Right-Sided Congestive Heart Failure in an Adult Dairy Cow

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Abstract

Priscilla, a four and a half year old Holstein cow, presented to the Large Animal Medicine Service of the Cornell University Hospital for Animals (CUHA) for evaluation of acute inappetance, weight loss, lethargy, watery diarrhea, and chronic hindlimb weakness. Three days after presentation, Priscilla developed signs of right-sided congestive heart failure including tachycardia, bilateral distended jugular veins, ventral edema, and a right-sided systolic murmur. She was also found to have tricuspid regurgitation, right atrial hypertension, bilateral pleural effusion, and hepatic congestion. Differential diagnoses for Priscilla's right-sided heart failure included endocarditis, cardiomyopathy, pericarditis, and heart failure secondary to pulmonary hypertension. No underlying cause of Priscilla's heart failure was determined. She was treated with digoxin, intravenous fluids, furosemide, and dexamethasone. Her clinical status did not improve with treatment and she was subsequently euthanized. Pathology revealed evidence of a pulmonary vasculopathy that may have caused pulmonary hypertension and right-sided heart failure.

Signalment

Priscilla, a four and a half year old Holstein cow from a 50 cow tie-stall farm, presented to the Large Animal Medicine Service at the Cornell University Hospital for Animals on September 6, 2002. Priscilla was at the beginning of her third lactation and freshened 5 days prior to presentation.

Chief Complaint

Priscilla presented to Cornell because of acute lethargy, weight loss, inappetance, and watery diarrhea that began 1 week prior to presentation.

History

Priscilla's owners reported that Priscilla had poor body condition over the past year despite not lactating for almost one and a half years. Priscilla also had a history of bilateral hindlimb weakness that began about two months prior to presentation. One day prior to presentation, Priscilla's regular veterinarian performed a right paramedian abomasopexy to correct a left displaced abomasum.

Clinical Findings

On physical examination Priscilla was depressed, dehydrated, and appeared colicky. She was tachycardic with a heart rate of 126 beats per minute. Aucultation revealed that Priscilla had poor rumen motility. Priscilla had no pings or abdominal distention.

A California Mastitis Test was performed and was moderately positive in her left hind quarter, suggesting mastitis. A hemogram and chemistry panel revealed dehydration, a neutrophilic leukocytosis with a left shift and mild toxic changes, hypokalemia and hypercalcemia. Priscilla had an elevated fibrinogen level. CUHA large animal surgeons performed a right flank exploratory laparatomy and the only significant finding was enteritis. Priscilla was started on symptomatic therapy for the weekend consisting of intravenous fluids, Banamine®, Naxcel®, and Amoximast® in her left hind quarter.

Three days later Priscilla's condition was dramatically changed. Although most of her gastrointestinal problems had resolved or were greatly improved, physical examination revealed tachycardia, bilateral jugular distention, a grade 2 out of 6 right-sided systolic murmur, and severe ventral edema of her submandibular and brisket areas despite normal total protein levels. Ultrasonography revealed severe bilateral pleural effusion, mild to moderate hepatomegaly, distended hepatic and portal veins, and gallbladder edema. Echocardiography revealed a normal sized heart with no gross abnormalities. There was minimal pericardial fluid. Doppler flow analysis of the right atrioventricular valve revealed moderate regurgitation during systole. There was no evidence of vegetative lesions or valve deformity. A catheter was placed in her left jugular vein with the end of the catheter positioned in the right atrium. The mean right atrial pressure was 25mmHg. Normal for an adult cow is from 0 to 5 mmHg.

Problem List

A list of Priscilla's problem's now included tachycardia, distended jugular veins, a right-sided systolic murmur, tricuspid regurgitation, ventral edema, right atrial hypertension, bilateral pleural effusion, and hepatic congestion. These signs all suggested that Priscilla was in right-sided congestive heart failure. Right-sided heart failure is not a common problem in adult dairy cows. It is the result of valvular regurgitation leading to volume overload, compromised ability of the heart to expand and fill with blood during diastole, or is secondary to pulmonary hypertension. The result of right-sided heart failure is increased pressure on the venous system leading to congestion.

Differential Diagnosis

Right-sided heart failure in adult dairy cattle may be due to endocardial disease, myocardial disease, pericardial disease, or secondary to pulmonary disease with hypertension.

Vegetative endocarditis is the most common endocardial disease in adult dairy cattle (1). Most often vegetative lesions on heart valves involve bacteria that have arrived hematogenously from sites of chronic infections elsewhere in the body. Possible sources of infections may include mastitis, metritis, abscesses, and thrombophlebitis (2). Vegetative lesions occur most frequently on the tricuspid valve and therefore often result in failure of the right side of the heart (1).

Diagnosis of vegetative endocarditis may be based on clinical signs that include intermittent fevers, shifting leg lameness, right-sided systolic murmurs, tachycardia, weight loss, inappetance, and signs of right-sided heart failure (3). Priscilla had all of these clinical signs. Cattle with vegetative endocarditis may also have a neutrophilic leukocytosis and elevated fibrinogen (3). Blood cultures may be useful for identifying a bacterial agent, particularly if antibiotic treatment is considered an option (1). Echocardiography may demonstrate vegetative lesions associated with heart valves. No lesions were detected during Priscilla's echocardiography. Also, the severity of pleural effusion that Priscilla had is not typically observed with endocarditis. Gross pathology will best demonstrate any vegetative lesions in the heart. Prognosis is poor for cows with vegetative endocarditis, however, if treatment is pursued, antibiotic therapy for at least four weeks will likely be necessary for successful treatment (3).

A second possible cause of Priscilla's right-sided heart failure was a primary cardiomyopathy. Cardiomyopathies may be due to genetic causes, such as the red gene-linked heritable cardiomyopathy in Holstein cows (2). Nutritional deficiencies such as inadequate Vitamin E, selenium, or copper in the diet may cause cardiomyopathies (2). Toxins, including ionophores or gossypol, may lead to myocarditis (2). Other potential causes of myocarditis include viruses, bacteria, or parasities (2). Definitive diagnosis of cardiomyopathy typically requires histopathologic examination of the myocardium (2). Infiltrative disease such as lymphosarcoma may also cause cardiomyopathy. Lymphosarcoma has a predilection for the right atrium and therefore may cause right-sided heart failure (1). Less than 1% of dairy cattle infected with Bovine Leukosis Virus

(BLV) develop lymphosarcoma (1). Priscilla was tested for BLV with an agar gel immunodiffusion (AGID) assay and she was negative. It would be rare but not impossible for a cow to have lymphosarcoma without being BLV positive. Ultrasonography, echocardiography, and radiology revealed no masses associated with Priscilla's heart or the rest of her thorax. A tube was placed in Priscilla's right thorax to drain pleural fluid for therapeutic and diagnostic purposes. A sample of the pleural fluid was collected and submitted for analysis. Pleural fluid cytology in cows with thoracic lymphosarcoma may reveal evidence of neoplasia, however, Pricilla's pleural fluid was a pure transudate that was compatible with congestive right-sided heart failure.

A third possible cause of Priscilla's right-sided heart failure was pericarditis. Pericarditis in dairy cattle may be due to hematogenous spread of bacteria. More commonly pericarditis is due to penetration of the pericardium by a foreign object extending from the reticulum. This syndrome is referred to as traumatic pericarditis or hardware disease. Pericarditis can cause congestive heart failure because elevated pressure around the heart impairs the ability of the heart, particularly the right ventricle, to fill with blood during diastole (2). Clinical signs of traumatic pericarditis may include an arched stance, reluctance to move, anorexia, fever, decreased milk production, signs of right-sided heart failure, muffled heart sounds due to pericardial effusion, and possibly a washing machine murmur associated with pericardial fibrin and effusion (3). Diagnostic tests such as the scootch or grunt test may help identify cranial abdominal discomfort. Cranioventral abdominal radiographs, when available, may be helpful for detecting hardware disease. Radiographs of Priscilla were taken and revealed no radio-opaque foreign objects

associated with the reticulum or pericardium. Priscilla also did not have muffled heart sounds and echocardiography revealed only a mimimal amount of pericardial effusion and normal pericardial thickness indicating that pericarditis was unlikely the cause of Priscilla's heart failure.

Pulmonary hypertension leading to secondary right-sided heart failure, a syndrome known as cor pulmonale, was a fourth possible cause of Priscilla's heart failure. Cor pulmonale in cattle often is a result of hypoxia induced pulmonary vasoconstriction due to causes such as high altitude disease or pneumonia (2). Priscilla had no adventitious lung sounds, dyspnea, nasal discharge, or other signs of pneumonia. Arterial blood gases were measured and were within normal ranges for an adult dairy cow. This indicated that pulmonary hypertension due to hypoxia was not the cause of her heart failure.

Prognosis

The most common causes of right-sided heart failure in dairy cattle were ruled out as possible etiologies for Priscilla's right-sided heart failure. The prognosis for dairy cattle with right-sided heart failure is poor. Therapy is typically only successful at supporting cows in heart failure to achieve short-term goals (3).

Treatment

Initial therapy included a maintanence rate of intravenous fluids for hydration while avoiding fluid overloading, dietary sodium chloride restriction to avoid fluid retention, and stall rest. Furosemide, a loop diuretic, and dexamethasone,a corticosteroid were

administered daily. After several days of this therapy, there was no improvement in Priscilla's clinical status. Because Priscilla was a successful show cow and had sentimental value to her owners, they elected to pursue further therapy despite her poor prognosis.

We decided to introduce digoxin, a cardiac glycoside, to her treatment regimen. Digoxin functions by inhibiting the Na,K ATPase exchanger in myocardial cell membranes (3). This leads to increased stores of calcium in myocardial sarcoplasmic reticulum and subsequently to increased force of contraction of the heart (3). Digoxin also slows the heart rate by increasing vagal tone through a centrally mediated mechanism (3).

Unfortunately digoxin is destroyed in the rumen, causes necrosis if given intramuscularly, and the intravenous form of digoxin is expensive (3). Therefore we developed several strategies for circumventing the rumen using the digoxin tablets. We first administered potassium chloride via a stomach tube because low potassium predisposes to digoxin toxicity and Priscilla was at increased risk for low potassium due to her furosemide therapy (3). We dissolved the digoxin in water, gave Priscilla an epidural to prevent peristalsis, and administered the digoxin rectally hoping that it would be absorbed from the rectum. Next, we dissolved the tablets in saline, filtered the suspension, and administered it intravenously. After both treatments Priscilla had no detectable digoxin in her blood.

Finally we administered the digoxin orally while attempting to avoid rumenal destruction of the drug by stimilating esophageal groove closure. In calves, the esophageal groove allows ingested milk to bypass the rumen and go directly into the abomasum. Sodium bicarbonate, which stimulates the esophageal groove to contract in adult dairy cattle, was administered followed immediately by the oral digoxin (4). Priscilla's blood digoxin level was still undetectable following this treatment

Outcome

Because Priscilla was not responding to medical therapy and her prognosis was poor, Priscilla's owners elected to donate her for teaching purposes to the Cornell University College of Veterinary Medicine. Over the next couple of weeks her tachycardia remained unresolved and she continued to lose weight despite a strong appetite. She was subsequently euthanized.

A necropsy was performed. Gross findings included severe pleural fluid, hepatomegaly, multifocal pulmonary congestion and consolidation, and mild enlargement of the right atrium and ventricle. No masses, abscesses, or thrombi were observed. Histopathology revealed evidence of a pulmonary vasculopathy. This vasculopathy included a mononuclear and neutrophilic infiltrate, dense fibrous connective tissue, and necrosis associated with small blood vessels in the lungs. This may have resulted in decreased compliance of the pulmonary vasculature with subsequent pulmonary hypertension and right-sided heart failure. The cause of this pulmonary vasculopathy is unknown.

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