1973

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Russ R. Weston
Iowa State University

Bruce L. Hull
Iowa State University

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Bovine Toxic Mastitis

Summary

Bovine toxic mastitis is an acute clinical syndrome manifested by acute inflammation of the mammary gland and severe generalized toxemia. It must be differentiated from such diseases as parturient paresis, acute peritonitis, torsion of the abomasum or cecum, and toxic metritis. Clinical pathology and bacteriology are helpful in making a definitive diagnosis, though the information obtained must often be used in retrospect because treatment with antibiotics, antihistamines, steroids, and fluids must be started immediately. The prognosis is usually poor although some success has been obtained with immediate treatment. This syndrome is becoming increasingly important in the dairy industry today.

Discussion

Bovine toxic mastitis is an acute clinical syndrome manifested by acute inflammation of the mammary gland and severe generalized toxemia. This syndrome is mentioned infrequently in the literature. The authors are using mainly clinical observations and records as a basis for this paper, which includes a general description and discussion of toxic mastitis, with specific references to one case that showed most of the signs expected in the syndrome. The case, a 5½-year-old Holstein cow, was first examined on the farm on September 10, 1972, by a clinician from the Ambulatory Clinic of Iowa State University. She was admitted to the clinic on September 11, 1972.

* Mr. Weston is a fourth year student in the College of Veterinary Medicine, Iowa State University.
† Dr. Hull is an Assistant Professor in Veterinary Clinical Sciences at Iowa State University.

History and Physical Exam

Toxic mastitis usually occurs from one week prepartum to two weeks post partum. The typical history received from the owner is depression with incoordination and trembling. Diarrhea caused by the toxemia is also commonly seen clinically. The temperature is usually 104.0°F or higher, however, if the toxemia is severe the temperature may be normal to subnormal. An increased heart rate and weak heart beat are common. Lung auscultation reveals rapid, shallow respiration and lung congestion. The rumen has no activity and may have gas accumulation. Also, a gas-filled organ can often be percussed in the right paralumbar fossa. The appearance of the eyes indicate depression and have injected sclera because of the vascular congestion. The uterus can be rectally palpated in various stages of involution. There may be evidence of gas-filled intestines. The internal iliac lymph nodes may be swollen. Palpation of the mammary gland often indicates no abnormality but discolored, watery milk may be obtained. The use of a cowside mastitis test, such as the California Mastitis Test, is recommended in case the changes in the milk are not visible to the naked eye. The Holstein showed many of these signs. She calved in the early morning hours of September 10, 1972. On initial examination, about six hours post partum, she exhibited trembling and then progressed to ataxia later in the day. Depression was evident, but diarrhea was not noticed until two days post partum. However, on initial examination on the farm no mastitis was observed. The temperature, 102.0°F, was not as high as expected with a typical tox-
ic mastitis. When the animal was admitted to the clinic, the classical signs, as described previously, became evident with severe watery mastitis in the left rear quarter and a temperature of 104.0°F.

**Differential Diagnosis**

Toxic mastitis may be very difficult to differentiate from other diseases which occur near the time of parturition. Care must be taken to make sure all organ systems of the animal are evaluated and the entire situation accessed. Probably the most important syndrome that must be ruled out is parturient paresis. Since the cow is often ataxic or down, the dairyman may call you on the assumption that it is actually parturient paresis. With parturient paresis you see ataxia or recumbency, however, you would expect to see a lowered body temperature and some body parts, such as the ears, may feel cool to the touch. In parturient paresis, you often have a classical picture of the cow's head turned, facing the rear. This is, of course, not high on the criteria list for a differentiation. With parturient paresis, there is no evidence of mastitis. Another means of differentiation, though only as a last resort, could be the response of the animal to calcium therapy. In the case under review, the differentiation between these two syndromes was very difficult, initially because the temperature was only 102.2°F, and no mastitis was observed. However, it did not appear to be a typical milk fever either. Calcium borogluconate was administered intravenously, with some response, but not what would be expected when milk fever is the only problem.

Acute peritonitis is another disease which must be differentiated. This could be the result of traumatic reticulitis or uterine laceration. With peritonitis, the temperature will rise, as in toxic mastitis. The animal will be off feed, with increased respiration and heart rate. Rather than diarrhea, however, constipation is more common with peritonitis, and trembling and progressive ataxia are not common. With traumatic reticulitis, you often can elicit pain from xiphoid sternal palpation.

The presence of a gas-filled organ on the right side of this clinical case requires you to differentiate toxic mastitis from abomasal or cecal torsion. Cecal torsion would be characterized by a normal temperature and lack of feces. Rectally, a large "sausage" shaped mass can be palpated on the right side, possibly extending into the pelvic cavity. Abomasal torsion usually occurs a few weeks after parturition and may be either acute or subacute. With acute abomasal torsion, as in toxic mastitis, circulatory shock is evident with increased heart rate and acute depression. However, the feces are scant, instead of loose, because of the intestinal obstruction. Rectally, the abomasum should be palpated below and to the right of the kidney. It is tense and does not "pit" on pressure.

Toxic metritis is also part of the differential diagnosis. It usually resembles toxic mastitis. However, the absence of mastitis and the presence of a very foul odor and a necrotic discharge from the vulva are helpful.

Clinical pathology is an important criterion in helping reach a definitive diagnosis. Unfortunately, however, the information is often not available until later and can be used only in checking the diagnosis made on the farm. The white blood cell count is an especially helpful parameter to evaluate, along with the differential count. Within a few hours after the onset of toxic mastitis, there is a severe leukopenia, usually below the minimum normal of 4,000 cells/cmm. and commonly around 2,000 cells/cmm. Both the neutrophils and lymphocytes are depressed, but the massive invasion of neutrophils into the tissues makes the lymphocyte count appear higher on differential. This leukopenia usually lasts a few hours, after which there is a regenerative left shift associated with a rising leukocyte count. The left shift is evidenced by the increase in band neutrophils and even some metamyelocytes. Below is a summary of white blood cell counts of a typical case of toxic mastitis.

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Day 1 Less than 2,000 cells/cm.
Day 2 Approximately 4,000 cells/cm.
Day 3 10,000–14,000 cells/cm.
Day 4 10,000–14,000 cells/cm.

The differential leucocyte count will usually show:

Day 1 Neutropenia, apparent lymphocytosis
Day 2 Left shift in neutrophils
Day 3 Neutrophilia with left shift
Day 4 Neutrophilia with left shift

In the case in review, the blood picture fits the classical syndrome very closely. The table below summarized the results:

<table>
<thead>
<tr>
<th>WBC</th>
<th>Differential</th>
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<tbody>
<tr>
<td>Day 1 1,875 cells/cmm.</td>
<td>Seg 10, Bands 8, Lympho. 75</td>
</tr>
<tr>
<td>Day 2 4,944</td>
<td>Seg 2, Bands 75, with meta-myelocytes, Lympho. 23</td>
</tr>
<tr>
<td>Day 3 13,200</td>
<td>Seg 3, Bands 74, with meta-myelocytes, Lympho. 23</td>
</tr>
<tr>
<td>Day 4 13,700</td>
<td>Seg 3, Bands 52, with meta-myelocytes</td>
</tr>
</tbody>
</table>

The white blood cell count is helpful in differentiating parturient paresis, abomasal or cecal torsion, acute peritonitis and toxic mastitis. Parturient paresis and abomasal or cecal torsion will show little change in the white blood cell count. Acute peritonitis usually shows a slight leukopenia initially, which may still be in the normal range, while toxic mastitis has a very definite leukopenia as explained earlier. However, a peracute, generalized peritonitis may also have a pronounced leukopenia.

Blood calcium is another parameter which is valuable in making a definitive diagnosis, but as mentioned previously, there is no cowside method for calcium determination. This helps differentiate parturient paresis from toxic mastitis. Normally blood calcium levels are approximately 10 mg%. In parturient paresis this can drop to 3–4 mg%. In our review case, the blood calcium was recorded at 5 mg%. Therefore, it is possible that this cow had a slight case of parturient paresis along with toxic mastitis.

Bacteriology is very important in determining the primary etiological agent and subsequently determining its sensitivity to various antibiotics. The most common bacteria causing toxemia are the gram (-) rods that release an endotoxin. These usually are *Escherichia coli*, *Aerobacter aerogenes*, *Pseudomonas aeruginosa*, and *Klebsiella spp.*

In our review case, *Staphylococcus aureus* was isolated and found sensitive to erythromycin, neomycin, streptomycin, tetracycline, and furacin. It was penicillin resistant.

**Treatment**

Toxic mastitis is a rapidly progressing disease and, therefore, treatment must be started immediately in order to have success. Since this is true, therapy must be started with an antibiotic which has been effective in similar cases. The antibiotic sensitivity tests then either confirm its efficacy or suggest another antibiotic which may be effective. Antihistamines are indicated to provide some symptomatic relief of the toxemia resulting from the absorption of the end products of protein destruction, which includes histamines. Antihistamines must be administered frequently, as often as once every four hours, to obtain satisfactory results. Steroids are also indicated to counteract endotoxic shock which is involved in toxic mastitis. They also give the animal a feeding of euphoria which may be valuable when stimulating the animal to rise after going down. Fluids are essential, as in any case of shock, to bring the electrolyte imbalance back to normal. Stripping out all quarters is also very important in

*Issue No. 1, 1973*
treatment to help remove some of the toxins in the mammary gland. This should be done as often as possible. Therapy as described above was used in the review case, however, it was not started soon enough to be effective. The prognosis for toxic mastitis is not good. If treatment is initiated early in the course of the disease, some success may be obtained. However, it usually leads to either recovery after prolonged therapy or to the "downer cow syndrome" terminating in death. In the specific case of *Staphylococcus aureus* toxemia, the toxic mastitis can lead very quickly to gangrenous mastitis which usually requires amputation of the teat or udder to save the animal's life for salvage slaughter. The review case developed areas of gangrene in the udder because of the *Staphylococcus aureus* infection. Despite rigorous therapy this cow progressed into a "downer cow syndrome" and died.

As treatment for toxic mastitis is always costly and often futile, prevention or early detection and treatment are essential. Early detection should be based on a complete physical examination rather than jumping to conclusions on the basis of suggestive signs. Prevention of toxic mastitis is similar to that for all other types of mastitis and should include good management and a dry-cow therapy program.

**BIBLIOGRAPHY**


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**Equine Rabies**

*Ronald C. Emerson*


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**Summary**

Rabies in the horse can be obscured by atypical clinical signs. One such case is discussed which resulted in exposure to four people. The clinical signs of equine rabies are reviewed, and a differential diagnosis is discussed. The policy of including rabies in your differential when a CNS problem is observed is again emphasized.

**Introduction**

Veterinary students are confronted with the disease known as rabies (lyssa, hydrophobia) from the first day of their freshman year. The disease is studied from the pathological, clinical, and public health point of view and yet this disease is often forgotten to be entered into the differential diagnosis.

**History**

On November 28, 1972, a 5 year old male quarter horse was admitted to Stange Memorial Clinic with the owner's complaint that the horse had been colicy for 2 days. The owner last noticed his horse eating on November 24 and 25, and drinking little water. The owner also noticed that his horse was slightly ataxic when forced to move on November 27 and his general attitude was lethargic since November 24. He noticed that the mucous membranes of the mouth were ulcerated. The feces were described as being of normal consistency.