A Case of Pulmonary Edema in a Horse Treated for Cardiac Ailments

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A rare case of pulmonary edema due to cardiac valve obstruction has been encountered in a racehorse. It is suspected that the pulmonary edema was caused by high blood pressure in the lungs due to mitral insufficiency resulting from bacterial endocarditis. If pansystolic heart murmur and rough bronchial sounds are detected during auscultation of a horse exhibiting flu-like symptoms, pulmonary edema and mitral insufficiency should be suspected.

Key words: arrhythmia, bacterial endocarditis, pulmonary edema, mitral insufficiency, echocardiography

Equine cardiac patients are known to exhibit arrhythmia, such as atrial fibrillation and atrioventricular (A-V) block, but the occurrence of these and other cases is extremely rare. Among horses affiliated with the Japan Racing Association, the incidence of such cardiac problems is only about 0.2% [2].

Arrhythmia in race horses, such as the atrial fibrillation that occurs while running is a serious hindrance to the running of the animal, but is usually transitory, and a synchronous rhythm can easily be recovered through defibrillation. Furthermore, most of the arrhythmia detected at rest by auscultation is a Class II A-V block. [1]; and electrocardiograph examinations while exercising have shown that there is absolutely no hindrance to the racing capability [1].

For a race horse, it is believed that any occurrence of a disorder from organic changes in the heart will have a serious influence on racing capability, but there are very few opportunities to actually observe this clinically. Pulmonary edema is known to arise in connection with pneumonia and general congestion due to cardiac valve obstruction [5], but we have not found any reports of pulmonary edema in race horses in Japan.

We have investigated a case of pulmonary edema from cardiac valve insufficiency, and present here several of our observations and findings.

Subject Animal

The subject was a female thoroughbred that entered the stables at the Training Center in the fall of her second year, but was left to pasture initially, reentering to prepare for her first race in the summer as a 3 year old. A heart murmur had been confirmed since the mare was 2 years old, but there were no particular clinical symptoms and the training proceeded as scheduled.

A fever of 38.7°C broke out 10 days after the first race. Since there was swelling of the mammary glands, treatment with antibiotics was made for contagious mastitis (Table 1).

Eleven days after the onset of fever, she began to cough and respiration became rapid. Auscultation of the chest revealed the sounds of labored bronchial respiration at the anterior of the chest on both sides. This led to the suspicion of pneumonia, and a
bronchoscopy was performed. The interpretation of these examination results is discussed later.

Treatment was continued according to the results from the bronchoscopy and a bronchoalveolar lavage, but there was no improvement of the symptoms. Approximately 1 month after the onset of symptoms, the heart murmur became louder than it had been previously, axillary and thoracic edema appeared, and jugular pulsation was observed.

**Table 1. Treatment**

<table>
<thead>
<tr>
<th>Days after onset of fever</th>
<th>Drug and procedure</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–10</td>
<td>Cephalothin sodium</td>
<td>10 g i.v. B.I.D.</td>
</tr>
<tr>
<td>11–16</td>
<td>Cephalothin sodium, Gentamicin sulfate, Flunixin meglumine</td>
<td>10 g i.v. T.I.D., 900 mg i.v. B.I.D., 500 mg/day</td>
</tr>
<tr>
<td>[13]</td>
<td>Bronchoalveolar lavage (BAL)</td>
<td></td>
</tr>
<tr>
<td>17–36</td>
<td>Minocycline hydrochloride, Flunixin meglumine</td>
<td>1000 mg i.v. B.I.D., 500 mg/day</td>
</tr>
<tr>
<td>[18, 24]</td>
<td>BAL</td>
<td></td>
</tr>
</tbody>
</table>

Administration of antibiotics and non-steroidal antiphlogistics and implementation of bronchovesicular wash.

**Fig. 1.** Bronchoscopy. Damage to the bronchial mucous membrane.

**Fig. 2.** Microscope exam of the bronchovesicular wash fluid. Simple quick stain with hema-color, 400x.
neutrophile in the bronchoalveolar lavage fluid, severe pulmonary edema was suspected.

**ECG and Phonocardiography Results**

The electrocardiogram (AB leads) performed on the 13th day after the onset of symptoms showed a slightly elevated heartbeat (60 beats/min) and normal waveforms. On the 24th day, an A-V block (Class II, Type I) appeared, and the P-wave became bisferient. On the 33rd day, atrial fibrillation was confirmed (Fig. 3).

A phonocardiography was performed on the 64th day. Auscultation revealed conspicuous arrhythmia and a murmur throughout the entire systolic phase. The heart murmur could be immediately identified on the Levine scale as a Level III-IV by placing a stethoscope to the thoracic wall when the area of the mitral valve was at its maximum. The systolic phase murmur for the entire phase between S I and S II was recorded on the phonocardiogram (Fig. 4), leading to a diagnosis of mitral insufficiency.

At the same time, a catheter was inserted intravenously as far as the heart. It was confirmed that the f-wave about 300/sec from inside the vein-apex lead.

**Echocardiography Results**

The condition of the mitral valve was observed by ultrasound using a LOGIQ500MD (GE Medical Systems, 2.5 MHz sector probe) on the left chest wall. Hyperplasia of the valve flaps was noticeable, and mitral insufficiency was confirmed (Fig. 5) and regurgitation to the left atrium during systole.

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**Table 2. Bronchovesicular wash fluid microbiological test results**

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>CFU/mL</th>
<th>Drug sensitivity test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Cephalothin sodium</td>
</tr>
<tr>
<td><em>Streptococcus zooepidemicus</em></td>
<td>$3 \times 10^5$</td>
<td>++</td>
</tr>
<tr>
<td><em>Pseudomonas aeruginosa</em></td>
<td>$2 \times 10^4$</td>
<td>–</td>
</tr>
<tr>
<td><em>Corynebacterium sp.</em></td>
<td>$2 \times 10^4$</td>
<td>++</td>
</tr>
<tr>
<td><em>Alcaligenes xylosoxidans</em> subs. <em>Xylosoxidans</em></td>
<td>$5 \times 10^2$</td>
<td></td>
</tr>
<tr>
<td><em>Escherichia coli</em></td>
<td>$5 \times 10^3$</td>
<td></td>
</tr>
</tbody>
</table>

**Fig. 3. ECG interpretation. Atrial fibrillation.**

**Fig. 4. Phonocardiogram showing mitral insufficiency.**

**Fig. 5. Echocardiogram showing mitral regurgitation.**
Pathological Anatomy Interpretation

Based on the clinical symptoms and the results of the clinical tests, it was clear that it would be extremely difficult to restore the horse to a race-worthy condition, so the animal was reassigned for use as a research animal. After further observation for some time, since there was no change in the symptoms in spite of having continued treatment, the animal was euthanized.

Light and moderate hypertrophy of the endocardium of both the left atrium and left ventricle, as well as clear roughening of part of the left ventricle were seen. There was also hypertrophy of the mitral valve, and an increased volume of fluid in the pericardial cavity (Fig. 6). Histologically, the fibrous hyperplasia and edema accompanying the infiltration of the inflammatory cells to the mitral valve were confirmed (Fig. 7).

A large volume of serum entered into the intraalveolar and stroma, causing swelling in the lungs, also know as edema (Fig. 8). It was confirmed

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**Fig. 4.** Phonocardiogram interpretation. Systolic murmur throughout the period between S I and S II.

**Fig. 5.** Echocardiography images. Mitral valve insufficiency.

**Fig. 6.** Macroscopic findings. Mitral valve edema, fibrous hyperplasia.
histologically that there was interlobular fluid retention in the lungs (Fig. 9).

**Discussion**

The proper functioning of the mitral valve depends on the components that make up the valve (septum and free wall of the flap, tendinous cord, mastoid muscle, and the left atrium). If one or more of these components is damaged, this causes a functional insufficiency of the valve [4]. In this case, it appears that mitral insufficiency was the result of hypertrophy of the mitral valve flap. The hypertrophy of the mitral valve is *a posteriori*, and one of the suspected causes of the disorder is bacterial endocarditis.

For this horse, mitral regurgitation occurred as a result of the mitral insufficiency, and the regurgitation caused an increase in the blood flow volume in the left atrium. This is suspected to have lead to an increase in pressure in the left atrium as well as pulmonary vein, shifting from the expansion of the left atrium to the high blood pressure in the lungs. It is believed that the high pulmonary blood pressure irritated the peripheral bronchial tubes, causing frequent coughing, and that the pulmonary edema occurred as the disease progressed.

The clinical symptoms of congestive heart failure resulting from mitral insufficiency include a variety from respiratory organ symptoms to vein enlargement and peripheral edema [4]. These kinds of clinical symptoms are connected to the speed of progress of the mitral regurgitation and the compensatory biological structural changes [4]. In acute mitral insufficiency, for example when a rupture of the tendinous cord occurs, changes in capacity and compliance of the left atrium caused by the sudden increase in left atrium pressure and pulmonary vein pressure occur in a short period of time, and acute left cardiac insufficiency arises [4].

In this case, in addition to the clinical symptoms of left cardiac insufficiency, which are coughing and pulmonary edema, there were also strong symptoms of right cardiac insufficiency, fluid retention, and jugular pulsation, leading us to believe that the mitral insufficiency progressed slowly. The treatment for this was limited to palliative treatments, such as the administration of ACE inhibiting medications and diuretics, since there is no effective cure [4].

In this case, although the existence of the murmur was known prior to the appearance of symptoms, without poor performance or any other clinical
symptoms, it was difficult to judge the murmur as prodromal symptom. This made it difficult to connect cardiac valve function abnormalities and clinical symptoms, and is believed to have delayed the final diagnosis. One of the authors had previously encountered a race horse that had contracted bacterial endocarditis, and had experience with the diagnosis of WPW syndrome from ECG and the diagnosis of mitral insufficiency through echocardiography [3]. As in this case, flu-like symptoms appeared several days after running in a race, and auscultation of the heart revealed an accelerated heart beat and a noticeable murmur starting from the middle of S I and fragmentation of S II.

When a racehorse is examined because of a fever that occurs after undergoing a severe stress, like running a race, it is necessary to make a habit of performing a cardiac auscultation. If there is notable coughing and you can hear pansystolic heart murmur and rough bronchial alveolar sounds, pulmonary edema and mitral insufficiency should be suspected, and echocardiography should be performed to make the diagnosis.

References