# Henrietta's Harrowing Heart

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**Signalment and Physical Exam**

Henrietta is an eighteen-month-old Cochin hen that presented for distended coelom, anorexia, and not defecating. Henrietta was quiet and dull with a poor body condition score; she had a very prominent keel with minimal pectoral muscle mass and she weighed 750 grams. Her mucous membranes were pale and she had a distended coelomic cavity that produced a fluid wave when gently palpated. Her heart rate was significantly elevated around 300-400 beats per minute, and she was in respiratory distress. Her feathers were unkempt, and tail feathers remained down throughout the exam. She also had extremely rough and scaly legs.

**History**

Henrietta was acquired from a backyard chicken breeder in Columbus, MS in October 2018. She was originally a part of the backyard flock, however after some health issues as a chick she became an inside chicken. She has her own room inside and lives with a rabbit and Great Dane.  She was on a diet of 7 grain scratch mixture with oyster shell supplements, apples, and apple sauce. She had hydrohen electrolytes added to her water every day. It is important to note that while Henrietta did have supervised outside time with the other chickens, she mainly resided inside and had access to the kitchen area where she was exposed to both cooking smoke and cigarette smoke. Henrietta also has a long-term history of sour crop, which was treated in January 2019 with ketoconazole and cisapride, and laying soft shelled eggs.

Henrietta presented to Mississippi State University College of Veterinary Medicine (MSU-CVM) Community Veterinary Services (CVS) on 11/13/2019 for a distended coelom, anorexia, and not producing feces. At that time, she had a 4-5-day history of inappetence, mucoid feces, inability to lay eggs, and labored breathing. The owners also reported that she had been lethargic and unsteady on her feet when she would attempt to walk.

**Diagnostic Approach and Considerations**

After Henrietta's initial physical examination on 11/13/2019, several differentials diagnoses were considered in regard to her severe ascites and respiratory distress, the top two differentials were egg binding or egg yolk peritonitis, with a lesser consideration given to viral diseases, such as, Marek's disease or avian influenza.

Both top differentials can be supported with radiographs of the coelomic cavity. Therefore, a lateral and ventro-dorsal image were taken. The radiographs revealed an unstructured interstitial coalescent alveolar pulmonary pattern within the pulmonary parenchyma bilaterally with decreased lung volume bilaterally. Significant decrease in serosal detail in the coelomic cavity and an enlarged hepato-cardiac silhouette was evident. For a diagnosis of either egg binding or egg yolk peritonitis, it would be expected to see either an egg or mineral opaque structures within the coelomic cavity, respectively. Neither of which appeared on the radiographs. Due to the radiographic evidence it was less likely that either of our current differentials were causing the clinical signs.

The radiographs of the coelomic cavity were unsupportive of the top differentials, so a coelomocentesis was performed. Around 180 mls of clear to straw colored fluid with minimal cellular debris was drawn from the coelom. Upon microscopic evaluation only 2-3 inflammatory cells and 2 clusters of gram-positive cocci were seen. These results definitively ruled out the current differentials and was evidence of disease processes involving the heart, liver, or diseases causing disturbances in oncotic pressure.

On radiographs, it was evident that there was pulmonary disease present. To help support this finding a choanal swab and gram stain was performed. The choanal swab revealed too numerous to count gram negative rods and the occasional gram-positive cocci. The normal flora

in the avian respiratory tract should be primarily gram-positive organisms. Since the swab showed an abundance of gram-negative organisms and with the support of the radiograph findings a bacterial pneumonia was diagnosed. A cloacal swab was taken at the same time and the gram stain was unremarkable.

Based off the first day of clinical presentation and diagnostics, Henrietta was sent home with oxytetracycline (16 mg/kg IM), meloxicam (1 mg/kg PO), and Ivermectin (0.8 mg/kg IM). The antibiotic was prescribed for the bacterial pneumonia and the meloxicam was prescribed to help alleviate any pain, discomfort, and inflammation. The Ivermectin was prescribed for the *knemidocoptes mutans* that were an incidental finding on the physical exam. At this time, the owners were warned that Henrietta had severe pathology and the prognosis was poor.

Henrietta presented the next week on 11/18/2019 for a recheck of her radiographs. During her physical exam, Henrietta still had a slightly distended coelom compared to her last visit, however her heart rate and respiratory rates were appropriate, and she was not in respiratory distress. On her radiographs, the previous pulmonary pattern was persistent and unchanged and there was an unchanged volume of coelomic effusion. During this visit, the fluid was unable to be removed via coelomocentesis. Due to the unchanged radiographs, Henrietta's antibiotic regimen was changed to include both oxytetracycline as previously prescribed and Clavamox (16 mg/kg PO). To help with the coelomic effusion Furosemide (1 mg/kg PO) was prescribed at this visit as an osmotic diuretic.

Henrietta presented for her third visit on 11/25/2019. On physical exam, Henrietta was bright and alert and was attempting to groom her feathers, unlike her previous visits. On her radiographs, her pulmonary pattern was still unchanged and her coelomic effusion was unchanged. Due to her unchanging pulmonary patterns and continued effusion it was decided to

continue her current medication as prescribed and add in enalapril (1 mg/kg PO). Enalapril was added to cause vasoconstriction and aid the hearts contractility. At this time, cardiac disease moved to the top of the differential list, however, if her radiographs remain unchanged after the addition of enalapril, our next most likely differential would be liver disease.

On 12/3/2019, Henrietta came in for her fourth visit. Her physical exam remains unchanged from her previous visit. It was elected to draw blood for a serum chemistry and complete blood count. Henrietta's send out bloodwork was received on 12/6/2019. She did not come in for a physical exam and her medications were adjusted based off her bloodwork which were as follows:

**Complete Blood Count:**

- TOTAL WBC'S (K/uL): 49.25 (12-30)
- ABS HETEROPHILS (K/uL): 18.2 (3-6)
- ABS LYMPHOCYTES (K/uL): 25.6 (7-17.5)
- ABS MONOCYTES (K/uL): 3.9 (0.15-2)
- REL MONOCYTES (%): 8 (0.1-7)
- PCV %: 36 (22-35)

**Serum Chemistry Profile with Electrolytes:**

- Calcium (mg/dL): 10.6 (13.2-23.7)
- Phosphorus (mg/dL): 3.5 (4.1-5.7)
- ALP (U/L): 1988 (10-106)
- Sodium (mEq/L): 153 (141.6-152.6)

Based on the bloodwork and the elevated liver enzymes it was elected to add on Milk thistle (5g/kg PO), vitamin E (200 mg/kg PO), and denamarin (60 mg/kg PO) as liver protectants.

On 1/7/2020, Henrietta came back for a recheck. On her physical exam, she was bright and alert. Her vitals were within normal parameters and on cardiopulmonary auscultation a gallop rhythm was heard, and crackles were present on the dorsal air sacs. Her coelom was mildly distended with a fluid wave present. A coelomocentesis was performed and 30 mls of

fluid was pulled off her coelomic cavity and the remainder of her physical exam was within normal limits. Her radiographs showed a normal pulmonary pattern and progressive coelomic effusion with an enlargement of the cardio-hepatic silhouette. Her current medications were continued including milk thistle, vitamin E, enalapril, furosemide, and denamarin as previously prescribed. At this visit, prednisolone (0.4 PO) and pimobendan (1.6 mg/kg PO) were added to her medication regiment to decrease the inflammation in her lungs and to increase cardiac contractility and decrease heart rate, respectively.

Henrietta returned on 1/28/2020, 2/19/2020 and 3/4/2020 for additional coelomocentesis where 120 mls, 180 mls and 120 mls of pure transudate was removed, respectively. A physical exam was not performed, and her medications were continued as prescribed at her previous visits. Henrietta continues to see her veterinarian on a regular basis for management of her presumed cardiac failure.

**Case Outcome**

Henrietta is currently doing well per her owner. Henrietta is still undergoing symptomatic treatment for her presumed heart failure. She is currently taking pimobendan and denamarin for her heart and liver pathology, respectively. She has coelomocentesis approximately every 7 days, but if her medications are not given, due to her aversion to eating them, there is an increase in how often Henrietta must receive her palliative coelomocentesis. At this time, it is unclear how long Henrietta can be maintained on her current therapy, however she has beaten all our expectations thus far.

**Pathophysiology**

There are a number of differences between the avian and the mammalian heart some of which have clinical implications in the pathophysiology and diagnosis of poultry cardiovascular

diseases.[1] As in mammals, it is four chambered and functionally divided into the right and left sides each consisting of an atrium and a ventricle.[1] The right ventricle is sickle moon shaped and surrounds the left ventricle. The wall thickness of the ventricle changes from the base to the apex of the heart.[1] The triangular right atrioventricular valve is muscular and unlike the right atrioventricular valve in the mammalian heart does not contain chordae tendineae.[5] The avian cardiovascular system is designed to match the high metabolic demands of birds, comprising a big heart, high heart rate, high cardiac output, and high blood pressure.[6] These unique high-performance features of the avian heart enable birds to fly, run, or dive.[6] Caged birds, in comparison with free living birds, are frequently compromised by restricted exercise, nutritional deficiencies and abnormal climatic conditions.[5] Combined with the birds physiologic makeup, the risk factor for cardiovascular disease in pet birds is significant.[5] Clinical signs that can indicate heart disease include dyspnea, periodic weakness, syncope, coughing, exercise intolerance, coelomic swelling, or lethargy.[6]

While there are a few structural differences between the mammalian and avian heart, the significant difference lies in the electrical conduction pathways of the heart. Investigation of the depolarization mechanism in the avian heart showed that primary waves of excitation originate from the apical regions of the interventricular septum.[2] Multi-focal depolarization activates the myocardial free walls, with the basal third of the septum the last region to be activated.[2] Furthermore, in addition to the right and left bundle branches, the avian conduction system also contains a middle, or recurrent bundle branch, and an atrioventricular purkinje ring.[2] The recurrent bundle branch separates from the bundle of His before the bifurcation of the right and left branches.[2] It then turns in course through the interventricular septum toward the aortic root at the base of the heart.[2] This branch, together with the AV purkinje ring, is intimately associated

with the right AV valve.[2] The arrangement is significant, as a conduction impulse will reach the muscular right AV valve, and effect active closure, before ventricular contraction is completed.[2] This requirement for an active phase of valve closure is not present in mammals.[2] It is possible that any process that interferes with the timing of these conduction events could predispose the bird to regurgitation of blood through an incompetent right AV valve and lead to hepatic congestion and ascites.[2] This is an important mechanism that when either cardiac or possibly pulmonary pathology are present can lead to right sided heart failure.

Diseases of the heart may occur as the result of congenital, infectious, toxic or idiopathic etiologies. A variety of cardiac abnormalities occur secondarily as acquired disease and/or compensation/decompensation due to other organ failure (i.e. lung and liver), neoplasia, or systemic infections.[5]

Avian cardiac diseases can be divided up into the following categories:

- Atherosclerosis[1,5,6]
- Congenital Diseases[5,6]
- Congestive Heart Failure (myocardial failure, valvular regurgitation)[5]
- Pericardial Diseases (pericarditis, pericardial effusion)[5,6]
- Myocardial diseases (myocardial failure)[5,6]
- Endocardial diseases (endocarditis, valvular insufficiency, valvular stenosis)[5,6]
- Circulatory Disturbances (shock, circulatory collapse)[5]

The pathophysiology of this specific case is most likely attributable to pulmonary pathology resulting in endocardial disease and valvular pathology. There are several possible mechanisms that can contribute to the process of heart failure and subsequent ascites in the avian

species. The pathogenesis can originate with a high basal metabolic rate induced by a number of factors including cold, moderate heat, activity, hyperthyroidism, elevated muscle mass and overeating.[2] It has been suggested that pathogenesis of pulmonary hypertension is associated with a vascular bed unable to accommodate increased cardiac output.[7] Elevated pulmonary arterial pressure caused by reduced pulmonary vascular capacity, polycythemia, increased blood viscosity, reduced erythrocyte deformability, pathological narrowing of lung capillaries and embolic blockage of the pulmonary circulation leads to a pressure overload in the right ventricle and, hence, right ventricular hypertrophy.[2] This leads to right ventricular failure, liver congestion and ascites.[2] The Frank Starling law of the heart describes an increase in contractility of the myocardium due to an elevated preload.[2] In terms of the right ventricle, preload is determined by cardiac venous return. This mechanism increases cardiac output by elevating stroke volume. If pulmonary vascular resistance increases, the afterload increases, leading to a pressure overload in the right ventricle.[2] One important consequence of ventricular hypertrophy is distortion of the atrioventricular valves and the production of regurgitant flow from the ventricle to the atrium during ventricular systole.[2] This is what then leads to backup of flow in the right atrium, subsequent congestion of the liver and then ascites.

Pulmonary hypertension (so-called pulmonary hypertension syndrome) may be the most common etiology connected with broiler ascites.[2] Pulmonary hypertension syndrome is a disease that occurs in some strains of fast growing chickens and turkeys which eventually leads to pulmonary hypertension, right heart failure, ascites and death.[9] Presumably the increased cardiac output associated with the fast growth and therefore the increased pulmonary blood flow results in a large increase of pulmonary arterial pressure due in part to a lack of pulmonary compensatory responses such as capillary recruitment and extension.[9] Right sided cardiac

hypertrophy may result from the pulmonary resistance increase that occurs as a consequence of the pulmonary capillaries acting as resistance vessels in birds.[2] This hypertrophy causes right atrioventricular valve regurgitation and the aforementioned sequela of this valvular pathology.

Pulmonary hypertension caused by left sided ventricular pathology can not only cause right sided congestion but also lead to interference with electrical pathways in the heart. Central pathology can interfere with the active closure of the right atrioventricular valve by disrupting its innervation from the recurrent bundle branch.[2] The specialist nature of the avian hearts conduction system means that a left ventricular lesion which includes the interventricular septum could disrupt conduction through the recurrent bundle branch, and hence affect the functioning of the right atrioventricular valve.[2] Thus, if there is an underlying left sided heart defect it can also lead to right sided heart failure by these two different mechanisms.

In this clinical case, the mechanism can only be presumed and could be a singular mechanism or a combination of several pathological processes. It is interesting to consider that the main clinical sign was ascites. And remembering that ascites is merely the endpoint of a number of reactions that the vascular system is forced to make when faced with a hemodynamic pressure imbalance across the capillary bed.[2] The combinations of environmental, nutritional and genetic events that lead to this endpoint are vast.[2] The etiology of ascites may best be viewed as a cycle of events within the cardio-respiratory circulatory systems that are interlinked.[2] Each component of the cycle may be subject to challenge, either from the environment or management, or due to functional deficit within the bird.[2] Congestive heart failure carries a poor prognosis but longevity may be prolonged by the use of symptomatic therapy including diuretics and palliative coelomocentesis.[8] There are many other common cardiac abnormalities that the

avian species are predisposed to, however, pulmonary hypertension, leading to right sided heart failure, liver congestion, and ascites is the most probable scenario for this clinical case.

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