic compression of the air sacs), a wide based stance, coelomic distension, anorexia, lethargy, and usually a history of previous or recent egg laying.

Diagnosis is based on physical examination, imaging and analysis of coelomic fluid. Ultrasound is the imaging modality of choice and facilitates ultrasound-guided coelocentesis. Coelocentesis is both a therapeutic and diagnostic procedure and often large volumes of fluid are drained, particularly from poultry. Anaesthesia or other methods of chemical restraint are often required, especially for parrots. However it may be able to be performed conscious in chickens. Typically, once coelocentesis is performed a more thorough coelomic ultrasound can be performed provided the bird is stable.

Analysis and cytology of the coelomic fluid and ultrasound findings such as metritis or retained eggs confirm the diagnosis. The coelomic fluid appears yellow to brown and is proteinaceous[5]. Usually it is a sterile peritonitis. Septic peritonitis is rare[3]. However there is often concurrent metritis or salpingitis warranting antibiotic therapy.

Initial management should prioritise stabilising the patient, which includes oxygen therapy and coelocentesis. Following this, supportive measures such as crop feeding and fluid therapy, as well as anti-inflammatories and broad-spectrum antibiotics for metritis are usually indicated. Some cases will, at least temporarily, respond to medical management and the use of a GnRH agonist such as Suprelorin®. Other cases require surgical intervention. In chronic or recurrent cases, surgical salpingohysterectomy may be indicated.

Preventative strategies include husbandry and environmental practices such as optimising nutrition, supplementing with calcium and vitamin D3 if necessary, managing photoperiod and other external drivers of reproductive activity. GnRH agonists such as deslorelin are also helpful and should be used at least annually in birds that have undergone salpingohysterectomy to prevent ectopic ovulation and coelomitis[3]. Some clients routinely have Suprelorin® implants placed in backyard chickens as a preventative measure.

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2. Small Mammals

2.1 Uterine Adenocarcinoma in Rabbits

Uterine adenocarcinoma is the most common neoplasm of female rabbits with a reported incidence rate of up to 80%. It is encountered frequently in middle aged does but can be seen as young as one year of age[6].

This neoplasm progresses insidiously until clinical signs such as haemorrhagic vaginal discharge, decreased fertility in breeding animals, or respiratory distress in advanced cases

are observed. Uterine adenocarcinoma is slow to metastasise but pulmonary metastases may be seen after one-two years[7]. Affected rabbits often show no clinical signs for months to years or there may be non-specific signs of illness like inappetence, weight loss or episodes of gastrointestinal stasis. Careful abdominal palpation will often indicate uterine abnormalities such as irregular thickening or masses.

Suspicion of uterine adenocarcinoma should prompt the clinician to pursue abdominal ultrasound for confirmation. While definitive diagnosis requires histopathological examination post-ovariohysterectomy, uterine enlargement and masses can be seen on ultrasound. Thoracic radiography is also recommended to rule out pulmonary metastases and this is considered mandatory if surgery is elected.

Surgical ovariohysterectomy is a curative treatment provided it is performed before metastasis occurs. Once pulmonary metastases have occurred the only options are short term palliative care, which may not be viable in every case, or humane euthanasia. Uterine adenocarcinoma is easily preventable by routine desexing of female rabbits between four and six months of age.

2.2 Ovarian Cysts in Guinea Pigs

Ovarian cysts are extremely common in entire female guinea pigs, particularly those aged over one year. Reported incidence rates range from 58-100%[8].

There are three types of cysts a guinea pig may develop including serous cysts (non-functional), follicular cysts (functional) and paraovarian cysts, which are rare. The type of cyst is not usually determined in a clinical setting but has some relevance to treatment as serous cysts are most common[8]. Ovarian cysts are typically bilateral and are commonly associated with concurrent uterine pathology such as cystic endometrial hyperplasia, endometritis or leiomyoma[9,10].

The pathogenesis is not fully understood but is believed to involve hormonal dysregulation. Clinical signs include bilateral, symmetrical, atraumatic alopecia and in some instances there is also pruritis (due to pressure of cysts on the abdomen), abdominal distension, there may be vulval discharge when there is concurrent uterine pathology and in some cases decreased appetite and lethargy as ovarian cysts can also be painful. In a number of cases, guinea pigs may show no clinical signs, especially in the early stages. Sometimes ovarian cysts are picked up incidentally on exam during abdominal palpation.

Diagnosis is based on abdominal palpation and confirmation with imaging modalities such as radiography and ultrasound, ultrasound being the preferred imaging modality. Ultrasound, performed under sedation or general anaesthesia also facilitates percutaneous ovocentesis, which is sometimes useful for temporary drainage of large ovarian cysts to improve patient comfort and reduce abdominal distension. It is also recommended to drain large cysts before surgery to improve surgical access and patient ventilation[10,11].

Treatment of choice is ovariohysterectomy, which provides a definitive cure[9,11]. Hormonal therapies using GnRH analogues such as deslorelin may temporarily reduce cyst size in the case of follicular cysts but are not curative.

Prevention involves elective desexing, ideally before six months of age, if possible as early as two-three months of age as sows reach sexual maturity by two months. Flank ovariectomy/ovariohysterectomy is a less invasive and highly effective method for elective spey in guinea pigs that also allows removal of most of the uterine horn.

2.3 Hyperoestrogenism in Ferrets

Hyperoestrogenism is a common condition in intact female ferrets (jills) that results from persistent oestrus. Ferrets are seasonal polyoestrous induced ovulators and if mating does not occur, they remain in oestrus causing elevated oestrogen (oestradiol) levels to persist, leading to bone marrow suppression of blood cell precursors, subsequent anaemia and ultimately, in advanced cases pancytopenia[11]. It is generally seen in young females. Hyperoestrogenism can also be a result of ovarian remnant syndrome[12].

Clinical signs include pronounced vulvar swelling, bilateral truncal alopecia that may be pruritic and if anaemia has developed there may be pallor and lethargy. Petechiae can be seen with thrombocytopaenia[11].

Diagnosis is based primarily on clinical history and physical examination, as well as haematology demonstrating non-regenerative anaemia and possibly leukopaenia, and thrombocytopaenia[11]. At a minimum, jills with compatible clinical signs should have a packed cell volume (PCV) checked, especially before proceeding with ovariohysterectomy surgery.

Treatment options include surgical desexing via ovariohysterectomy or chemical desexing by inducing ovulation with GnRH analogue deslorelin (Suprelorin® implant), inducing ovulation with a vasectomised hob or breeding. Severely anaemic ferrets with a PCV < 15-20% require a blood transfusion[11].

Prevention is achieved by surgical or chemical desexing using a Suprelorin® implant at approximately 6 months of age. Suprelorin® implants have been shown to be an effective method of chemical sterilisation for up to 43 months in jills but should be repeated annually as the duration of action can be shorter in some individuals[13].

2.4 Mammary Neoplasia in Rats and Mice

Mammary tumours are frequently encountered in rats and mice, though their biological behaviour differs between species. Both sexes are affected but females are over-represented. In rats, mammary tumours are predominantly benign fibroadenomas that despite being benign grow to a significant size, impeding mobility and often become traumatised and ulcerated. In mice, mammary tumours are mostly malignant adenocarcinomas. Mammary tumours in rats have been shown to be under the influence of prolactin[11]. Oestrogens are also believed to play a role and adipose tissue is a major source of extra-glandular oestrogen, therefore body condition is another important risk factor[14].

These tumours can arise anywhere along the extensive mammary chain, which spreads from the neck to the inguinal region and dorsally to the shoulders and ilium[11]. Clinical signs include palpable subcutaneous masses, often rapidly enlarging in size. There may or may not be secondary skin trauma.

Diagnosis is based on physical examination, and histopathology post-surgical excision confirms tumour type. Fine needle aspirate samples for cytology are poorly sensitive for differentiating between benign and malignant mammary tumours.

Treatment for benign mammary tumours is surgical excision, and prognosis depends on tumour type and completeness of removal. Measures that can be taken concurrently include ovariohysterectomy/ovariectomy or placing a Suprelorin® implant to reduce recurrence[11]. Ongoing treatment with cabergoline may also help to reduce recurrence due to its prolactin inhibitory effects. Whilst surgical excision of mammary adenocarcinomas is possible, it is more challenging to achieve surgical margins, there is typically rapid tumour regrowth and often concurrent metastases[11].

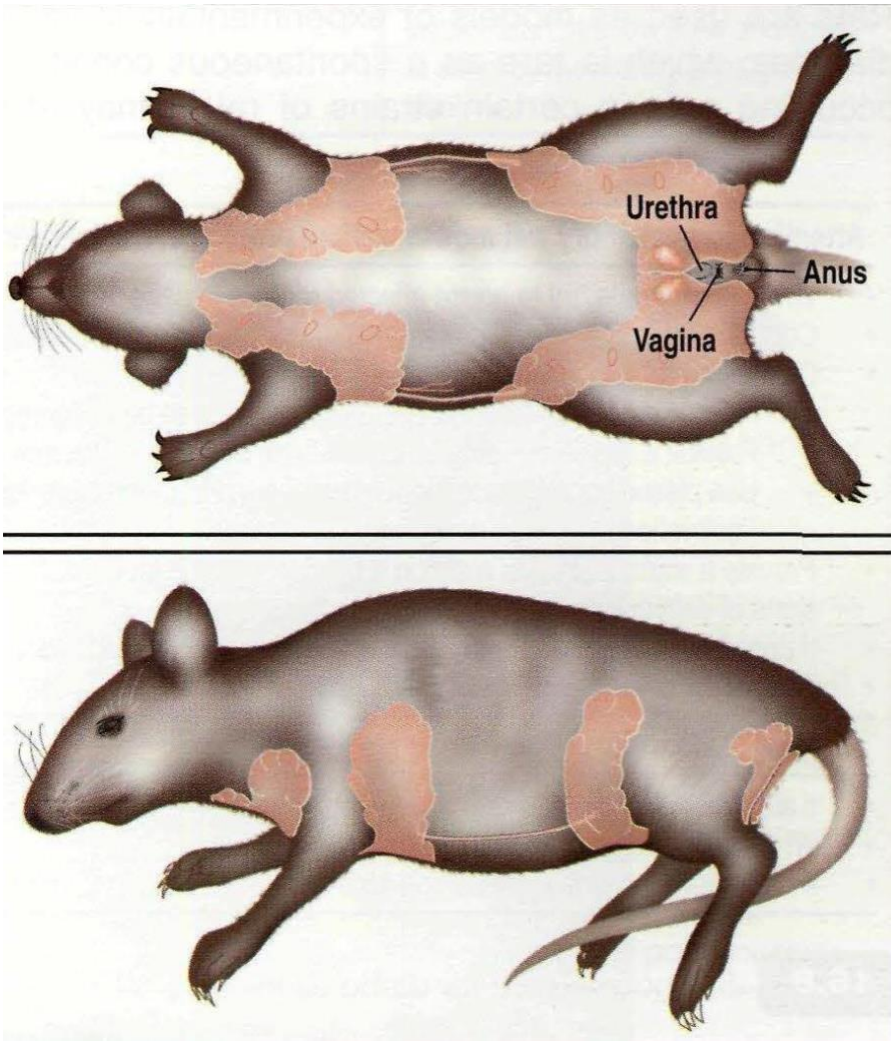

Fig. 1. Mammary gland anatomy of a female rat. *Source: BSAVA Manual of rodents and ferrets. 2009.*

Early desexing, ideally by or at three months of age, particularly in rats, has been shown to significantly reduce the incidence of mammary neoplasia. Female rats that underwent ovariectomy at 90 days of age had significantly lower incidence of mammary tumour development (4% vs 47% in entire females)[11,15]. Desexing can be achieved surgically by ovariectomy or ovariohysterectomy or chemically using a 4.7mg deslorelin implant, which

needs to be repeated every 12 months but surgical desexing has been shown to be the most effective method of preventing mammary tumours in female rats[14].

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3. Reptiles

3.1 Cloacal Prolapse

The cloaca is the common passage where urogenital and gastrointestinal tracts meet. Cloacal prolapse is a common emergency presentation in reptiles occurring when there is pathology of the internal structures of the cloaca, the cloaca itself or intracoelomic disease or increased intracoelomic pressure causing the cloaca, colon, reproductive tract, urinary structures or multiple organs to protrude through the vent[16].

Cloacal prolapse is a sign of an underlying condition and usually excessive straining. Conditions commonly associated with cloacal prolapse include gastrointestinal parasitism, constipation, cloacoliths/uroliths/enteroliths, reproductive disease, sexual overactivity, dystocia, hypocalcaemia, obesity, muscular weakness or spinal abnormalities[16].

Signalment varies but is often seen in young to middle-aged lizards and chelonians of both sexes. It is also seen in pythons but less commonly[16]. Clinical signs are obvious and include a red, swollen mass protruding from the vent.

Diagnosis is primarily visual but should include investigation of underlying causes with faecal testing (faecal floats, wet preparations), imaging including plain radiography, potentially contrast radiography, ultrasound and possibly cloacoscopy, and bloodwork to look for indicators of infection and organ dysfunction.

Initial treatment is aimed at reducing the prolapse. Swelling should be reduced with a supersaturated solution such as a concentrated sugar solution. Under sedation or anaesthesia the prolapse needs to be cleaned and reduced, ensuring that individual prolapsed cloacal components are identified and noted during this process if applicable. This also helps to narrow down differential diagnoses[16]. For example intestinal prolapse is more likely to indicate parasitism or other intestinal pathology. The prolapse can then be reduced into the vent and stabilised temporarily with a purse string or transverse sutures. Analgesia is also important and antibiotics may be required where there has been prolonged exposure and secondary contamination of tissue[16].

Long term management and preventing recurrence is focused on addressing the underlying cause. Prognosis varies with severity and underlying cause but if treated promptly and the underlying cause can be addressed and remedied, prognosis is usually good.

As is the case for many diseases in reptiles, cloacal prolapse often reflects suboptimal husbandry. This may pertain to factors causing nutritional secondary hyperparathyroidism (NSHP) such as inadequate UVB, heating or calcium in the diet or poor hygiene practices promoting parasite burdens. Therefore it is not surprising that prevention focuses on optimal husbandry, parasite control, and nutritional management.

3.2 Pre-Ovulatory Follicular Stasis (POFS) in Bearded Dragons

Pre-ovulatory follicular stasis (POFS) is a common reproductive disorder in captive female bearded dragons. It is also seen in other lizard species and sometimes chelonians, but it is predominantly observed in bearded dragons. It occurs when follicles develop on the ovary but fail to ovulate or be resorbed[17]. Follicles accumulate and over time become inflamed, necrotic and infected[17].

POFS is poorly understand but frequently appears to develop in bearded dragons subject to inappropriate environmental conditions (temperature, humidity, UV lighting) and/or poor nutrition[17,18].

Signalment includes adult female bearded dragons that have reached sexual maturity (12 months). Clinical signs are vague but include lethargy, anorexia, coelomic distension, weakness and reluctance to move. Follicles can usually be palpated in the coelom on physical exam[17]. Gentle palpation is essential to prevent follicle rupture and yolk peritonitis.

Diagnosis is made through radiography or, more sensitively, via ultrasound showing enlarged, retained follicles. Blood work is also recommended to look for inflammatory markers as well as to assess overall metabolic health and organ function, particularly prior to proceeding with surgery.

Initial treatment focuses on medical stabilisation with supportive care including fluid therapy (SC, IO, IV), nutritional support, anti-inflammatories and antibiotics. Once the patient has been stabilised surgical intervention via ovariectomy or ovariosalpingectomy to remove the retained follicles is necessary[19].

Prevention involves ensuring optimal husbandry, especially correct thermal gradients, UVB exposure, and a nutritionally balanced diet. Some clinicians recommend prophylactic ovariectomy for bearded dragons.

Conclusion

Reproductive disorders are common across exotic species and present unique diagnostic and therapeutic challenges. It’s important for clinicians to be aware of the common reproductive disorders affecting birds and exotic pets and to be able to address them accordingly. Even in cases where referral may be required for further diagnostic investigation or surgical treatment, being able to diagnose and stabilise exotic patients with reproductive disease can be imperative in increasing their chance of recovery and survival.

Appropriate age desexing in applicable species, ensuring optimal husbandry, nutritional management, and client education remain cornerstone strategies for prevention. Prompt diagnosis and intervention can significantly improve outcomes.

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