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SCIENTIFIC COMMUNICATION

MASTITIS AFTER INDUCED MAMMOGENESIS IN A NULLIPAROUS GOAT

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ABSTRACT

This is a case report on a nulliparous Toggenbourg goat, one year of age that, after being submitted to superovulation with human menopause gonadotrophin, presented mammogenesis and lactogenesis. Both neoformed mammary glands were naturally infected with β-hemolytic *Staphylococcus aureus* and evolved clinically in different forms. The left half evolved to acute catarrhal mastitis that responded positively to treatment using sodium cloxacillin, whereas right mammary gland evolved to phlegmonous gangrenous mastitis, with teat loss. The mammary tissue remaining had to be surgically removed. The present report emphasizes that multi-tissue effect should not be ignored when hormonal therapy is used. The potential risk of induced mammogenesis in nulliparous animals and the nosological diversity that mastitis may present should be considered, once the etiological agent and host were the same, and different inflammatory responses were observed in the two halves.

KEY WORDS: Goat, milk, mastitis, mammogenesis.

RESUMO

MASTITE EM CABRA NULÍPARA APÓS A INDUÇÃO DA MAMOGÊNESE. Relata-se o caso de uma fêmea caprina nulípara da raça Toggembourg, com 1 ano de idade, que após ser submetida à superovulação com gonadotrofina da menopausa humana (hMG) apresentou mamogênese e lactogênese. Ambas as mamas neoformadas foram naturalmente infectadas com *Staphylococcus aureus* β-hemolítico e evoluíram clinicamente de formas distintas. A glândula mamária esquerda evoluiu para uma mastite catarral aguda que respondeu positivamente ao tratamento com cloxacilina sódica, enquanto a mama direita evoluiu para mastite flegmonosa gangrenosa, com desprendimento do teto, sendo o tecido mamário restante removido cirurgicamente. O presente relato salienta a importância de não desconsiderar o efeito multitecidual que a terapia hormonal apresenta, o potencial risco da mamogênese induzida em nulíparas e a diversidade nosológica que a mastite pode apresentar, uma vez que o agente etiológico e o hospedeiro eram os mesmos, com resposta inflamatória distinta das mamas.

PALAVRAS-CHAVE: Caprino, leite, mastite, mamogênese.

The mammary gland is a complex tissue that varies according to its development, secretion and involution. These variations are controlled by complex mechanisms involving combination of hormones, paracrine local action and autocrine factors (KNIGHT, 2001).

The mammary gland starts to develop in puberty, stimulated by the estro cycle progesterone and estrogen, and mammogenesis effect of prolactin and growth hormone (GH) (SENGER, 2003). However its greater development occurs during pregnancy, mainly in the last trimester, when mammary parenchyma is transformed in structures filled by alveoli that synthesize and actively secrete milk (SWENSON; REECE, 1996; REECE, 1997; SEJRSEN, 1999; KNIGHT, 2001). Before parturition, cells in the alveolar lobe undergo biochemical and morphological changes, becoming able to produce milk (SEJRSEN, 1999).

In goats, the udder presents some morphofunctional particularities. It is formed by two mammary glands and two teats located in the inguinal region. Six to nine large ducts meet in a teat cistern that

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presents a ventral orifice (SMITH; SHERMAN, 1994; SWENSON; REECE, 1996; SENGER, 2003).

The present case report studies a nulliparous Toggenbourg female, one year of age, superovulated with 1200 UI (600 UI of FSH and 600 UI of LH) of human menopause gonadotrophin (hMG). For the synchronization of oestrus was utilized a sponge impregnated with 60 mg of medroxiprogesteron of acetate for 10 days. Forty eight hours before the removal of the sponge, the medication with hMG has started by musculature IM, administrated in decreasing doses every 12 hours, for 30 consecutive days, at 8 and 9 AM and 8 and 9 PM

After the treatment, the mammary gland developed increased parenchyma volume and presence of galactorrhea. It then became progressively sensitive to palpation and showed signs of local inflammation. Inflammation of each half of the mammary gland evolved differently. The left half maintained discrete inflammation and milk presented clumps, what was characterized as catarrhal mastitis. Right half evolved from inflammation to necrosis (phlegmonous gangrenous mastitis), with cooling of the gland and production of bloody secretion. The right half became dry and dark, and the teat and skin were lost with consequent exposure of the remaining parenchyma (Fig. 1), which was surgically removed.

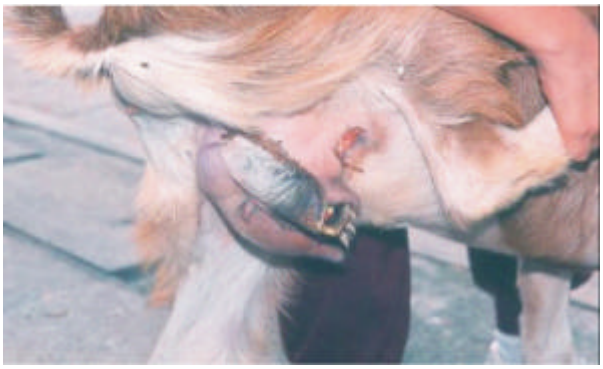

Fig. 1 - Mammary gland presenting mastitis, acute catarrhal on the left half and phlegmonous gangrenous with skin loss on the right half.

In the microbiological examination of the milk from the left half and from the right parenchyma *b*-hemolytic *Staphylococcus aureus* was identified. This agent was considered by NDEGWA (2001) as the greatest prevalent agent in caprine mastitis, what was complemented by PAES et al. (2003), showing the greater susceptibility of the animals in the beginning of lactation.

Treatment followed the recommendations by SMITH;SHERMAN (1994) and MENZIES;RAMANOON (2001), using antibiotic therapy and surgical removal of the remaining necrosed tissue (BEZEK; HULL, 1995). Sodium cloxacillin was used, with good results in the left half, although LIMA JÚNIOR et al. (1993) and CASTRO et al. (2001) reported gentamycin and cephalothin as presenting the greatest *in vitro* sensitivity. In dairy cows, outbreaks of gangrenous mastitis caused by *Pseudomonas aeruginosa* have provoked the death of the animals just days after the administration of mammary treat to dry the milk. Despite the routine use of cloxalin in these herds, drier hygiene and use of large spectrum antibiotics, efficacious against gram-negative microorganisms, were prescribed (CUTTLER, 2003; MILNES; PLATTER, 2003; POWER, 2003).

No systemic changes were observed. Another interesting finding was that although the same agent was isolated from both glands, different clinical manifestations were observed. According to (SILVA et al., 2005) the *S. aureus* is characterized by its pathogenicity, which is due to its production of a wide variety of exoproteins, such as alpha, beta and delta hemolysins, that contribute to bacterial invasion and inhibit the host immune response. The same bacterium with different pathogenicity caused different clinical signs in the same host. Therefore, the immunological system of the host responded differently for each mammary gland.

Exogenous hormones, like these used in a superovulation program, change the concentration of endogenous hormones, leading to multi-tissue effects, including the mammary parenchyma. A nulliparous goat, with mammary glands not mature, when submitted to this stimulus can either overcome the problems without any complications, or present greater susceptibility to changes in the mammary gland and milk production. So the importance of this report is to emphasize that care should be taken in relation to the selection of embryo donors, avoiding hormonal treatment for superovulation in nulliparous goats, mainly those of dairy lineages, once the endocrine multi-tissue effect favors the iatrogenic mammogenesis reported in the present study, which culminated in the clinical complication observed.

Another example of the endocrinous multi-tissue effect and its dependence on the functional condition of the mammary gland is described for GH that, if used in the beginning of the lactation, does not affect the volume of mammary tissue, whereas if administered in the middle of the lactation, determines an increase in the parenchyma tissue (SEJRSEN, 1999; BOUTINAUD et al., 2003).

The present report emphasizes that multi-tissue effect should not be ignored when hormonal therapy is used. The gonadotrophin is not responsible for the mastitis, but we emphasize that it is necessary to take certain precautions while selecting the animals and the adequate the superovulation techniques. The

potential risk of induced mammogenesis in nulliparous animals and the nosological diversity that mastitis may present should be considered (GARCIA et al., 1994; PUGH, 2005), as the etiological agent and host were the same, and different inflammatory responses were observed in the two mammary halves.

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