Challenging Cardiac Cases

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The Patient with Two Diseases

As students of veterinary medicine we are taught that when we "hear the pounding of hooves we should look for horses not zebras" [1]. For the majority of our patients this is a rational approach where we wisely follow a diagnostic path and treatment plan based on logical conclusions drawn from physical examination and patient history. For patients with an abnormal cardiac auscultation, it makes sense for us to pursue a possible cardiac explanation for the patient that is tachypnoeic and coughing or syncopal. However, as practitioners we need to remain cognizant that despite an abnormal cardiac auscultation, clinical signs that may be compatible with heart disease may alternatively be attributable to concurrent non-cardiac disease. Similarly, when patients develop new clinical signs while receiving cardiac medications care should be taken not to dismiss these as side effects and discount the possibility of newly emerging disease.

The Patient with Compensated Congestive Heart Failure and Azotaemia

Azotaemia commonly develops in patients receiving chronic moderate-high-dose diuretic therapy for congestive heart failure. The origin of the azotaemia may be multifactorial and include pre-renal (secondary to diuretic use and/or low cardiac output) and renal (concurrent primary renal disease or progression of pre-renal to irreversible nephron loss due to severe and/ or longstanding hypoperfusion). Distinguishing pre-renal from renal aetiology is not possible by urine specific gravity since urine will be isosthenuric once receiving diuretic therapy. The distinction is academic for these patients and does not change management approach.

Where diuretic dose has been selected and adjusted upward appropriately in response to recurrent congestive signs, reduction of diuretic dose will result in recurrence of congestive failure. Reducing diuretic dose with the aim of alleviating azotaemia in an otherwise well patient is misguided and potentially dangerous.

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Since patients with heart failure are dependant on angiotensin II - mediated vasoconstriction of the efferent renal arteriole in order to maintain glomerular filtration rate (GFR), an angiotensin converting enzyme inhibitor (ACEi) has the potential to attenuate GFR and exacerbate azotaemia. While the long-term beneficial effects of ACEi in patients with congestive heart failure are well documented, the gains are small and for patients with significant azotaemia, the ACEi should be discontinued.

For a patient that is outwardly well (eating, drinking, interactive etc), receiving the minimum diuretic dose to maintain eupnoea and no ACEi, azotaemia need not be addressed irrespective of the degree to which BUN and creatinine elevated. Rather, the owner should be instructed to monitor appetite, as well as continued monitoring for recurrent tachypnoea. Significantly azotaemic patients are at risk for clinical signs of renal disease (anorexia, dehydration, depression, vomiting) and if owners do not seek veterinary attention promptly upon loss of appetite, continued diuretic administration will rapidly precipitate worsening signs.

[1]Dr Theodore Woodward, University of Maryland, c.1940

The Patient with Compensated Congestive Heart Failure, Azotaemia and Anorexia/ Dehydration

For some patients, particularly those receiving moderate-high-dose diuretic therapy, azotaemia will be sufficient to precipitate signs of anorexia, depression, dehydration and infrequently, vomiting.1 For these patients, rehydration is necessary to remove the offending uraemic toxins. Since the vasculature is a continuum, it is not possible to give fluids to rehydrate the kidneys and concurrently administer diuretics to keep the lungs dry. Temporary discontinuation of diuretics is essential and for some, parenteral fluid administration necessary, but there is the very real risk of precipitating congestive heart failure and recurrent pulmonary oedema in so doing.

As a general rule, diuretics need be temporarily discontinued and fluid administered while closely monitoring for signs of pulmonary oedema. If the patient is receiving other medications (pimobendan, beta-blockers, NOT ACEi), these should be continued. Fluid given intravenously via an

automated pump is best since accidental administration of excess volumes is less likely than via a gravity-fed line. Respiratory rate should be taken q1hr. Any elevation in respiratory rate above 30bpm in a non-anxious patient may be indicative of recurrent pulmonary oedema warranting pulse oximetry and thoracic radiography. If radiographs confirm development of congestive failure, immediate discontinuation of fluid therapy is indicated. Although it may seem more benign to administer fluids subcutaneously, absorption may be incomplete if peripheral perfusion is compromised by heart disease or it may precipitate recurrent pulmonary oedema if large volumes are absorbed quickly.

The end-point of fluid therapy is NOT resolution of azotaemia. Achieving this is unlikely to be possible without precipitating congestive failure, rather fluid therapy should be continued until the patient is brighter and eating. Once fluid therapy is discontinued, oral diuretics will need to be resumed. This can be delayed while the patient remains eupnoeic but at first sign of a resting respiratory rate >30bpm, diuretics should be recommenced. This may be within a few hours or the 1-2 days, but generally not longer. A lower dose of diuretic than the patient was previously receiving may be initially sufficient to maintain eupnoea but the owners must be instructed to carefully watch resting respiratory rate and increase dose when necessary.

The Patient with Decompensated Congestive Heart Failure, Azotaemia and Anorexia/ Dehydration

For some patients, fluid therapy for clinical signs associated with azotaemia may precipitate congestive failure before resolution of anorexia and dehydration. Unfortunately, the vasculature is a continuum such that it is not possible to concurrently rehydrate the kidneys with fluids and dehydrate the lungs with diuretics. Administration of fluids will increase renal perfusion, augment GFR and alleviate azotaemia but it will also increase pulmonary capillary hydrostatic pressure and facilitate transudation of fluid out of the vasculature and into the pulmonary interstitium and alveoli. Conversely, diuretics will reduce circulating fluid volume and attenuate pulmonary capillary pressures to facilitate resolution of pulmonary oedema but will also reduce renal perfusion and exacerbate azotaemia. For patients with azotaemia sufficient to cause depression and anorexia that are concurrently hypoxaemic and tachypnoeic with cardiogenic pulmonary oedema, a balance is not

possible and euthanasia is advised. For cardiac patients with congestive failure that manifests as pleural effusion or ascites without pulmonary oedema, medical diuresis can be replaced with centesis prn, and this may be sufficient to alleviate or avoide clinically-significant azotaemia.

The Patient with a "Normal" Heart but Signs Suggestive of Congestive Heart Failure

Congestive heart failure results primarily as a consequence of elevated hydrostatic pressure in the pulmonary capillaries (left sided heart failure) and/ or the systemic capillaries (right sided heart failure). Capillary hydrostatic pressure rises subsequent to elevations in venous and atrial pressure as a consequence of maximal eccentric atrial hypertrophy in the face of progressive cardiac dysfunction. In diagnosis of congestive heart failure, measurement of atrial pressures via catheterization is optimal; right heart catheterization allows direct measurement of pulmonary capillary wedge pressure (which equates to left atrial pressure) and right atrial pressure. Since this is not practical for our clinical patients, atrial size is a reasonable surrogate whereby, radiographic evidence of severe left or right atrial enlargement suggests that compensatory growth is likely exceeded and congestive failure is possible.

However, there are some notable exceptions. With acute changes in atrial load, as occurs with aortic or mitral endocarditis or mitral chordae tendinae rupture, the atrium does not have time to grow and normalize pressures such that pulmonary oedema can develop without left atrial enlargement. Cor triatriatum sinister and cor triatriatum dexter are congenital defects which obstruct venous return within the proximal atria resulting in congestive failure without atrial enlargement. The index of suspicion for heart disease may be further reduced because these defects may not produce an auscultable abnormality and, unlike other causes of right heart failure, cor triatrium dexter does not result in jugular venous distension. Pericardial effusion will produce generalized cardiomegaly and signs of right-sided congestive heart failure when it accumulates slowly, but rapid accumulation of small volume effusates can produce the same clinical consequences without appreciable cardiomegaly on thoracic radiographs. Similarly, constrictive pericarditis may produce minimal change in the cardiac silhouette on thoracic radiographs despite significant functional

consequences and severe ascites. Pericardial diseases may not produce any auscultable abnormality but jugular distension should be appreciated and dilation of the caudal vena should be evident on thoracic radiographs. All of these structural diseases can be identified via specialist echocardiography.

The Patient with an "Abnormal" Heart Without Signs Suggestive of Heart Disease

The identification of apparent heart disease in an otherwise healthy patient or a patient with clinical signs not suggestive of heart disease can be difficult to interpret.

A common example is the identification of an enlarged cardiac silhouette on thoracic radiographs of a patient with normal cardiac auscultation. Here, thoracic conformation and/ or pericardial fat are often to blame. For dogs that have narrow (on DV films) or shallow (on lateral films) chest conformation, the heart will appear enlarged. Very few dogs are both shallow- and narrow-chested such that acquisition of two thoracic views will often facilitate identification of a normal cardiac silhouette in one view of a dog with a truly normal heart. Generally, if advanced heart disease is present, the will heart appear enlarged in both views. For overweight dogs and cats, pericardial fat can result in a false assumption of cardiac disease and this must be considered when assessing radiographs of any obese patient.

Another example of falsely diagnosing heart disease can be encountered by echocardiographic examination of dehydrated cats. Here, the reduction in circulating fluid volume can result in pseudohypertrophy sufficient to meet the criteria for hypertrophic cardiomyopathy.2 As such, any echocardiographic examination should be ideally performed with the patient is known to be euvoluemic.

The Patient with Heart Disease and Risk of Polycythemia Not Congestive Heart Failure

Eisenmenger syndrome is defined as the process in which a left-to-right shunt caused by a congenital heart defect causes increased flow through the pulmonary vasculature, causing pulmonary hypertension, which in turn, causes increased pressures in the right side of the heart and reversal of

the shunt into a right-to-left shunt. In veterinary patients, Eisenmengers syndrome can be seen in a small proportion of dogs with cats with large ventricular septal defects or patent ductus arteriosus.3 Tetralogy of Fallot, defined at as a constellation of cardiac defects, which include valvular pulmonic stenosis, a non-restrictive ventricular septal defect (VSD), dextraposed aorta and right ventricular hypertrophy, most commonly results in right-to-left shunting of blood through the VSD.

These diseases with right-to-left shunting do not pose a risk of congestive heart failure. Rather, these patients are chronically hypoxaemic as a result of mixing of deoxygenated (right heart; PaO2 45mmHg) and oxygenated (left heart; PaO2 100mmHg) blood. This hypoxaemia produces cyanosis of the mucous membranes and stimulates the release of erythopoetin and secondary polycythemia. The polycythemia is a compensatory response to augment the oxygen carrying capacity of blood but when PCV exceeds 65%, blood viscosity precludes adequate perfusion of capillary beds. The result is clinical signs of cyanosis, exercise intolerance, dyspnoea and syncope.

A common pitfall is to identify dyspnoea or cyanosis in the presence of a heart disease (heart murmurs may be low-grade or absent in these patients; abnormal cardiac silhouette on radiographs is typical) assume congestive failure and instigate diuretic therapy without echocardiographic diagnosis. Unfortunately, diuretic therapy will worsen clinical signs by increasing blood viscosity. Rather, medical management primarily requires reduction in blood viscosity by medication (hydroxyurea) or repeated phlebotomy. Cyanosis with these patients is typically more impressive than cyanosis produced by even severe pulmonary oedema and the identification of cyanosis with an elevated PCV or an elevated PCV alone in any non-dehydrated patient warrants echocardiography.

References

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