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Impact of Subclinical Mastitis on the Health of The Mammary Gland

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Abstract: The aim of our study was to see the impact of subclinical mastitis on the health of the mammary gland knowing that they pass unnoticed. To address our problem of fragments belonging to the mammary gland of dairy cows brought to the slaughter house after the fall of milk yield due to subclinical mastitis. Among the 52 samples from mammary glands of dairy cows with subclinical mastitis 32 showed no change while the remaining tissue samples showed various degrees of inflammation. Even suffering from subclinical mastitis udder tissue structures was abnormal.

Key words: Bovine · Mastitis · Epithelial Cells · Apoptosis · Tissue Damage

INTRODUCTION

Bovine mammary glands are exposed to diverse bacteria throughout lactation and nonlactating periods. Pathogens commonly isolated from mastitis milk can be classified as non- contagious (most are environmental) and contagious microorganisms. Mastitis is the most costly infectious disease of dairy cattle.

Milk production drops sharply and the milk from treated cows can be marketed, however, the losses associated with subclinical mastitis are more serious, it is obvious that prevention against mastitis is a major concern in the dairy herd. In very severe cases, the infection progressed via the ductile system to produce a limited inflammatory reaction but with an extensive involvement of the secretory tissue [1].

Heald [2] observed that mammary tissues from lactating cows inoculated with S. *aureus* exhibited less milk synthesis and secretion, as evidenced by more interalveolarstroma and involuting alveolar epithelium and less alveolar luminal space compared with uninfected contra lateral controls.

The mammary tissue damage has been shown to be induced by either apoptosis or necrosis [3]. Two distinct types of cell death can be distinguished by morphological, biochemical and molecular changes in dying cells. The aim of our paper was to consider relationships between structure of the bovine mammary gland and the impact of subclinical mastitis.

MATERIALS AND METHODS

52 samples from mammary glands of dairy cows introduced to the slaughterhouse due to problems of subclinical mastitis underwent histological sections to highlight potential tissue damage.

These samples collected immediately after slaughter were preserved in jars containing formalin to ten percent (10%), then they were transported to the pathology laboratory.

Anti Mortem Examination: General physical and clinical examinations were conducted before slaughters in cows. The clinical form of mastitis has been detected by the presence of signs of inflammation (reddening, heat and pain). Subclinical mastitis were diagnosed by C.M.T. (California mastitis test).

Generally, cows brought to the slaughterhouse were accompanied by a slaughter certificate for various reasons, but the cows selected for the realization of our study were suffering from drop in milk production due to mastitis.

It should be noted that some subjects showed no clinical signs, pathological form was subclinical.

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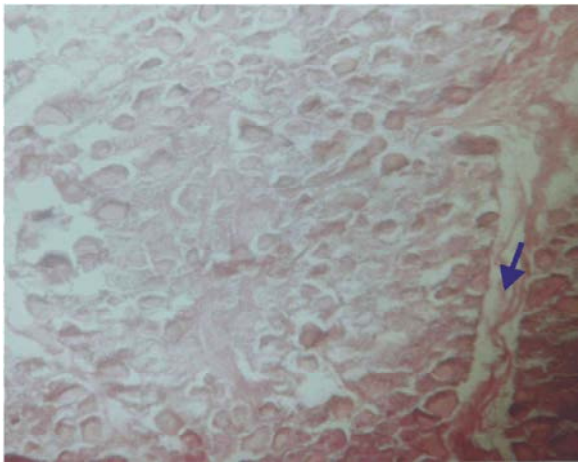

Fig. 1: Udder lactating large acini, spans connective very thinned (blue arrow) (Stain H&E, X40)

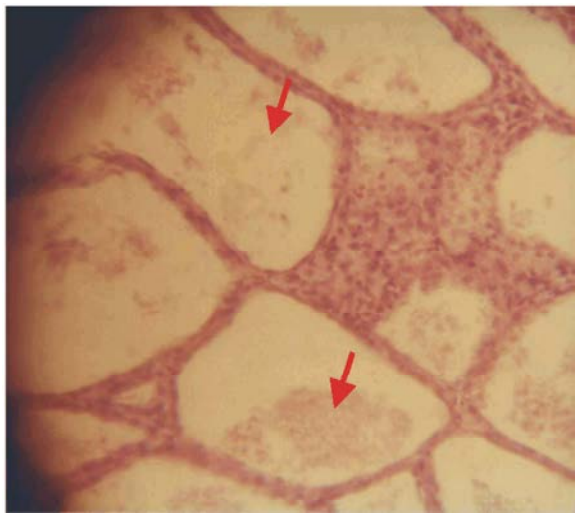

Fig. 2: Teat-end activity acini are wide light (red arrow) (Stain H&E, X100)

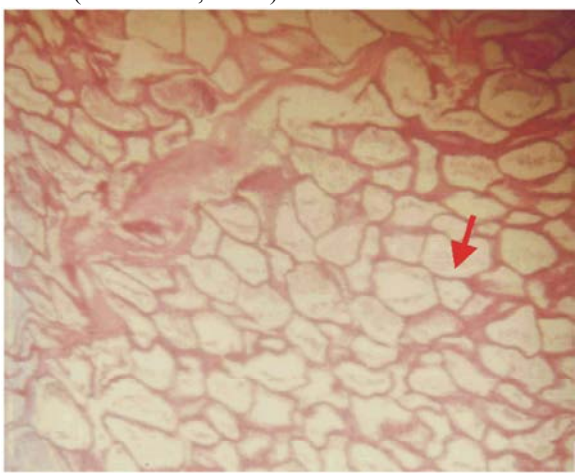

Fig. 3: Breast lactating activity in mammary acini (Red arrow) (Stain H&E, X40)

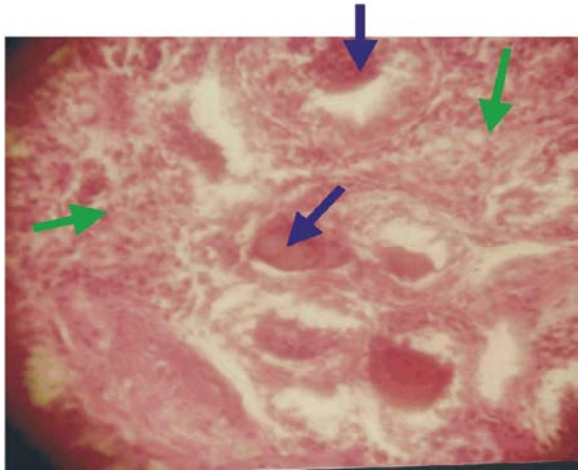

Fig. 4: Intense inflammatory reaction, destruction of a large number of cells, Light filled with blood (blue arrow) and invaded neutrophils (green arrow) (Stain H&E, X40)

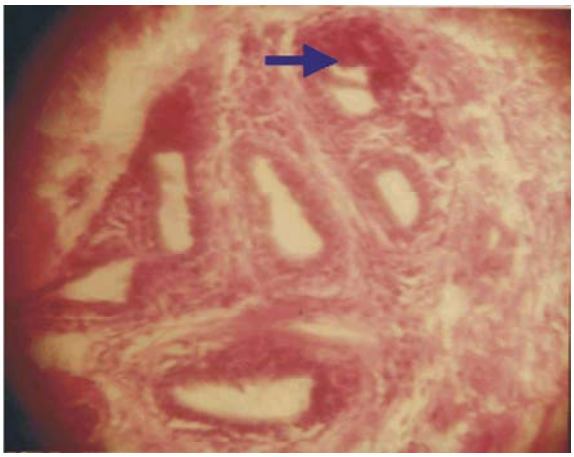

Fig. 5: Foci of fibrosis with intense tissue reaction (blue arrow), congestion and the presence of inflammatory edema (Stain H&E, X40)

RESULTS

Some samples (n = 32) or 61.53%, were structurally normal tissue, the breasts in full operation with large acini, cells had normal contours and connective tissue trabeculae are very thin. No clinical signs at their unit lactiferous, while others had a normal tissue structure.

Other samples have shown images of a teat-end activity, manifesting as basal cell and cubic (Fig 1.2, 3).

The second category of samples (n = 18) or 34.61% showed inflammatory lesions different from the disappearance of the alveolar lumen, through fibrosis to the complete destruction of the parenchyma (Fig 4- 5, 6).

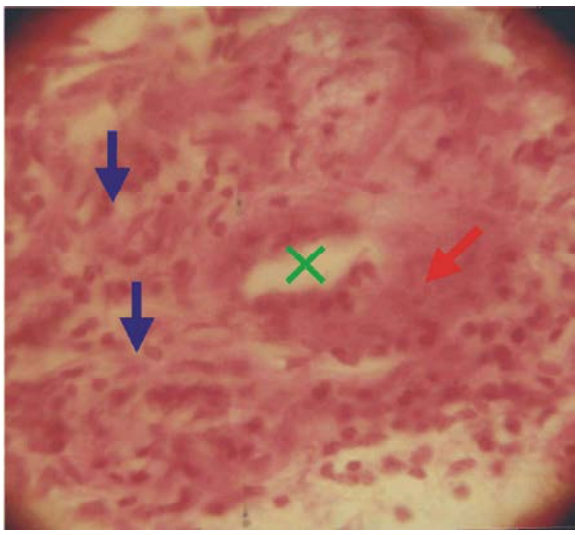

Fig. 6: Destroyed cell (X), with a very large intense inflammatory reaction and polynuclear invasion (blue arrow), with foci of congestion (red arrow) (Stain H&E, X40)

DISCUSSION

Histological analyses have been widely used since the 1970s and are still being used today for assessing damage to secretory tissue in the bovine mammary gland caused by mastitis pathogens. For example, Benites *et al* [4] examine the 131 mammary parenchyma from 184 slaughtered dairy cows for existence of microorganisms and histopathological changes. Of all the samples from which microorganisms were isolated, only 3.1% did not show histological changes. The remaining 96.9% of samples showed inflammatory response.

According to Zhao and Lacasse [3] lesions of the breast tissue reduces the number and activity of epithelial cells and therefore contributes to lower milk production with increasing proportions of lymphocytes and macrophages and a decrease cell number of polymorphonucléaires [5].

One of the most common types of chronic mastitis is caused by S. aureus.

Histopathological responses of lactating tissue to experimental or naturally occurring S. ureus mastitis were extensively studied during the 1970 and 1980. Chandler and Reid [6]examined mammary parenchymal tissue samples from lactating cows infected naturally with S.aureusand reported a massive polymorphonuclear neutrophils infiltration and necrosis of secretory tissues. In addition, Heald [2] observed that mammary tissues from lactating cows inoculated with S. aureus exhibited less milk synthesis and secretion, as evidenced by more interalveolarstroma and involuting alveolar epithelium and less alveolar luminal space compared with uninfected contralateral controls.

Staphylococcus aureus produces toxins that destroy cell membranes, directly damage milk producing tissue and induce necrosis in bovine mammary glands [7- 8]. Initially, the bacteria damage tissues lining the teat and gland cisterns within the quarter.

Some bacteria produce toxins that actually destroy cell membranes and damage milk producing tissue, while other bacteria are able to invade and multiply in bovine mammary epithelial cells before causing cell death [9].

According Trinidad *et al.* [7], mammary glands infected with Staphylococcus aureus, showing an increase of connective tissue inter-cellular and a reduction in epithelial cells and alveolar lumen. These same authors report an invasion leukocyte, mainly lymphocyte and polymorphonuclear.Oxidative damage to epithelial cells of the mammary gland is among the causes of the decline phase of lactation in mammals.

Chandler and Reid [6] examined samples of bovine udder infected with Staphylococcus aureus and have noticed a parenchymal leukocyte infiltration intense necrosis with a very important level of secretory tissue.

The enzymes involved in bovine mammary tissue destruction were investigated by Mehrzad *et al* [10] using an endotoxin-induced mastitis model. They reported that mastitic milk proteases hydrolyze casein, gelatin, collagen, hemoglobin, mammary gland membrane proteins and lactoferrin, indicating that mastitic milk proteases have a broad spectrum of activities.

Multiple assay methodologies and different terminologies have been used by different research groups to study and describe tissue damage during mastitis, respectively. Thus, a brief introduction to types of cell death is warranted. There are two distinct types of cell death, apoptosis and necrosis, which may be distinguished by morphological, biochemical and molecular changes in dying cells. Apoptosis is a process of deliberate suicide of a cell in multicellular organisms. The process of apoptosis was originally distinguished from necrosis on the basis of cellular ultra structure [11]. Apoptosis may be identified by a characteristic pattern of morphological changes including nuclear and cytoplasmic condensation, nuclear fragmentation and formation of apoptotic bodies [12].

When the infection persists and the channels are blocked, the milk within the alveoli increases the pressure there, the secretory cells lose their ability to synthesize

and the cells begin to atrophy. Substances released by white blood cells cause destruction of cellular structures, which are replaced by scar.

Conflict of Interest Statement: None of the authors of this paper has a financial or personal relationship with other people or organizations that could inappropriately influence or bias the content of the paper.

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