

**“HONEST DOC, HE WAS  
NORMAL YESTERDAY...”**

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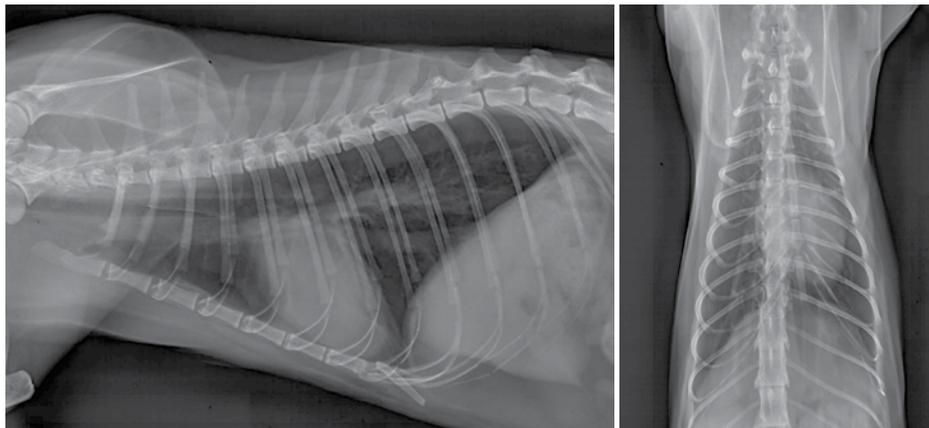


**Y**ou don't have to be in practice but a few months until you hear a cat owner make that statement over a very ill cat. As you complete your history and physical exam and gather lab tests and imaging, you are convinced that either the owner is very inattentive or a liar or the cat has an amazing ability to cover up disease until it reaches the crisis stage. We are all convinced that we have seen all three scenarios. However, is it possible that a cat can develop a very serious illness within 24 hours?

**Case Report**

Ty, a 6 year old Mn DSH was presented on Monday. His owner, a long-time cat owner with a history of being very responsive to her cats' physical conditions, said she let him outdoors the night before. He came in that morning very anxious and seemingly painful. Ty did not want to be pickup up; he cried and resisted when his owner did so to put him in his carrier. She assumed he had been injured or the victim of a cat fight or dog attack.

Figure 1



Lateral (a) and DV (b) views of the chest were not remarkable. We read them as normal.

During his physical examination we could not localize pain. His mucous membranes were pink. He had a rapid heart rate (248 bpm) with no murmur auscultated. Abdominal palpation did not reveal a large, firm, painful bladder, as we expected. However, his respiration was about 45 breaths per minute.

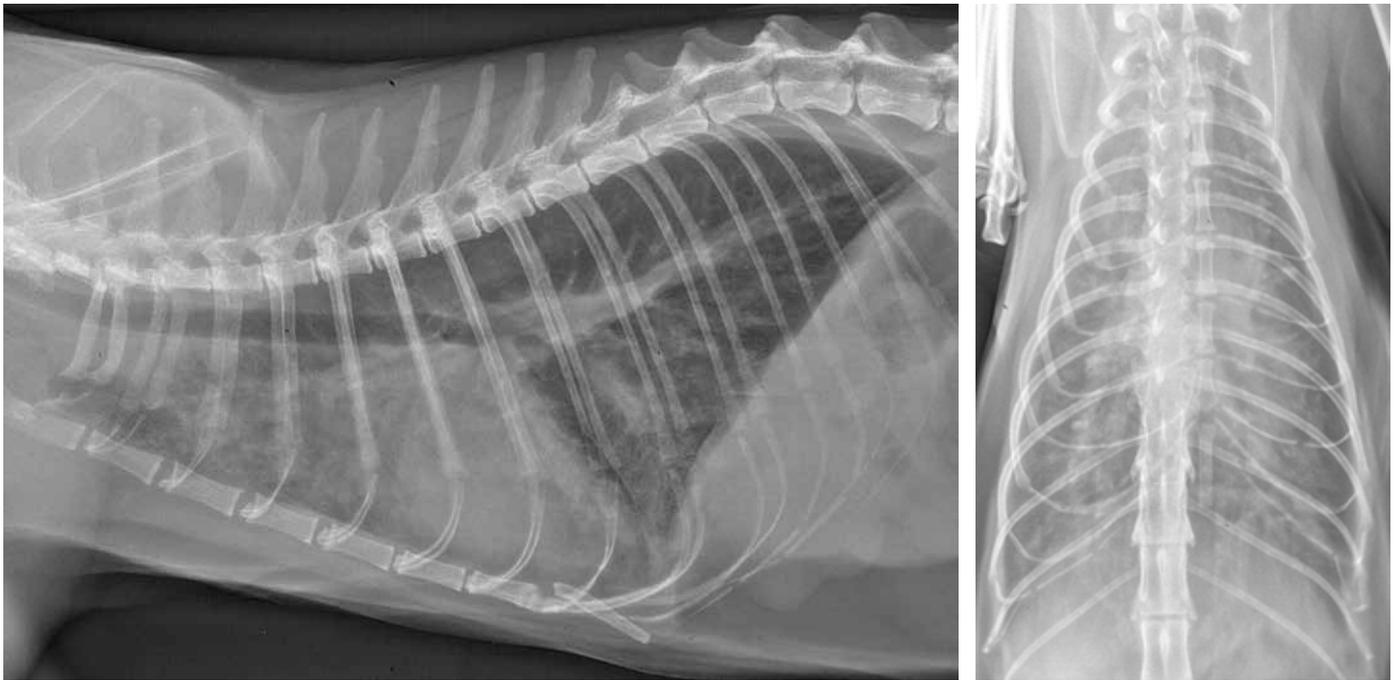
A blood panel, consisting of a CBC, chemistries, and electrolytes, was normal. We made a chest radiograph. (See Figure 1.) We could not diagnose anything of significance. The diaphragm was intact, there were no rib fractures,

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and the lung fields appeared normal. Consequently, we gave him buprenorphine for pain and an injection of cefovecin expecting to address the undiagnosed condition. He was hospitalized for the night.

The next morning Ty was not significantly improved. He did not eat during the night but we still could not localize pain. The only thing that seemed concerning was the continued respiration rate of about 40 breaths per minute. We considered making another radiograph but we hated to waste the owner’s money doing so. We kept him for the day expecting to send him home that afternoon if he was not worse. After lunch, his respiration rate increased to 50 breaths per minute. With some continued hesitation, we decided to make another chest radiograph. (See Figure 2)

Figure 2



Lateral (a) and DV (b) views of the chest showed moderate pulmonary edema.

We could not believe our eyes. The clear lung fields from the day before were now fluid filled. We kept asking ourselves, “What can make that change so quickly?” We were pretty sure he had not drowned in his water bowl or experienced electric shock in his cage. It looked like congestive heart failure, but CHF does not occur that quickly, we kept telling ourselves. So, just to prove that he could not be in CHF, we did an echocardiogram. (See Figure 3)

To our shock, his interventricular septum and left ventricular freewall were significantly thickened. His left atrium was enlarged. This cat had hypertrophic cardiomyopathy and had gone into congestive heart failure right before our eyes!

Our treatment protocol changed direction on a dime. Ty was started on benazepril, spironolactone, furosemide, and nitroglycerin paste and placed in an oxygen cage. We assured the owner that, even

though CHF causing pulmonary edema was very serious, it usually responded well to treatment. We told her he needed another 24-48 hours in the hospital.

The next morning, Ty was a new cat. His respiration rate was 28 bpm, he was much more active and alert, and he had eaten all of his food overnight. His medications were continued although he was placed in a normal hospital cage. He continued to do well throughout the day.

The next morning we decided to make another chest radiograph before discharging him. (See Figure 4) He was discharged on benazepril, spironolactone, aspirin, and furosemide. His owner was instructed not to let him go outdoors until his recheck in 4 days.

At the 4 day recheck, Ty was his old self. The owner reported normal activity and that Ty was getting more difficult to keep indoors. As we examined him, he tried

to bite us. That was the sign that the old Ty was back. We reduced the dose of his furosemide. We told the owner to taper it over the next week and resume it only if his respiration rate increased.

We rechecked Ty 1 month later. His chest radiograph remained normal as did his activity. His owner was very pleased to have her old Ty back.

### Discussion

We still marvel at Ty's radiographs on Day 1 vs. Day 2. The onset of CHF and pulmonary edema occurred within 24 hours, and we had documented that, indeed, some cats can develop a severe, life-threatening state literally overnight. Obviously, the cardiac changes caused by hypertrophic cardiomyopathy did not occur that rapidly, but the onset of clinical signs was remarkable. HCM is more likely to present like this than most diseases because the disease is often present in an asymptomatic form for months to even years. It is truly a perfect storm for an acute onset.

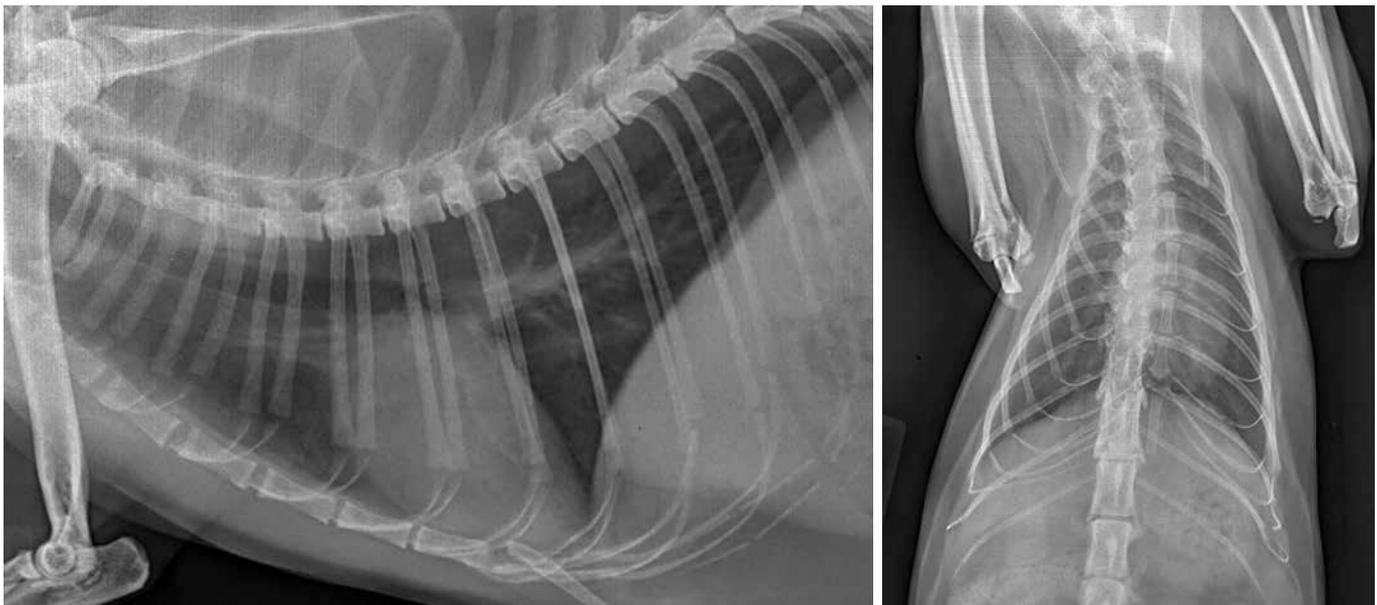
Morals of the Story: 1) Not all owners are liars or inattentive. 2) Sometimes cats go from “normal” to “crisis” in just a few hours. 3) Cats are definitely not small dogs. One major difference is coughing and heart failure. Only rarely will cats have a “cardiac cough.” 4) Mild to moderate hypertrophic cardiomyopathy does not change shape of the cardiac silhouette, as seen on radiographs, so its diagnosis is often missed when thoracic radiographs are employed. An echocardiogram is the ultimate diagnostic test. 5) Resist the urge to “avoid wasting the client's money” by failing to repeat a test, even within 24 hours.

Figure 3



Echocardiogram showing thickening of the interventricular septum (IVS) and left ventricular free wall (LVFW) (0.84 and 0.82; N = 0.4-0.6 cm). Fractional shortening was 66% (N = 35-55%). Note the large papillary muscle (arrow). The other view showed mild enlargement of the left atrium (LA:Ao = 2.0; N < 1.6).

Figure 4



Lateral (a) and DV (b) views of the chest returned to normal after 48 hours of treatment for congestive heart failure.