posed the Angora goat to the development of rhodococcal pneumonia, given the infrequency of this disease in the goat. It is unknown whether this goat became infected before or after its purchase, although the advanced state of the abscesses suggests the infection was acquired prior to arrival at its final destination. This disease may be more frequent than is presently recognized in ruminants. The gross lesions were thought to be compatible with C. ovis infection at the time of submission, and the diagnosis of *R. equi* would have been missed if bacterial isolation had not been attempted. Routine aerobic bacterial isolation tests should be performed on all ruminants submitted for diagnostic evaluation that exhibit pneumonia and abscessation of tissues to establish the true incidence of this disease.

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**Sources and manufacturers**

a. Difco, Detroit, MI.

b. Cleveland Scientific, Bath, OH.

c. BBL, Becton Dickinson, Cockeysville, MD.

d. GIBCO Laboratories, Lawrence, MA.

e. Sigma Chemical Co., St. Louis, MO.

f. Forma Scientific, Marietta, OH.

g. BioMerieux Vitek, Hazelwood, MO.

**References**


Cystic adenomatous hyperplasia of the equine allantois: a report of eight cases


With the exception of a number of infectious diseases causing placental lesions, very little is known about the noninfectious placental lesions and their importance in animals. There are only a few reports of tumors and anomalies of the placenta of domestic mammals. In equine species, various placental lesions have been reported: dropsy of the fetal sacs, increased length of the umbilical cord predisposing fetal strangulation, excessive torsion and allantochorionic necrosis at the cervical pole, twin placentation, premature placental separation, choriocchoric villar hypoplasia, adventitial placentation, invagination of the allantochorion in twin pregnancy, and pathologic calcification of the allantochorion. Other abnormalities reported only rarely have been multiple cysts on the allantoic surface and hyperplasia or dysplasia or tumors originating from the allantoic epithelium. There are no such reports in the human literature. Here we describe the gross and microscopic pathologic changes in 8 cases of adenomatous hyperplasia of the equine allantois.

Fresh placenta from 5 mares and portions of placenta fixed in neutral buffered 10% formalin from 3 mares were submitted for laboratory evaluation. In addition, 1 foal and fresh tissues from 2 foals were also submitted. Fresh placenta and tissues from the foals were cultured for aerobic bacteria. Placentas were also cultured for fungi. Portions of the placentas and tissues from the foals were fixed in neutral buffered 10% formalin, trimmed, embedded in paraffin, sectioned at 4-7 μm, and stained with hematoxylin and eosin. Special stains such as Gram’s and periodic acid-Schiff were also performed in a few cases.

Breed, age, reproductive history, and state or country of origin of each mare are summarized in Table 1. Two originated from Illinois, 2 from California, 1 each from Pennsylvania and Connecticut, and 1 each from Brazil and New Zealand.

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Lesions were confined to the placentas. There was variation in size, location, and distribution of lesions. The placentas had areas of thickness ranging from 10 to 30 cm in diameter. The thickening was due to the presence of many raised round to oval firm nodules ranging from 0.5 x 0.5 cm to 2 x 2 cm in diameter (Fig. 1). In one placenta, there was a large, pale, firm mass 4 x 2 x 2 cm arising from the allantoic epithelium (Fig. 2). On cut surface, this mass and others had numerous cysts filled with serous to mucoid exudate, and some contained white flecks. Microscopically, the nodular masses were composed of numerous irregularly formed acinar-cystic structures lying adjacent to a basal zone of fibrous connective tissue (Fig. 3). These acini were lined by a single layer of cuboidal epithelial cells, which often formed intraluminal papillae (Fig. 4). Some of the acini were empty, but many contained eosinophilic proteinaceous material, sloughed epithelial cells, and scattered neutrophils.

There were large areas in the chorions that lacked villi. These areas were generally on the chorionic surface fused to the nodular areas of the allantois. The surface of the affected chorion was rough and granular in appearance and covered with a fibrinous material. Histologically, there was chronic active inflammation of the chorionic epithelium, with loss of chorionic villi. In other sites of the placenta, necrosis of the chorionic villi was extensive and accompanied by infiltration of neutrophils, lymphocytes, few plasma cells, and macrophages mixed with fibrin and necrotic debris. There was no significant inflammation on the allantoic side.

One of the foals that was submitted along with the placenta was determined to have died with cardiac tamponade. Gross and histologic examination of tissues from 3 other foals (kidney, liver, lung, and spleen) did not reveal any lesions of diagnostic significance. *Escherichia coli* was cultured from 3 placentas. No fungi were isolated from 3 cases in which isolation was attempted.

Tumors or anomalies of the placenta have been reported only rarely. Chorionepithelioma-like tumor has been described in the dog, cat, and ox, and cystic or hydatid mole or tumor-like growths in the placenta also have been described. There are 2 reports that included 3 cases of adenomatous hyperplasia of the allantois in horses. More recently, a report described a large number of similar cases

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Breed</th>
<th>Age (yr)</th>
<th>Reproductive history</th>
<th>Geographic source</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Thoroughbred</td>
<td>9</td>
<td>Abortion at 8 months of gestation; 3 normal foals before</td>
<td>Illinois</td>
</tr>
<tr>
<td>2</td>
<td>Quarterhorse</td>
<td>13</td>
<td>Delivered weak immature live foal, which died 1 day later; 7 normal foals before</td>
<td>Illinois</td>
</tr>
<tr>
<td>3</td>
<td>Appaloosa</td>
<td>5-6</td>
<td>Abortion at 10 months of gestation; 1 normal foal before and 1 normal foal since</td>
<td>Connecticut</td>
</tr>
<tr>
<td>4</td>
<td>Thoroughbred</td>
<td>Not available</td>
<td>Abortion at 7 months of gestation; other history unknown</td>
<td>Pennsylvania</td>
</tr>
<tr>
<td>5</td>
<td>Thoroughbred</td>
<td>8</td>
<td>Delivered 40-day live premature foal, which died 8 days later; 1 normal foal since</td>
<td>Brazil</td>
</tr>
<tr>
<td>6</td>
<td>Thoroughbred</td>
<td>12</td>
<td>Abortion at 9 months of gestation</td>
<td>New Zealand</td>
</tr>
<tr>
<td>7</td>
<td>Quarterhorse</td>
<td>25</td>
<td>Delivered normal foal; 20 normal foals before</td>
<td>California</td>
</tr>
<tr>
<td>8</td>
<td>Quarterhorse</td>
<td>8</td>
<td>Abortion at 10 months of gestation; 1 normal foal since</td>
<td>California</td>
</tr>
</tbody>
</table>

Figure 1. Severely thickened placenta from a mare. Multiple raised nodules of various sizes are distributed on the allantois.
associated with chronic or chronic active placentitis. A variety of bacteria and Aspergillus spp. were cultured. Most of these cases were from late gestation. The membranes had multiple raised gray-white to gray-red round to oval nodules of various sizes that bulged into the allantoic cavity. Microscopically, these nodules were similar to those described here.

The origin of the placental nodules appears to be the allantoic epithelium because they are localized on allantoic surface and the hyperplastic epithelium bears close resemblance to the allantoic epithelium. The cause or the significance of the adenomatous hyperplasia is difficult to determine. The severe placentitis may have acted as an irritant or stimulus resulting in hyperplasia. All mares had severe bacterial placentitis; E. coli was cultured from 3. One of the recent reports was associated with a variety of bacteria, including Nocardia spp., E. coli, Leptospira spp., Pseudomonas...
Figure 4. Tubular acinar structures in the placenta of a mare are lined by cuboidal epithelium with papillary projections. The lumen contains proteinaceous material, necrotic debris, and inflammatory cells.

spp., and others. Aspergillus was identified in 5 cases. Special stains and culture failed to reveal mycotic agents in the placentas in the present report.

Hormones might affect the development of allantois hyperplasia. These hormones in turn might also promote cervical relaxation, predisposing the mares to placentitis.

Adenomatous hyperplasia in these 8 mares probably was not the primary cause of in utero or neonatal death. Bacterial placentitis is a more likely cause. Most of these mares had had normal foals without complication prior to this incident, and at least 3 have subsequently had 1 healthy foal. Therefore, although the cause of adenomatous hyperplasia of the placenta cannot be determined, it is probably of little significance in the subsequent reproductive life of these mares.

The wide national and international geographic distribution of cases of placentitis in this report suggests that allantoic hyperplasia is ubiquitous in equines. It is also probably more common than reported in the literature (1 laboratory has seen more than 60 cases in a few years) because small cystic structures are often missed during examination of the placenta.

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References