Diagnosis, Differential Diagnosis & Treatment of Abdominal Swelling

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Birds lack a diaphragm and so do not have an abdomen and thorax, but a coelomic cavity containing all the organs. In many situations this cavity is (incorrectly) referred to as the abdomen, more for the convenience of a comparison to mammalian problems.

If the bird has **Abdominal Swelling** look for the following key features.

- Faecal matting or collecting at the vent or ventral tail base
- Convex abdominal profile
- Perching problems as the distended abdomen protrudes between the legs
- Tail bobbing as the inflation of air sacs is compromised
- In advanced/chronic cases the skin may change – excoriation, ulcers or xanthomatosis

Diagnostic Approach

- Collect History
- Palpate abdomen
  - take care too vigorous may rupture an air sac and cause asphyxiation if fluid is present
  - palpation under ventral edge of sternum should not elicit pain, if pain is present, suspect hepatic problems
- In small birds, moistening the ventral abdomen with alcohol should allow you to view abdominal contents and detect an enlarged liver or intestines.
- Observe behaviour
- Perform abdominal aspirate and stains of smears
  - Gram Stain
  - Cytology stain e.g Diff Quik
- Radiographs (high or ultra-detail film)
  Plain
  Barium
- Clinical Pathology
  - Complete Blood Count + Biochemistry (include Bile Acids if possible)
  - Chlamydia screening
  - Blood lead & Zinc levels
- Endoscopy +/- Exploratory Surgery

Differential Diagnosis:

1. **Fluid**
   - **Ascites**
   - **Peritonitis**
2. **Fat**
3. **Egg**
   - **Retained Egg/Point-of-lay**
   - **Egg binding**
4. **Abdominal Hernia**
5. **Enlarged Organ**
   - **Liver**
   - **GIT**
   - **Reproductive System**
   - **Neoplasia**

1. Fluid

a) Ascites may be associated with cardiac disease (Right-sided or generalised), Liver disease or Liver congestion

b) Peritonitis may be from a septic or non-septic origin. Common causes are: egg yolk peritonitis; perforation of intestinal wall; infections caused by bacteria or fungi.

Clinical Signs

Dyspnoea (tail bobbing)
Poor exercise tolerance
No fat deposition at other sites

Diagnosis

History
Abdominoscentesis with Fine Needle Aspiration & smears of fluid
Internal organs are less distinct on palpation

2. Fat

Obesity is a common problem in many species, particularly birds with little opportunity for exercise and fed high fat diets containing seeds such as Sunflower, Safflower or peanuts.

Common species where this is encountered: Sulphur Crested Cockatoo; Galah; Budgerigar; Quarrion; Amazon Parrots.

Clinical Signs

Dyspnoea (tail bobbing)
Poor exercise tolerance
Fat deposition in various sites subcutaneously (lipomas & xanthomas)

Diagnosis

History
Abdominoscentesis with Fine Needle Aspiration & smears from masses
Internal organs are less distinct on palpation

Lipaemia on blood sampling – but may not show increased liver enzymes even, with fatty liver syndrome

Treatment

Dietary management (decrease fat, Increase fibre such as green leafy vegetables)
Gradual increase in exercise
Beware sudden bouts of anorexia may precipitate fatty liver syndrome

3. Egg

a Physiologically Retained Egg or on the Point-of-lay

There may not be a problem and the bird is undergoing normal egg laying and the egg is still having the shell deposited while it is in the shell gland.

History

Breeding in normal season
Not a high risk species for egg binding
No sudden onset of cold weather
Diet contains calcium and is consumed well
This is the first egg to be laid
The bird is not distressed

Clinical Signs

Bird is not depressed
Bird is perching normally with normal behaviour & posture
No cyanosis of legs
Temperature of feet is normal
No lameness or perching with base-wide stance
Abdominal distension
Palpable egg in oviduct

Treatment

Close observation

b Egg binding

History

Breeding out of normal season
More common in small birds (Quarrion, Budgerigar, Lovebird, Canary, Finches)
Sudden onset of cold weather
Diet low in calcium
Cessation of egg laying when more would be expected

Clinical Signs

Depression – "Sick Bird Look"
Sitting on bottom of the cage
Upright stance & tenesmus (Penguin-like posture)
Increased abdominal effort (tail wagging/bobbing)
Cyanosis of legs from vascular congestion caused by retained egg
Feet may feel cooler than usual
Lameness or perching with base-wide stance
Abdominal distension
Palpable egg in oviduct

Treatment

Provide a warm, humid environment that is stress-free.
Provide Fluid therapy to control dehydration & shock
Gently palpate egg, try to expel – use extreme care to apply pressure at left & right cranial pole of egg
and in a ventral direction – pressure in a dorsal direction may exacerbate renal problems
Aspirate egg contents (per cloaca or trans-abdominal) to collapse egg
Dose with Calcium drops PO
A small number will require surgical intervention
The smaller the bird, the more urgent is the need to relieve the problem, as they have limited energy reserves

4. Abdominal Hernia

Herniation of abdominal wall muscles may contain viscera, especially intestines. This may result in changes to consistency of faeces and resemble diarrhoea. Xanthomatosis may be present concurrently with a hernia.

Many birds feel as if they have a hernia but it is actually a thinning of the abdominal musculature.

Common in female parrots particularly Galahs, Sulphur Crested Cockatoos and Budgerigars. More prominent during the breeding season and in birds that are obese. May be associated with ovarian dysfunction.

Other less common causes of acquired herniation:

trauma
egg binding
abdominal mass placing pressure on abdominal wall
endocrinopathy (rare - e.g. Cushing's)

Clinical Signs

separation of abdominal muscles at ventral midline
convex abdominal profile in which viscera and/or fat may be palpable

Diagnosis

palpation abdomen – it is doughy & can feel loops of bowel
radiographs
loss of abdominal profile
organ displacement
viscera present in hernial defect (contrast medium may assist)
Microhaematocrit
lipaemia present
Abdominal aspirate
Gram stain to rule out peritonitis etc.
wet smear or Sudan IV - fat globules present

Treatment

Control diet (reduce obesity)
Remove or minimise stimuli that promote breeding (human interaction, mate, and day length)
Avoid the temptation to perform surgical repair
In high-risk species, consider oviductectomy once obesity is controlled

5. Enlarged Organ

a **Hepatomegaly**

Viral – Pacheco's or Avian Herpesvirus
Chlamydophila
Bacteria – Salmonella, Colibacillosis, Yersinia
Parasites – Sarcocystis, Atoxoplasma
Fatty Infiltration
Neoplasia – Bile Duct carcinoma, Lymphocytic leukaemia

Common Diagnoses: Parrots - chlamydiosis or fatty liver
Canary & Finches -hepato-splenomegaly due to lymphocytic leukemia, malaria

b **Gastrointestinal Tract**

Heavy Metal Poisoning
Bacterial Enteritis (e.g. Clostridia in Lorikeets)
Proventricular Dilatation Disease
Occlusion of intestinal lumen
- Parasites (Ascarids, Tapeworms, Gizzardworms)
- Foreign Body
- Enterolith

Stenosis of intestinal lumen
- Tumour
- Granuloma (Mycobacteria)
- Stricture

Extraluminal Compression of intestinal lumen
- Volvulus
- Intussusception
- Hernia

Cloacal Distension
- Papillomatosis
- Cloacalith

Common Diagnoses: Parrots – Heavy Metal Poisoning, Ascarids
Canary & Finches – Tapeworms, Gizzardworms

c. **Reproductive System**

- Female
  - Egg peritonitis
  - Cystic Ovary
  - Neoplasia of Ovary – carcinoma, adenocarcinoma
- Male
  - Physiological enlargement during breeding season
  - Neoplasia of testis – common in Budgerigar

d. **Neoplasia**

Other than the neoplastic diseases listed above, some of the common tumours encountered are:
- Renal adenocarcinoma – common in Budgerigar
- Intestinal leiomyosarcoma
- Intestinal carcinoma or adenocarcinoma
- Hepatic adenocarcinoma

Appendix 1

Diseases to consider with Liver Disease

Infectious Agents

(a) Bacteria - coliforms, Yersinia, Salmonella, Streptococcus, Staphylococcus
ie. any bacteria capable of causing septicaemia

(b) Chlamydia (*Chlamydophila*)

Chlamydia psittaci is a common cause of a septicaemic syndrome in parrots and pigeons, with occasional outbreaks in finches. It has recently been reclassified as a modified Gram negative Bacterium – *Chlamydophila psittaci*

Differential Diagnosis in Birds
The clinical and pathologic presentation of chlamydiosis is so variable that it can normally be ruled out only with laboratory investigations. The more common rule-outs include infections with Herpesvirus, Paramyxovirus, Influenza A virus (none of these three are currently reported in Australia) and Enterobacteriaceae, particularly salmonellosis.

CNS signs should be differentiated from Newcastle disease and salmonellosis.

Conjunctivitis in ducklings and goslings needs to be differentiated from Influenza A infection and mycoplasmosis. In parrots and pigeons the differential diagnosis should include haemophilus and mycoplasmosis.

Clinical signs
The symptoms will vary with the age of the bird and the region in which the chlamydia localise. It is not uncommon for the chlamydia to be seen after the bird has been immunosuppressed by a concurrent disease.

Chlamydia is the most common cause of air sacculitis and septicaemia in Australian parrots and pigeons. Occasionally seen in Budgies and finches. Generally it not as acute an onset as with bacterial septicaemia. Associated with times of stress in an aviary. Very common in Neophema (Bourke, Scarlet-chested, Turquoisine, Elegant, Blue-wing etc.) and Polytelis (Princess, Superb, Regent) parrot species. Outbreaks begin in late Summer, peak in Autumn and remain high throughout Winter.

Acute infections resulting in death are more likely in young psittacines and in non-psittacine species other than the chicken and pigeon.

Onset can be variable form acute to chronic. Usual incubation is 7-14 days but can be as short as 2 days or as long as 98 days.

Four Chlamydia Syndromes: Peracute; Acute; Chronic; Asymptomatic Carrier

Clinical Signs

Peracute

Sudden Death
Death with few premonitory signs of illness

Acute (any combination of the following signs)

Soiled vent	Listlessness
Diarrhoea	Ruffled feathers
Inappetance	Dyspnoea (tail bobbing)
Reluctant to move	Blepharitis/conjunctivitis
Prostration	Ocular/nasal discharge (serous or mucopurrulent)
Convulsions	Weight loss

Chronic

Rhinorrhoea	Wasting of pectoral muscles (prominent keel bone)
Cachexia	Ruffled feathers/huddled posture
Soiled vent	Feather loss around one or both eyes
Blepharitis/conjunctivitis	Nostrils plugged with exudate
Swollen eyelids/cloudy corneas	Retarded growth of young birds

Asymptomatic Carrier

No clinical signs but intermittently shed chlamydia

Factors Affecting Prognosis

Interspecies transfer of chlamydia (in aviaries and pet shops) can change the physico-chemical properties, antigenic composition, toxic components and the host spectrum of the organism.

Surveys indicate that between 30 and 70% of the birds (mostly parrots) have anti-chlamydial antibodies. Clinical disease is precipitated mainly by husbandry and management procedures.

Chlamydia can usually be detected in the faeces ten days prior to the onset of clinical signs.

Carriers may begin to shed the organism following a stressful event.

Antibody production with an active infection may be poor, and birds that survive infection are often still fully susceptible to disease.

Diagnosis

(1) Clinical Examination - this is where initial suspicions will be raised. Definitive diagnosis is not possible simply with clinical examination but it will form the basis for further examination. Psittacosis is a major problem in many Australian parrot species.

The typical signs include:

Conjunctivitis
polyuria
diarrhoea
rhinitis,
blepharitis
conjunctivitis
keratitis
plugging of nares
sneezing

(2) Clinical Pathology.

Many cases of chlamydiosis can only be confirmed in the laboratory. Some of the following tests are available in practice, others are only suitable for major laboratories. Pathologists still disagree as to the most useful test for all clinical cases as some birds are intermittent shedders but are negative on serology. In contrast, serology may be positive in birds, which are not currently shedding. To detect whether a bird is infected or currently shedding chlamydia, a combination of antibody and antigen detection must be used. Until recently most of the available diagnostic tests were only suitable for post-mortem diagnosis. Recently there has been an expansion of the ante-mortem diagnostic techniques.

Full Blood Count and Biochemistry profile.

leukocytosis with a left shift
toxic heterophils
monocytosis
low PCV
regenerative anaemia
raised SGOT.

Antibody Detection

1 Complement-fixation Test (CFT) - direct or indirect
2 Latex Agglutination (LA)
3 Both CFT and LA are useful diagnostically but are only historically applicable, as you need to demonstrate a three to fourfold increase in titre between acute and convalescent samples.
4 Competitive BELISA is of use but the criteria for a positive sample are still being researched.
5 Indirect ELISA is currently being researched at the University of Minnesota.
6 Elementary Body Agglutination titre. This technique is being developed as a screening test for both clinically ill and normal birds. Early research suggests it is of use in psittacines but may not be applicable for pigeons. In combination with culture and LA it may be of use in the future.

Chlamydia Organism (antigen) Detection

1 Immunofluorescence. This is can be used to differentiate live organisms from dead ones.
2 ELISA. Available as Oxoid Clearview tests. Both are designed to detect chlamydia in humans, particularly the venereal form. The presence of some Gram negative bacteria can give a false positive reaction if faecal smears are used. There needs to be a correlation with clinical signs. These tests are not suitable for screening tests.
3 Isolation and growth of chlamydia from faeces or tissue by tissue culture or egg inoculation.
4 PCR Gene probe. This Technology is used to amplify a portion of the DNA of chlamydia, enabling very low levels of the organism to be detected. Non-viable organisms can be detected with tremendous sensitivity and specificity (approx. 90%). Research is being carried out to eliminate false positive reactions – in many cases this has been due to contamination during collection and handling as even a small amount of contamination will be significant when amplified.

(3) Radiology - plain radiographs in some instances may show splenic enlargement. This does not allow a specific diagnosis, as there are several causes of splenic enlargement. This finding merely adds support to a suspicion.

(4) Post-mortem Examination

The lesions in most avian species are similar.

Common lesions:

Fibrinous exudate on several serosal surfaces
Splenomegaly (more common in psittacines than pigeons)
Hepatomegaly (from generalised to focal necrosis. Rounded edges)
Airsacculitis
Peritonitis
Pulmonary congestion
Catarrhal enteritis
Flaccid bowel with watery/gelatinous contents

Other causes of similar Post Mortem signs:

E. coli and other Enterobacteriaceae
Salmonella
Pasteurella
Yersinia
Mycoplasma

(c) Viruses - several viruses after their viraemic stage will localise in hepatic tissue.

Herpesvirus

(i) **Parrot herpes - Pacheco's disease** can occur in all species except Patagonian Conure (Cyanoliseus patagonus) which is recognised as a carrier. This is associated with few signs other than sporadic outbreaks of sudden deaths with no premonitory signs. Mortality rates can be high.

(ii) **Falcon Herpes** causes an acute and fatal syndrome in raptors including owls. Does not appear to be a problem in pigeons. Diagnosis - by histopathology, from biopsy or post mortem collection of samples, revealing inclusion bodies in hepatocytes and cells of reticuloendothelial system eg. spleen.

(iii) **Pigeon Herpes** In pigeons it causes an acute syndrome with low mortality but transmission to psittacines causes an acute syndrome with high mortality.

(iv) **Duck Viral Enteritis** is carried by wild ducks and may spread to any captive waterfowl they contact.

Signs at P.M.:

generalised tissue haemorrhage
GIT -haemorrhage & diphtheritic membranes
Liver- miliary necrotic foci

(v) **Reovirus** can cause infectious myocarditis in goslings 5-21 days old

Clinical signs

sudden death
anorexia
polydipsia
conjunctivitis
mild nasal discharge
dyspnoea
diarrhoea - only occasionally (watery, grey/white)

Diagnosis

Viral isolation
Cloacal swabs
samples from affected organs

(vi) **Retrovirus: Lymphoid Leucosis** is primarily a disease of chickens and other poultry. On rare occasions it has been reported in: Budgerigars (*Melopsittacus undulatus*) and raptors such as the Black-shouldered Kite (*Elanus notatus*)

(d) Protozoa

(i) *Trichomonas* spp. in Budgerigars and pigeons.

Diagnosis

Examine direct smears
histopathology

(ii) *Histomonas* spp. This is a parasite of the caecae and liver. It occurs mainly in peafowl, grouse and turkeys and may be seen in other gallinaceous birds such as Guinea-fowl, quail, chickens and pheasants. It may affect any age group but is most common less than 12 weeks. Common name for this disease is Blackhead and is associated with the ingestion of eggs of caecal worms of the *Heterakis* genus (eg. *H. gallinarum, H. isolonche*) that act as a paratenic host to transport *Histomonas meleagridis* past the acid environment of the proventriculus. Losses can be heavy, associated with *Heterakis gallinae* damage to caecum

Clinical signs

ascites
polydipsia
hepatomegaly
diarrhoea
    brown-black to watery yellow
    occasionally blood present
weight loss
deaths (up to 50% of a flock in a severe outbreak)

Diagnosis

history
faecal flotation for Heterakis

Post Mortem Examination

areas of focal necrosis in liver of gallinaceous birds, examine direct smears for motile protozoa especially Peafowl (*Pavo cristatus*), Californian Quail (*Lophortyx californica*), Chukar Partridge (*Alectoris graeca chukar*)
enlarged caeca (up to 10 times normal)
caecal contents -blood and fibrinous exudate
caecal nodules, ulceration, hyperaemia and oedema
caecal smear - take sample from edges of lesions look for motile flagellated protozoans
hepatomegaly & liver has large areas of necrosis
yellow circumscribed ring lesions (not in chickens)

(iii) ***Atoxoplasma***

Present in many bird species but only regarded as a pathogen in Canaries and other passerines, particularly Gouldian Finches fostered under Bengalese Finches. Primarily a disease of fledgling birds or juveniles up to 9 months of age. Mortality rates may approach 80%. Adults will shed the oocysts for months but remain asymptomatic.

This is very host specific parasite. Canary pathogens are not a problem in finches and vice versa. It is regarded as having a direct life cycle with no intermediate host being involved. Paratenic hosts (mites and other insects) may play a role in transmission.

Clinical signs

anorexia
depression
diarrhoea
may occur in birds not shedding oocysts in their faeces

Diagnosis

Spray anterior aspect of abdomen with alcohol to view contents (transillumination may help visualisation)
enlarged liver, distended intestines
faecal flotation to demonstrate oocysts - smaller oocysts than Eimeria or Isospora
Post mortem - lesions may vary with different bird species
impression smears from heart, liver or pancreas (Giemsa)
splenomegaly
liver - white pinpoint necrotic foci
pancreas - pale, swollen, nodular

(iv) ***Cochlosoma***

This is a free-living flagellated intestinal parasite that is transmitted via the oral-faecal route. It is primarily a problem in juveniles and nestlings (6-12 weeks old) as adults appear to be asymptomatic carriers. More commonly seen in Australian finches, especially the Gouldian Finch (*Erythrura gouldiae*), that are being fostered under Bengalese Finches (*Lonchura domestica*, *L. striata*) which are asymptomatic shedders.

Clinical signs

weight loss
depression
dehydration
passing whole seed in the droppings
sudden death

Diagnosis

direct warm saline smear of fresh droppings
motile protozoan with six anterior flagella and an anterior ventral sucker
post mortem examination
enteritis
intestines contain yellow material or whole seed
direct saline smear of intestinal scrape

(v) ***Cryptosporidia***

This is a common problem in nearly all birds as it is not host specific. Mammalian forms are not pathogens in birds. It is an opportunistic pathogen and is normally regarded as a secondary invader. It has a direct life cycle. This pathogen has a preference for invading epithelial surfaces and is most commonly a problem in the lining of the gastrointestinal, respiratory and urinary tracts.

This pathogen has a preference for invading mucosal epithelial surfaces and is most commonly a problem in the lining of the gastrointestinal, respiratory and urinary tracts.

Clinical signs

depression
anorexia
conjunctivitis
nasal discharge
coughing/sneezing
enlarged periorbital sinus
dyspnoea
audible respiratory sounds
diarrhoea
death

Diagnosis

direct warm saline smear of scrapings from epithelial surfaces either ante-mortem or post-mortem
direct warm saline faecal smear
smallest of all coccidian oocysts seen in birds
contain four naked sporozoites
The oocysts are shed in small numbers and are quite small, making detection difficult.
This may be improved by centrifuging the faeces after diluting in concentrated salt solution or using
Sheather's flotation.
Stained faecal/epithelial surface scraping smears
Modified Acid-fast
cryptosporidia stain pink against a blue background
Giemsa
Carbolfuchsin
Periodic Acid Schiff Auramine-O
Post mortem examination
excessive mucous in respiratory tract
dilated intestines containing yellow fluid

Aflatoxin

Toxins from Aspergillus flavus have been recorded to cause:

fatty liver
focal hepatocyte necrosis
cirrhosis
bile duct proliferation
poor growth
reduced resistance to infectious diseases.

History

feeding soaked seed

access to stale fruit or other soft foods
Musty odour to food
Food not stored properly.
Conditions to promote fungal contamination:
darkness,
humidity higher than 85%,
Optimum temperature is 25-30 C,
High Oxygen and low carbon dioxide concentrations ie good aeration.
High-risk foods: peanuts, nuts and cereals, bread, cheese, beans, fruit juice and meat.

Diagnosis
Toxin can be recovered from samples submitted.
Post Mortem Examination & Histopathology to detect:
- fatty liver
- focal hepatocyte necrosis
- cirrhosis
- bile duct proliferation.

50% of aflatoxin B1 will fluoresce bright yellow-green under U.V. light such as a Woods lamp - a rapid screening test but interpret with care, false positives and false negatives can occur.

Fat infiltration

History
Inappropriate diet (high in carbohydrate and fat and often protein as well)
combined with minimal to no exercise.
Be suspicious if bird is obese on examination.
Spend some time discussing diet - especially any "treats"

Liver biopsy may aid diagnosis. Take care as birds with liver disease may have slow clotting time.

Neoplasia

Occasionally reported in the literature. May be primary or secondary.

Diagnosis
- liver biopsy
- radiograph to assess cardio-hepatic silhouette.
- Aspirate any ascitic fluid for cytology.

Haemochromatosis (Iron Storage Disease)

Commonly seen in Mynahs overseas.
Occasionally in Birds of Paradise and Quetzals.
Associated with excessive Iron intake, which results in lysis of erythrocytes (low PCV). This is followed by deposition of haemosiderin in liver and secondary hepatic damage.

Clinical signs
- Dyspnoea
- Vomiting (in young birds)
- Ascites
- convex abdominal profile
- Weight Loss

Diagnosis
- Ascites - yellow fluid

modified transudate with mononuclear cells and macrophages
specific gravity 1.013 - 1.018
Radiograph - hepatomegaly
Blood collection
Low TPP,
Increased SGOT, SGPT, ALP, LDH and Bilirubin