

Avian Abdominal Fluid Cytology

Sue Jaensch

Collection of abdominal effusion for cytological analysis is a common procedure in avian medicine. It can be a highly diagnostically rewarding procedure, however expectations of this single test should not be exaggerated to clients.

As in companion mammal medicine, in many cases evaluation of avian abdominal fluid will lead only to a non-specific classification (transudate, modified transudate, exudates, haemorrhage), with further definition based on the predominant cell type. Do not be put off by this, as this type of definition, along with anatomical definition of the site of effusion (see below), can result in very useful information. In some cases, specific diagnoses can be provided such as with egg yolk peritonitis, septic peritonitis and exfoliative neoplastic disease.

Anatomy

There are 16 (potential) spaces where fluid can accumulate in the coelomic cavity.

8 airsacs

8 pleuroperitoneal cavities

The latter are divided as follows:

- Pericardial
- Left and right pleural
- Left and right dorsal hepatic
- Left and right ventral hepatic
- Intestinal

The pleuroperitoneal cavities have close associations with particular organs, specifically:

- Pericardial: heart
- Left and right pleural: lungs
- Left and right dorsal hepatic: left and right liver lobes
- Left and right ventral hepatic: left and right liver lobes, left ventral also proventriculus and ventriculus
- Intestinal: gonads and intestines with their suspending mesenteries. Abdominal air sacs also penetrate this cavity.

All these potential spaces are blind, except the left dorsal hepatic cavity, which connects with the intestinal cavity. The liver is effectively isolated from the rest of the abdominal cavity by the post-hepatic septum.

The most common sites for fluid accumulation across avian species are the hepatic and intestinal pleuroperitoneal cavities and airsacs.

Why is this anatomy important?

It is possible for disease or certain organs to have effusions confined to a single cavity.

- inflammation of intestinal or reproductive tracts in the intestinal peritoneal cavity
- effusions in the ventral hepatic space are classically associated with hepatic disease
- proventricular or ventricular perforation may be confined to the left ventral hepatic peritoneal cavity.

With advances in imaging, this is likely to become of increasing diagnostic importance, and may allow rapid narrowing down of a diagnostic investigation of an effusion. Note however, then the membranes separating these cavities are not robust, and after the initial phase of disease, effusion may be present in more than one cavity.

As a reflection of this fragility, fluid in the intestinal pleuroperitoneal cavity may gain access to the abdominal air sacs protruding into the intestinal space, and result in acute pulmonary distress.

Non-localised peritoneal effusions are generally inflammatory or malignant

Collection of abdominal fluid

A 21 to 25 G needle inserted on the midline is directed to the right side to avoid the ventriculus (but must rely on imaging and palpation to determine the site of the fluid accumulation and safe access points).

Peritoneal washes can be attempted in birds without effusions, but are difficult to achieve due to the tiny space present. Most likely will collect an airsac wash.

Little or no fluid can be collected from the abdomen of normal birds, but if collected has low cellularity: occasional mesothelial cell and macrophages.

Classification of effusions

Transudates:

- Odourless, transparent fluids with low cellularity ($<1 \times 10^9/L$), SG <1.020 , protein <30 g/L.
- Classically contain macrophages and mesothelial cells.
- May result due to oncotic pressure changes or circulatory disturbances.
- Differentials include hepatic cirrhosis, cardiac insufficiency, hypoproteinaemia
- Not all transudates result in abdominal distension, and collection may be difficult.

Modified transudates:

- Cell counts $1 - 5 \times 10^9/L$ protein usually <30 g/L, SG usually <1.020
- Mononuclear effusions with mesothelial cells and rare heterophils. Mesothelial cells may be reactive, possibly in sheets (DDx neoplastic cells).
- Occur with hydrostatic pressure changes or irritation of long standing transudates.

Exudates:

- Cell counts $> 5 \times 10^9/L$, protein $> 30 \text{ g/L}$, SG > 1.020 .
- Acute exudates are predominantly heterophilic, but macrophages are rapidly recruited and mixed cell inflammation occurs within hours of an acute onset. Lymphocytes and plasma cells may be present if more chronic.
- Frequently viscous and tend to clot.
- Differentials include septic peritonitis, egg related peritonitis, abdominal neoplasia. Ovarian cystadenomas in older hens are common causes of exudates.

Haemorrhagic:

- Erythrophagocytic phagocytosis and no platelets.
- Must differentiate pathological haemorrhage and peripheral blood contamination.

Aetiologies

Inflammatory

Result in protein rich effusion with fibrin, mesothelial cells and inflammatory cells. This contrasts with classical airsac inflammation with a dry purulent exudate

Infectious

Chlamydophila

Fibrinous peritoneal exudate, pericarditis, bronchopneumonia, nephrosis and splenitis. Fibrin exudation in the peritoneal and epicardial spaces. Systemic histiocytic inflammation.

Fungal

Usually extension of fungal airsacculitis or pneumonia. Acute: oedema, scattered heterophils and fibrin deposition. Chronic: yellow or white caseous exudate.

Protozoal

Serous and fibrinous serositis with *Toxoplasma* in passerines.

Viral (Uncommon)

1. EEE in neonatal and juvenile psittacines. Transudate to modified transudate. Affects all serosal surfaces, reactive mesothelial cells common. Hepatopathy and pulmonary oedema
2. Polyomavirus. Hydropericardium and ascites, possible subcutaneous oedema. Low cellularity. Widespread haemorrhages. Heterophilic serositis.
3. Herpesvirus (Pacheco's) Multifocal petechiation. Acute hepatic necrosis. Mild effusion (perihepatic spaces)
4. Systemic pox. Proliferative peritonitis.

Bacterial (Uncommon)

Usually secondary to gastroenteritis or perforation of the gastrointestinal tract. Acute to subacute: heterophilic infiltrates, oedema and fibrin deposition. Mycobacteria produces granulomatous inflammation with macrophages, multinucleate giant cells and lymphocytes and plasma cells.

Non-infectious

Egg yolk peritonitis.

Varies in severity from subclinical to life threatening. Ascites is most commonly seen with cockatiels and waterfowl. Small amounts of yolk material may be reabsorbed without clinical signs. Yolk material (fats and proteins), result in a histiocytic response. Fluid may be non-inflammatory or inflammatory, with or without sepsis. Yolk or fat globules are common (amorphous to basophilic, variably sized protein globules). Mild haemorrhage, and mild heterophilic inflammation may be present in non-septic forms. Birds often present in respiratory distress with large, distended abdomens. Coliform bacteria are the most common isolates in septic egg yolk peritonitis.

Visceral gout

Deposition of urates on serosal surfaces. Commonly pericardial and serosal surfaces of the proventriculus, ventriculus and liver. Degenerative heterophils may be present.

Haemorrhage

Trauma, neoplasia, toxins (rat bait etc). Evaluate for platelets and assess macrophage contents for an estimation of duration of the lesion.

Avocado toxicity

May have hydro pericardium due to myocardial disease. Low USG (<1.014), clear, may be infiltrate of heterophils.

Neoplastic

Neoplasia of the pleural / peritoneum are usually metastatic.

Lymphosarcoma

Common in psittacines and passerines. No definitive link to viruses at this time. Results in thickened and opaque mesentery

Oviduct and ovarian neoplasia: May implant widely in the peritoneal cavity.

Pancreatic neoplasia

Malignant gastric neoplasia: May implant on the serosa of the ventriculus, intestine, pancreas and lungs.