Epidemiological features of Morel’s disease in goats

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Abstract

Morel’s disease caused by Staphylococcus aureus subsp. anaerobius was diagnosed for the first time in Poland in October 2006 in a goat flock. A second infected flock was found two months later. The course of the disease in both flocks was observed for 15 – 17 months. Clinical manifestation was confined to abscesses located near major superficial lymph nodes, mostly: superficial cervical, subiliac, parotid and mandibular. At necropsy no other lesions were found. The incubation period was estimated at 3 weeks. Clinical signs were seen both in young and adult goats and up to 7 abscesses in one animal were noted. Abscesses tended to persist for 1 to 5 months, then rupture and heal completely. The initial high in-flock point prevalence in both flocks (93.6% and 84.4%) dropped to approximately 10 – 30% during next 3 – 4 months. Until the end of the observation period the in-flock point prevalence remained at this level and only single abscesses were observed, mainly in young animals. No influence of the concurrent caprine arthritis encephalitis virus (CAEV) infection on the clinical course of Morel’s disease was noticed.

It is to be concluded that the clinical course of Morel’s disease in a goat flock resembles caseous lymphadenitis (CLA). However, in Morel’s disease abscesses occur more frequently in young goats and are located near, not inside, the lymph nodes, as in the case with CLA. Also, the incubation period of Morel’s disease seems to be shorter (3 weeks versus 2 – 6 months in CLA).

Key words: Morel’s disease, Staphylococcus aureus subsp. anaerobius, abscess, epidemiology, goat

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Introduction

Morel's disease is caused by *Staphylococcus aureus* subsp. *anaerobius*. The bacterium was first isolated in 1920 (Aynaud 1922) and found to affect small ruminants (Bajmoc et al. 1984, Fuente et al. 1985, Alhendi et al. 1993, Fuente et al. 1997, Sanz et al. 2000), with one case described in a dog (Oliveira et al., 2006) and a few in humans (Crawford at al. 1994, Over et al. 2000, Friedberg et al. 2003, Peake et al. 2006).

The disease occurs mostly in Africa and Asia. So far it has been reported in Kenya (Shirlaw et al. 1962), Sudan (El Sanousi et al. 1989), Tunis (Ben Said et al. 2002), Saudi Arabia (Alhendi et al. 1993) and Somalia (Pegram 1973). Only a few outbreaks have been reported in Europe: Hungary (Bajmocy et al. 1984), Spain (Quiteria et al. 1996), Denmark (Möller et al. 2000), Croatia (Habrun et al. 2004) and Poland (Kaba et al. 2007).

The clinical symptoms are similar to those observed in caseous lymphadenitis (CLA) caused by *Corynebacterium pseudotuberculosis*. Morel's disease leads to formation of abscesses located mainly in close association with superficial lymph nodes mostly in the head region (Möller et al. 2003, Kaba et al. 2007). Clinical signs occur mainly in young animals within the first 6 months of life (Fuente et al. 1997).

Epidemiological data concerning Morel's disease in goats, such as incubation period, persistence of clinical signs, course of the disease in a flock and changes of prevalence in time is limited. In October 2006 the disease was recognized for the first time in a goat flock in Poland (Kaba et al. 2007). A second infected flock was found two months later. The outbreaks occurred while both flocks were subject to an approximately five-meter wide corridor. All goats were dehorned. A common room was used for milking and seronegative animals. The offspring of seropositive goats was isolated immediately after birth and the kids were raised in separate premises. The goats were not grazed.

Subcutaneous abscesses had been prevalent in these goats for many years. Serological and microbiological investigations carried out in 2001 – 2002 confirmed the presence of CLA in this flock. The abscesses had been swabbed on various occasions – *C. pseudotuberculosis* was isolated five times and *Arcanobacterium pyogenes* once (Nowicki 2008).

In early spring 2005 the owner purchased a clinically healthy 3 year-old buck from Germany and kept it in confinement until mating. In the autumn of 2005 this buck joined the group of CAE seropositive does for 1 month, and was then moved to CAE seronegative females. Because no goat became pregnant, the buck was withdrawn from the flock.

In early spring 2006 the owner reported a significant increase in the number of goats with subcutaneous abscesses.

**Flock B** Flock B was established in December 2005, on the basis of 25 does purchased from flock A. At the time of delivery no abscesses were seen in these animals. The first abscesses appeared one month later and the number of affected goats tended to increase with time. During 2006 – 2008 eighteen goats from various herds with no history of subcutaneous abscessation were introduced into this flock. As in flock A, all kids were raised separated from their mothers. The kids were grazed on pasture, which had been used earlier by adult goats.

All animals in both flocks were clinically examined by palpation for presence of abscesses. Data on the age as well as number and location of abscesses found during each examination were recorded.

The monitoring started in October 2006 and was completed in February 2008. In flock A clinical examinations were carried out 9 times.

In flock B the animals were examined 7 times with the first examination in December 2006. For each examination an in-flock point prevalence was calculated as a proportion of diseased animals to the total number of goats in the particular group.

**Microbiological examination** In both flocks infection with *Staphylococcus aureus* susp. *anaerobius* was confirmed by microbiological examination of material collected from abscesses as well as from internal organs (liver, kidney, lung, and spleen) and mesenteric lymph nodes from autopsied goats.

Samples were transported to the laboratory where they were streaked, as soon as possible, on Columbia agar with 5% sheep blood and incubated under both microaerophilic and aerophilic condi-
tions for 48 hours at 37°C. The identification of *S. aureus* subsp. *anaerobius* was initially performed based on the growth in microaerophilic conditions and lack of growth in aerophilic conditions, microscopic examination (presence of Gram-positive cocci) and biochemical features (positive coagulase test, negative catalase and clamping factor tests). The identification of each isolate was confirmed genetically. Template DNA was obtained from pure cultures of strains. A few colonies were resuspended in 180 µl of TE buffer pH 8 (10 mM Tris-Cl, 1mM EDTA). Then 10 µl lysostaphin (SIGMA) were added. After incubation for 20 minutes at 37°C, 20 µl of proteinase K was added and the solution was incubated for 10 minutes at 37°C followed by 10 minutes at 75°C. The DNA was subsequently isolated with Genomic Mini (A&A Biotechnology) according to the manufacturer's recommendations and PCR of species-specific fragments of the *S. aureus* gene encoding 23S rRNA (5’primer sequence ACGGAGTTCAAAAGGACGAC, 3’primer sequence AGCTAGCCCTAACCAGTAC, PCR program 37 times 94°C for 40s, 64°C for 60s, 72°C for 75s) was performed. The strains were additionally investigated with PCR for sequences encoding *staphylococcal thermostable nuclease* (nuc) (5’primer sequence GGCGATTGATGGTGATACGGTT, 3’primer sequence AGCCAAGGCTTGACGAACTAAAGC, PCR program 37 times 94°C for 40s, 55°C for 30s, 72°C for 30s) and coagulase (coa) (5’primer sequence ATAGAGATGCTGGTACAGG, 3’primer sequence GCTTCCGATTGTTCCAGTGTC, PCR program 30 times 94°C for 40s, 58°C for 60s, 72°C for 60s) (Cabral et al. 2004).

*C. pseudotuberculosis* was identified based on microscopic examination (presence of Gram-positive, pleomorphic rods, singly or in pairs, often in “V” formation, creating “Chinese letters”) and biochemical properties testing using the Api Coryne System (Biomerieux) including a positive catalase test.

**Serological examination for CAE** Sera were collected in both flocks during the first clinical examination. The ELISA tests (Chekit-CAEV/MVV, Dr. Bommeli AG, Bern, Switzerland) were carried out according to the manufacturer's recommendations.

**Postmortem examination in flock A** Autopsies were performed on 4 goats with subcutaneous abscesses – 3 adults and one kid – and in 10 clinically healthy kids. Specimens for microbiological tests were obtained from all abscesses as well as internal organs (liver, kidney, lung, and spleen) and mesenteric lymph nodes.

**Results**

**Clinical examination**

**Incubation period** In 5 goats introduced into flock B clinical signs of Morel’s disease emerged after approximately 3 weeks of the first contact with infected animals.

**Prevalence of the disease** The in-flock prevalence is displayed in Fig. 1, 2 and 3. In flock A the point prevalence reached 93.6% within 2 months of onset of the disease and dropped to approximately 20 – 30% during the following 3 months. Similarly, in flock B the point prevalence decreased from 84.4% to about 10 – 20% within 3 – 4 months.

**Location, number, size and persistence of abscesses** The abscesses were located subcutaneously in various regions of the body (Fig. 4 and Table 1), often close to the lymph nodes. During the first months of the outbreak the presence of 2 to 7 abscesses in one goat was a common observation, whereas at the end of the study in all diseased animals only single abscesses could be noted. The time between appearance of an abscess and its spontaneous rupture varied from 1 to 5 months (Fig 6). Mature abscesses were usually approximately 10 cm in diameter, while the largest reached 30 cm (Fig. 7). Most abscesses tended to disappear after two months, no scars remained and hair grew back.

**Postmortem examination**

No profound abscesses and no other lesions of internal organs were discovered neither in 3 diseased adult goats nor in 11 kids.

Superficial abscesses (from one to six per an individual) were found in three adult goats and in one kid and all were located close to the lymph nodes.

**Microbiological examination**

*S. aureus* subsp. *anaerobius* was cultured in 43 of 46 swabs collected from live animals manifesting clinical signs. In three cases *C. pseudotuberculosis* was isolated.

As regards the post-mortem material, *S. aureus* subsp. *anaerobius* was isolated in pure culture from all superficial abscesses in 3 adult goats and one kid. *S. aureus* subsp. *anaerobius* was not found in internal organs and lymph nodes obtained from all autopsied animals.
Fig. 1. The in-flock point prevalence of Morel's disease in goats during the observation period.

Fig. 2. Percentage of young (< 1 year-old) and adult (> 1 year-old) animals among goats with Morel's disease.

Fig. 3. Prevalence of Morel's disease among CAE seropositive and CAE seronegative goats (flock A).
Table 1. Location of subcutaneous abscesses in goats with Morel’s disease.

<table>
<thead>
<tr>
<th>Region or lymph nodes near which abscesses were located</th>
<th>Flock A</th>
<th></th>
<th>Flock B</th>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of abscesses</td>
<td>Percent of all abscesses (%)</td>
<td>Number of abscesses</td>
<td>Percent of all abscesses (%)</td>
<td>Percent of all abscesses (%)</td>
</tr>
<tr>
<td>Cervical superficial lnn.</td>
<td>338</td>
<td>36.90</td>
<td>66</td>
<td>35.87</td>
<td>36.38</td>
</tr>
<tr>
<td>Subiliac lnn.</td>
<td>315</td>
<td>34.39</td>
<td>54</td>
<td>29.35</td>
<td>31.87</td>
</tr>
<tr>
<td>Parotid lnn.</td>
<td>96</td>
<td>10.48</td>
<td>24</td>
<td>13.04</td>
<td>11.76</td>
</tr>
<tr>
<td>Mandibular lnn.</td>
<td>90</td>
<td>9.83</td>
<td>20</td>
<td>10.87</td>
<td>10.35</td>
</tr>
<tr>
<td>Mammary lnn.</td>
<td>40</td>
<td>4.37</td>
<td>8</td>
<td>4.35</td>
<td>4.36</td>
</tr>
<tr>
<td>Neck</td>
<td>21</td>
<td>2.29</td>
<td>10</td>
<td>5.43</td>
<td>3.86</td>
</tr>
<tr>
<td>Chest</td>
<td>8</td>
<td>0.87</td>
<td>0</td>
<td>0.00</td>
<td>0.44</td>
</tr>
<tr>
<td>Cheek</td>
<td>3</td>
<td>0.33</td>
<td>0</td>
<td>0.00</td>
<td>0.16</td>
</tr>
<tr>
<td>Ear and wattle</td>
<td>2</td>
<td>0.22</td>
<td>2</td>
<td>1.09</td>
<td>0.65</td>
</tr>
<tr>
<td>Sternal lnn.</td>
<td>1</td>
<td>0.11</td>
<td>0</td>
<td>0.00</td>
<td>0.05</td>
</tr>
<tr>
<td>Tail base</td>
<td>1</td>
<td>0.11</td>
<td>0</td>
<td>0.00</td>
<td>0.05</td>
</tr>
<tr>
<td>Popliteal lnn.</td>
<td>1</td>
<td>0.11</td>
<td>0</td>
<td>0.00</td>
<td>0.05</td>
</tr>
<tr>
<td>Total</td>
<td>916</td>
<td>100</td>
<td>184</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Fig. 4. Percentage of abscesses located subcutaneously in different regions of the body in goats with Morel’s disease.
CLA caused by \textit{C. pseudotuberculosis} is a very well documented condition which leads to abscess formation in small ruminants. It is a chronic disease characterized by subcutaneous abscession of one or more lymph nodes. Less frequently it may also result in pneumonia, hepatitis, mastitis, spontaneous abortion or stillbirth (Coetzer and Tustin 2004). CLA occurs in many countries (Batey 1986, Ellis et al. 1987, Skalka et al. 1998, Ben Said et al. 2002), including Poland (Kaba 2001) and has significant economic importance. Large scale epidemiological studies on the prevalence of this infection in goats have been carried out in Poland twice and revealed that the percentage of infected flocks increased from 13\% (10 out of 76 flocks) in 1996 (Kaba 2001) to 66\% (43 out of 65 flocks) in 2002 (Nowicki 2008). Moreover \textit{Arcanobacterium pyogenes}, the opportunistic pathogen commonly found in the environment and frequently isolated from suppurative lesions in goats, was isolated occasionally in both studies (El Sanousi et al. 1989, Gezon et al. 1991). However \textit{S. aureus} subsp. \textit{anaerobius} had never been detected until October 2006 (flock A). This seems to be the first outbreak of Morel’s disease in Poland (Kaba et al. 2007).

Non-numerous subcutaneous abscesses had been noted regularly in flock A for many years. CLA was
Fig. 7. Numerous abscesses caused by *Staphylococcus aureus* subsp. *anaerobius*.

microbiologically and serologically confirmed to be the only cause and its mild course suggested the presence of significant flock immunity to this endemic infection. In spring 2006 the number of animals with subcutaneous abscesses, as well as the intensity of clinical signs significantly increased. Up to 7 abscesses on an individual animal were found simultaneously, reaching up to 30 cm in diameter (Fig. 7).

A new pathogen – *S. aureus* subsp. *anaerobius* - was found responsible for the change in clinical manifestation. Among 30 isolates from samples collected during 5 visits to this flock, *S. aureus* subsp. *anaerobius* accounted for as many as 28 while *C. pseudotuberculosis* for only two. Morel’s disease must have been introduced into this flock in autumn 2005. Between spring 2005 and early autumn 2006 only one goat, a buck imported from Germany, was introduced into this flock. The owner has not observed any abscesses or enlarged lymph nodes in this animal. In autumn 2006 during the mating season the buck joined the group of CAE seropositive does, and was then moved to CAE seronegative does. The dynamics of Morel’s disease in both these groups indicates that the disease appeared first in CAE seropositive does and with some delay in CAE seronegative animals (Fig. 3). This corresponds to the history of moving the imported buck within the flock, supporting the hypothesis on the source of *S. aureus* subsp. *anaerobius* infection in this outbreak. As an insufficient reproductor the buck was euthanized. Unfortunately, neither autopsy nor any further investigation of this case was conducted, making verification of this hypothesis impossible. There are no reports about Morel’s disease from Germany, but misdiagnosis with much more prevalent CLA cannot be excluded.

There are no data on the incubation period for Morel’s disease. In flock B infected with *S. aureus* subsp. *anaerobius* 5 goats developed abscesses 3 weeks after being introduced into the flock. It cannot be excluded that some abscesses in these 5 goats could have been caused by *C. pseudotuberculosis*, as this bacterium was also present in flock B. However, among 16 swabs taken from this flock *C. pseudotuberculosis* was isolated in one case only, whereas in all others *S. aureus* subsp. *anaerobius* was cultured. The incubation period of Morel’s disease in natural outbreaks in goats seems therefore to approximate 3 weeks. For comparison the incubation period of CLA in goats is much longer and varies from 2 to 6 months (Williamson 2001). The different incubation period for CLA and Morel’s disease may explain why these diseases predominate in different
age groups. Our observations indicate that if Morel’s disease is introduced into a flock for the first time (epidemic stage of the infection), it affects both young (less than 1 year) and adult goats to the same degree. This could be observed in flock B where the prevalence in young and adult animals was similar from the onset of the disease (Fig. 2). Although both age groups were kept separately, the limited experience of their owner resulted in grazing of young animals on pastures used also by adult goats, so the isolation was not tight. In flock A after the introduction of the suspected buck abscesses were seen mostly among goats older than 1 year (Fig. 2). However, young animals were kept in separate premises and strict isolation was probably the reason for which the disease emerged in kids with delay. Nevertheless, at the end of the observation period, when Morel’s disease was endemic in both flocks, as much as 65 – 80% of goats with abscesses were less than 1 year old (Fig 2). This is consistent with earlier observations (Fuentes et al. 1997) and seems to be a significant difference as compared with CLA, where clinical symptoms are rarely noted in animals younger than 1 year (Holstad 1986). Acquired immunity of older goats to *S. aureus* subsp. *anaerobius* could explain this observation.

As a CAE control program was carried out in both flocks the kids were raised without access to caprine colostrum. Lack of maternal antibodies probably resulted in a much higher prevalence rate in young animals than in adults in the endemic stage of the infection. Little is known about the immunity to *S. aureus* subsp. *anaerobius* in goats, but it has been suggested that maternal antibodies may persist in the offspring for up to 20 weeks (Rodwan et al. 2004).

Increasing immunity of the goats can explain the gradual decrease in disease prevalence and the intensity of clinical manifestation in individual animals in both flocks. Clinical signs of Morel’s disease in goats were most evident during the first 3 – 4 months after infection of the flock and then gradually disappeared (Fig. 5 and 6).

Clinical and postmortem examination confirmed that in the Morel’s disease the abscesses are located mostly near superficial lymph nodes, symmetrically on both sides of the body, most frequently near the cervical superficial (36%) and subiliac (32%) lymph nodes. Less frequently the abscesses were located in the head region next to the parotid (12%) and mandibular (10%) lymph nodes (Table I). This indicates that in Morel’s disease the abscesses are located in similar regions as in CLA, where the lesions are seen mostly in the anterior part of the body, in the parotid (37%), mandibular (27%) and less frequently in the cervical superficial (18%) and mammary lymph nodes (10%) (Menzies and Muckle 1989). It has been well documented that CLA spreads mostly through damaged and undamaged skin (Ellis et al. 1987, Williamson 2001, Dorella et al. 2006). This route of infection has also been postulated for *S. aureus* subsp. *anaerobius*. Superficial location of the abscesses in goats with Morel’s disease seems to confirm this statement. Domination fights frequently occur in goat flocks and use of horns results in wounds located especially on the head. In Morel’s disease the head is also expected to be the most frequent location for abscesses. However, in both flocks investigated in this study the goats were dehorned. This may explain why in these outbreaks the head was not the most frequent site of abscess formation (Fig. 4).

No internal abscesses or lesions caused by *S. aureus* subsp. *anaerobius* were found in 14 autopsied goats. This is consistent with earlier reports where only superficial abscesses were recorded in Morel’s disease. Although in CLA the superficial form of the disease also predominates in goats, in 20% of diseased goats internal abscesses can also be found (Batey 1986). During both outbreaks described in this study the process of forming and maturing abscesses (from formation to spontaneous rupture) lasted from 1 to 5 months, which resembles CLA (Pegram 1973).

Previous data indicated that in Morel’s disease the abscesses develop not in the lymph node, as in *C. pseudotuberculosis* infection, but in the adjacent area of the node (Menzies and Muckle 1989, Williamson 2001, Fontaine and Baird 2008). This is consistent with our observations in which we have not noted any abscesses inside the lymph nodes. Most of these abscesses were located near the nodes.

In the group of CAE seropositive goats the prevalence of Morel’s disease was initially over 3-fold higher than in CAE seronegative animals (Fig. 3). However, this difference was probably due to the fact that the buck suspected to be the source of *S. aureus* subsp. *anaerobius* joined the group of CAE seropositive does first, so the seronegative ones were infected later. In the next examination, after 4 months, abscesses were more frequently seen in CAE seronegative goats and later, as the overall prevalence decreased, 20 – 40% of animals from both groups manifested abscesses. It seems that the CAEV infection had no influence on the course of Morel’s disease in this flock.

In conclusion, an outbreak of subcutaneous abscessation in a goat flock suggests that two infectious diseases should be considered: Morel’s disease and caseous lymphadenitis (CLA). The most distinct differences are: shorter incubation period and higher prevalence rate among young goats in Morel’s disease, as well as location of abscesses near, not inside, the lymph nodes as is the case in CLA. However, the clinical course of Morel’s disease in a goat flock resembles the course of CLA, thus making clinical misdiagnosis possible and might explain the relatively rare recognition of Morel’s disease in Europe.
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References


