Basrah Journal of Veterinary Research,Vol.17, No.3,2018
Proceeding of 6th International Scientific Conference,College of Veterinary Medicine
University of Basrah,Iraq

HISTOPATHOLOGICAL STUDY OF MAMMARY GLAND INFECTION IN DAIRY BOVINE

Alaa T. Abdulwahid[*] , Zainab W. khutair[**], Raghad I. Abd[***]

[*]Department of Veterinary Public Medicine, College of Veterinary Medicine, University of Basrah, Iraq.

[**]Department of Veterinary Microbiology and Parasitology, College of Veterinary Medicine, University of Basrah, Iraq.

[***] Department of Clinical Laboratory Sciences, Pharmacy College, University of Basrah, Iraq.

Keywords: Mammary gland ,Bovine ,slaughter house.

Corresponding Author: alaatariqabdulwahi@gmail.com

ABSTRACT

A total of nine mammary glands of bovine were collected from slaughter house in Basrah city and Surgical Department of Veterinary College for histopathological examination. Histopathological examination of mammary glands revealed that there was an acute, chronic, and necrotizing case in mammary gland tissue.

INTRODUCTION

Bovine mastitis is one of the most important diseases affecting the dairy industry. It is an economic burden on milk producers all over the world [1]. An inflammation of the mammary gland parenchyma is characterized by pathological changes in udder tissue [2]. The diagnosis of mastitis is based on clinical signs (swelling of the udder, tender to the touch, fever, and depression). For subclinical mastitis cases, the diagnosis depends on the leukocyte numbers in the milk [3]. Both clinical and subclinical mastitis can affect the milk composition and reduce milk production [4, 5]. During infection of the udder with mastitis there is lowering of lactose, casein, and reduction in natural

acidity. However, there is an increase in chloride content, soluble nitrogen, and ash content [4].

Etiology of mastitis can be bacterial and non-bacterial pathogens, such as mycoplasms, fungi, yeasts, and chlamydia [1]. These pathogens infect the udder via the teat canal and multiply in the milk of the teat and mammary cisterns [6]. The damage of tissue mammary gland can be caused by pathogens and their products. Toxins produce by certain type of pathogen can be destroyed cell membranes and damaged milk-producing tissue. While, other type of pathogen have the ability to invade and proliferate within the epithelial cells before causing cell death. In addition, mastitis is characterized by an increase of somatic cells, which in turn cause damage of the blood-milk barrier and mammary epithelium [7]. Taken to gather, this work aimed to study the histopathological changes in the mammary gland.

MATERIAL AND METHODS

Experimental design:

The study was carried out on nine mammary glands of bovine. They were collected from slaughter house in Basrah city and Surgical Department of Veterinary College for histopathological examination.

Histopathological examination:

At slaughter, the udder was incised quickly and mammary gland parenchyma was fixed in 10% formalin at room temperature for 24 h. The specimens were then removed from the buffered-formalin and dehydrated through a graded series of ethanol and xylene prior to paraffin embedding. After that, the specimens were embedded in paraffin, sectioned at the thickness of 5 microns using rotary microtome, mounted on slide, and stained with haematoxyline-eosin as described by [8]. The slides were then examined under light microscope (Olympus) to detect and describe any histopathological changes in mammary gland parenchyma induced by bacteria and non-bacterial pathogens

RESULTS

The udder section from healthy bovine revealed no pathological lesions with normal alveoli and glandular structure (Figure1). However, the tissue sections of mastitic bovine revealed inflammatory changes. Microscopically, acute cases of mastitis showed hyper secretion in acini of duct which appeared as pink or orange color in the lumen of duct some of acini (Figure2). There was also hemorrhagic intralobular space with degeneration of epithelial lining cell, slightly inflammatory cells, and odematus fluid in many area of tissue (Figure3). Some of the duct is dilated and there were congested blood vessels (Figure 4).

Chronic cases of mastitis revealed that there was hyperplasia of duct with connective tissue proliferation (Figure 5, 6). There was infiltration of inflammatory cell associated with dilated some acini and hyperplastic (Figure 7). Excessive amount of fibrosis with dilated of acini was also seen (Figure 8). Adenocarcinoma and aplastic epithelium cell with swelling of hemorrhagic area was also seen (Figure 9, 10).

Necrotic cases of mastitis revealed that there was necrotic area which appeared as replaced by excessive amount of inflammatory cells, newly blood vessel, and loss of structure disorentation of mammary gland tissue. This necrotic area pressed caused pressure atrophy to the other tissue which closed to some acini and dilated other (Figure 11, 12).

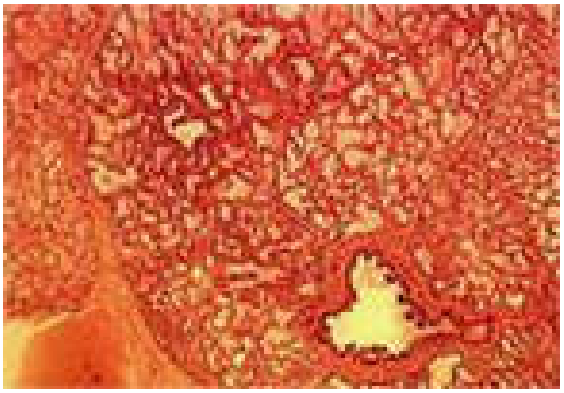

Fig 1 : No pathological lesions with normal alveoli and glandular structure [9].

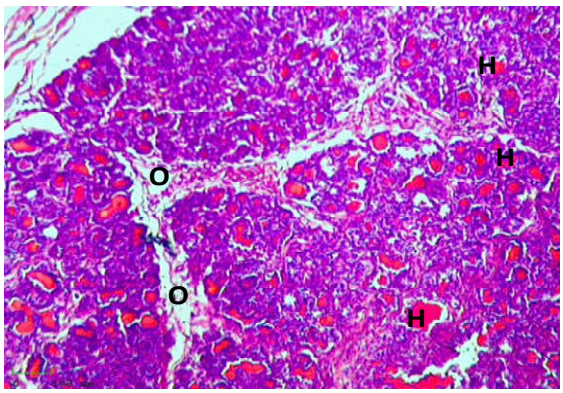

Fig 2 :Section of mammary gland of bovine showing hyper secretion of acinor duct(H) and intralobular odema(O). X10 H and E

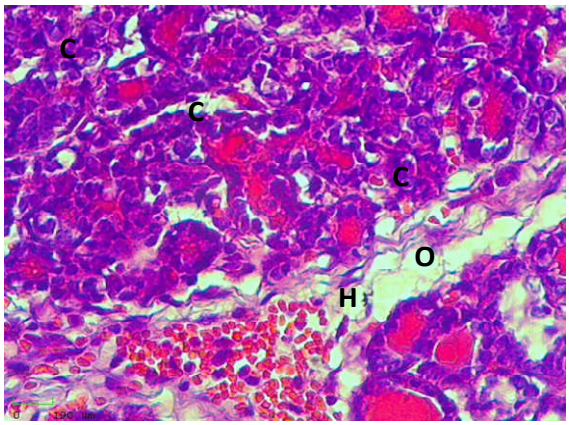

Fig 3: Section of mammary gland of bovine showing closed some of duct (C), haemorrhage (H)with intralobular odema(O) and degeneration of epithelial cells

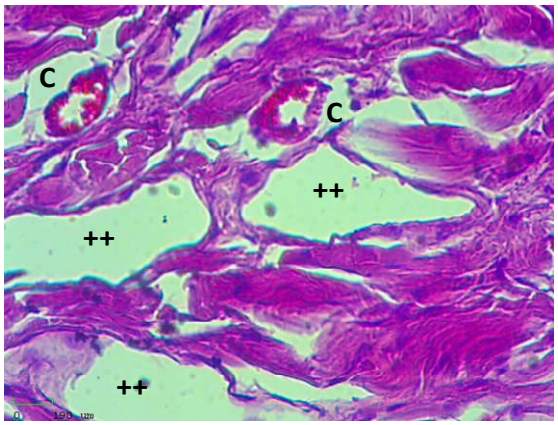

Fig 4 : Section of mammary gland of bovine showing large glandular duct near by acini dilated the duct (++), congested blood vessels(C)

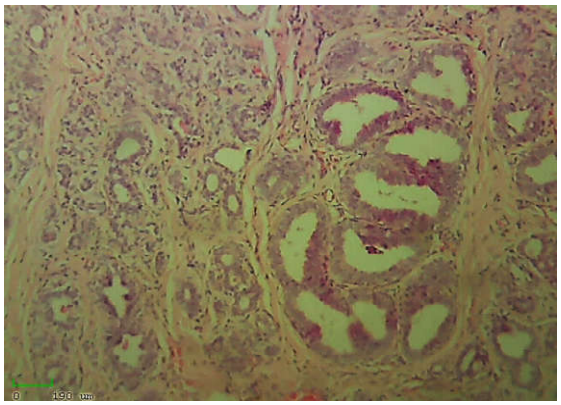

Fig 5: Section of mammary gland of bovine showing hyperplesia of mammary gland. x10

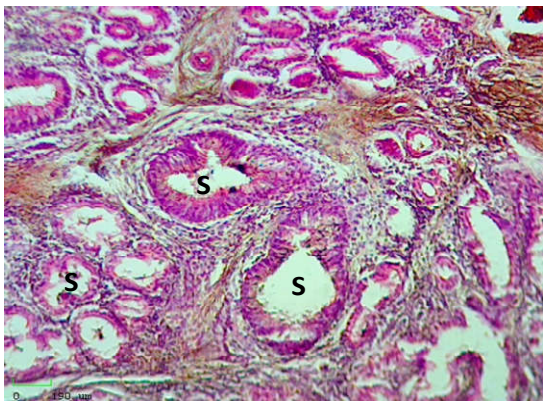

Fig 6: Section of mammary gland of bovine showing some of the mammary duct with papillary epithelium proliferation in some area(S).

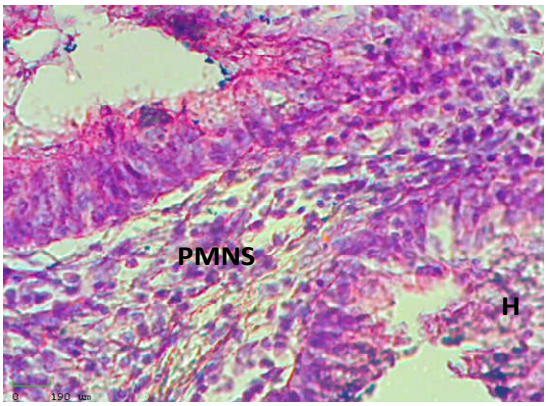

Fig 7 : Section of mammary gland of bovine showing presence of inflammatory cells(PMNs) in periductal region. Some areas prominent acni and hyperplastic (H) .X40

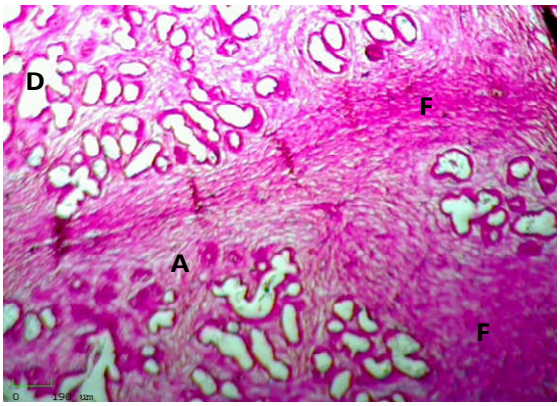

Fig 8 : Section of mammary gland of bovine showing excessive fibrosis (F) with atrophy some acini (A) and dilated the other (D)

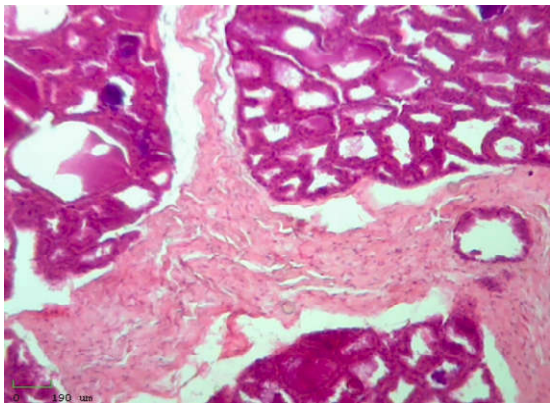

Fig 9: Section of mammary gland of bovine showing adenocarcinoma . X10

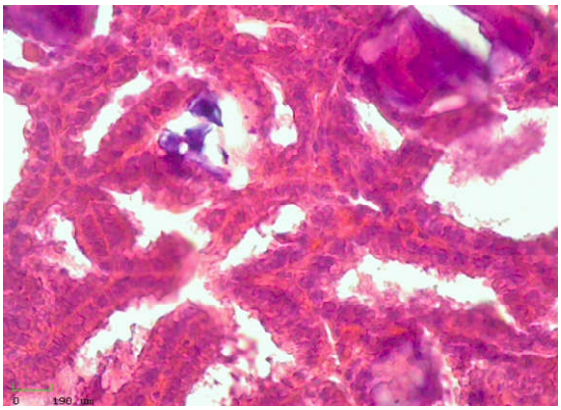

Fig 10: Section of mammary gland of bovine showing aplastic epithelium cell with swelling of hemorrhagic area. X40

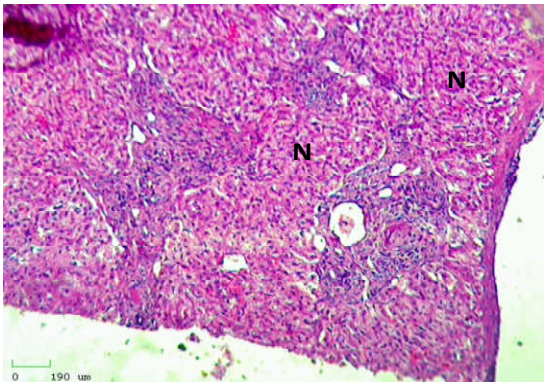

Fig 11: Section of mammary gland of bovine showing necrotic area(N) with closure of acini. X4

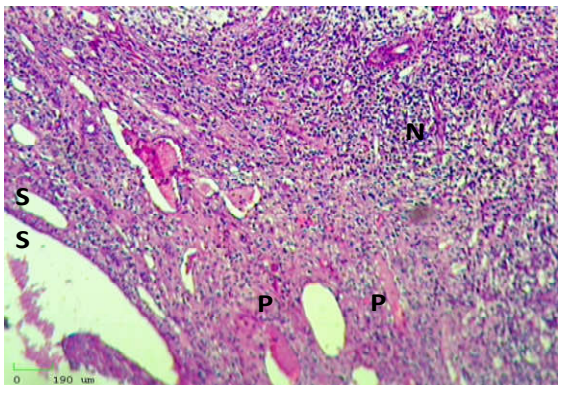

Fig 12: Section of mammary gland of bovine showing necrotic area of tissue (N) with accumulation pretentious materials (P) and the acini are different in size(S).X10

DISCUSSION

Histopathological examination of the acute, chronic, and necrotic mastitis cases is in agreement with previous studies [10, 11, 12]. During acute mastitis, it has been found that tissue changes might be due to endotoxic injury to the microvasculature of the alveolar walls and mammary interstitium, stimulating huge neutrophils emigration to clean the gland of organisms within few days [13]. The acute reaction develops as the initial inflammatory response to clear the tissue of mammary gland from all organisms. If the acute reaction fails to complete clear the tissue of mammary gland from all pathogens, chronic reaction will develop. The major chronic lesion found in the current study was the fibrosis.

In chronic mastitis, It has been found that fibrosis was due to the response to chemotactic factors released either from cells damaged by organism or from the organism itself [14]. A bovine mammary adenocarcinoma was due to inflammatory infiltrate composed of lymphocytes and plasma cells that has glandular characteristics [15]. The hyperplastic activity in chronic cases is formed by the tissue reaction. It has been found that the hyperplastic activity could be repair of the damaged epithelial lining [16].

In necrosis mastitis, generation of leukocidin or haemolysin from pathogens might be cytotoxic and promoting tissue necrosis [16, 17]. In conclusion, nine bovine udders were studied microscopically. The inflammation were classified into acute, chronic, and necrosis.

REFERENCES

1. Wellenberg GJ, Van Der Poel WHM, Van Oirschot JT 2002. Viral infections and bovine mastitis: A review. Vet Microbiol. 88(1):27-45.

2. Muhee A, Malik HU, Asharaf I, Shah OS, Jan A, Muheet I, Rather W ,and Muzamil S.2017. Biochemical and Mineral Alterations of Milk Chemistry in Mastitis. In tJCurr Microbiol AppSci. 6(7):4591-4594.

3. Badiuzzaman M, Samad MA, Siddiki SHMF, Islam MT, Saha S 2016. Subclinical mastitis in lactating cows: comparison of four screening tests and effect of animal factors on its occurrence. Bangladesh J Vet Med. 13(2):41-50.

4. Malek dos Reis CB, Barreiro JR, Mestieri L, Porcionato MA de F, dos Santos MV 2013. Effect of somatic cell count and mastitis pathogens on milk composition in Gyr cows. BMC Vet Res. 9.

5.Hogeveen H, Huijps K, Lam TJGM 2011. Economic aspects of mastitis: New developments. N Z Vet J. 59(1):16-23.

6. Petzl W, Günther J, Mühlbauer K, Seyfert H, Schuberth H, Hussen J, Sauter-Louis, C, Hafner-Marx A. and Zerbe H. 2016. Early transcriptional events in the udder and teat after intra-mammary Escherichia coli and Staphylococcus aureus challenge. Innate Immun.22(4):294-304.

7. Zhao, X. and Lacassem, I. P. 2008: "Mammary tissue damage during bovine mastitis: Causes and control." J. Anim. Sci.; 86: 57-65.

8. Mandefrot Meaza Zeleke 2016. morphometrical, pathological and bacteriological study of mammary gland of cows with subclinical and clinical mastitis in selected farms and abattoirs in central ethiopia.

9.https://en.wikivet.net/Mammary Gland Anatomy %26 Physiology

10. Shibahara, T. and Nakamura, K. 1999: "Pathology of Acute Necrotizing Mastitis Caused by Staphylococcus aureus in aDairy Cow." JapanInternational Research Center forAgricultural Sciences, April19.

11. Deriabin, D. G. and Kurlaev, P. P. 2000: "The role of staphylococciin the occurrence, developmentand chronicity oflactation mastitis."ZhMikrobiolEpidemiol Immunobiol ;2:118-21.

12. Nieberle, K. and Cohrs. P.1931: Lehrbuch der speziellen oa thologischen Anatomi c der Hall . G ustave Fischer. Jcna: 578-588

13. Hogan, J. and Larry S. K. (2003): "Coliform mastitis."Vet Res.; 34: 507-519.

14. McGavin, M. D.; Carlton, W. W. and Zachary, J.F. 2001: "Thomson's Special Veterinary Pathology." 3rd ed, A Hsrcourt Health Sciences Company SL Louis London Philadelphia Sydney Toronto.

15. Kristi, L. and Dosher, D.V.M (2009). Small Animal Critical Care Medicine.

16. Quinn, P.J., Markey, B.K., Carter,M. E., Donnelly, W.J. and Leonard, S.C. 2002: "Veterinary Microbiology and Microbial diseases." 1st Publishing, A Blackwell Science Comp.

17. Winter, P.; Schilcher, F.; Fuchs, K. and Colditz, I. G. 2003:"Dynamics of experimentally induced Staphylococcus epidermidis mastitis in East Friesian milk ewes." J. Dairy Res.; 70: 157-164.