The Influence of Ketosis as a Risk Factor on Mastitis Occurrence during Early Lactation Period in Dairy Cows

Dejan Janevski[1], Miodrag Radinović[1], Jovana Krivokapić[1], Jovan Stanojević[1], Petar Dodovski[2], Vladimir Stojanoski[2], Tijana Gichova[2]

[1]Department of Veterinary Medicine, Faculty of Agriculture in Novi Sad. R Serbia, Trg Dositeja Obradoviča 8, 21101 Novi Sad,Serbia

[2]Veterinary Faculty Bitola, University "St. Clement Ohridski" - Bitola, Bitola, Republic of North Macedonia, Prilepska bb. 7000 Bitola

Keywords: *bacteria, cows, ketosis, milk, Staphylococcus aureus.*

Abstract. *The study aimed to assess the impact of ketosis in cows during early lactation, immediately postpartum, on the development of mastitis as a secondary disease and its potential role as a risk factor for recurrent mastitis. This was achieved by monitoring affected udders throughout one lactation period. The research involved N = 156 Holstein Friesian and Simmental cows, divided into three groups of N = 52: the first group included cows with primary postpartum ketosis and secondary mastitis, the second group consisted of cows with mastitis but no ketosis, and the third served as a healthy control group. Ketosis was diagnosed through laboratory analysis of blood, milk, and urine samples for the presence of ketone bodies. Mastitis detection involved clinical evaluation of the udder and microbiological identification of causative pathogens from milk samples. Cows in the first group were monitored throughout lactation to determine the prevalence of recurrent mastitis and identify key risk factors contributing to its recurrence.*

The findings revealed that recurrent mastitis was diagnosed in 24 cows across both mastitis-affected groups, with Staphylococcus aureus identified as the primary pathogen responsible for recurrence in 87.5% of cases. Additionally, a statistically significant difference in milk yield was observed between the control group and the mastitis-affected groups ($P < 0.05$). These results suggest that metabolic disorders may contribute to the recurrence of mastitis caused by common pathogens and that mastitis has a significant impact on milk yield in dairy cows.

Introduction

The peripartum period and early lactation are marked by significant physiological adaptations that enable the body to adjust to its new state. These adaptations involve endocrine and metabolic changes, primarily driven by the increased energy demands in late pregnancy and the initial stages of lactation. A key metabolic alteration during this period is an energy deficit, resulting from heightened glucose utilization and decreased blood glucose levels. Concurrently, fat mobilization occurs through the release of unsaturated fatty acids to compensate for the increased energy requirement (Bernabucci et al., 2005; Sumathi et al., 2008). Excessive fat mobilization, coupled with the liver's limited capacity for catabolism, leads to an accumulation of acetyl coenzyme A (CoA) and ketone bodies. When these metabolic adjustments surpass the body's ability to utilize acetyl CoA and ketone bodies as energy sources, their concentration increases, resulting in ketosis. This condition is characterized by hyperketonemia, ketonuria, and the presence of ketone bodies in milk and exhaled air. Additionally, ketosis is associated with hypoglycemia and a reduction in hepatic glycogen stores. The interplay between negative energy balance, ketosis, and immunosuppression predisposes animals to secondary diseases linked to ketosis (Oetzel, 2007; Seifi et al., 2011; Ayano et al., 2013).

Mastitis is among the most prevalent conditions arising from metabolic disturbances during the peripartum period, particularly in early lactation. It is an inflammatory condition of the mammary gland, commonly caused by pathogenic microorganisms that enter through the teat canal and proliferate in the glandular tissue (Vikova et al., 2017). Recurrence of mastitis is particularly significant, as evidence suggests that negative energy balance and hypoglycemia during early lactation compromise both systemic and local immune defenses in the mammary gland. This impairment contributes to disrupted lactogenesis and a decline in milk production (Ahmadzadeh et al., 2009; Frey, 2013; Ruegg, 2017; Jamali et al., 2018).

The primary objective of this research is to identify the key risk factors associated with recurrent mastitis and to examine the impact of ketosis, as a metabolic disorder occurring post-parturition and during early lactation, on both the incidence of mastitis and overall milk yield in the studied cows.

Materials and methods

For the purpose of this scientific study, three groups of fifty-two cows (N = 52) were examined

Correspondence to: Dejan Janevski,
e-mail: dejanjanevskinovisad@gmail.com
tel.: +389 077 711 862

separately, all originating from the same farm (totaling 156 cows). These cows were raised under uniform hygienic conditions, maintained in a free-stall housing system, and fed according to standard nutritional guidelines for high-yield dairy cows in full lactation within an intensive milk production system. The study population consisted of high-producing Holstein Friesian (HF) and Simmental (S) breeds. The first group (N = 52; N = 31 HF, N = 21 S) comprised cows diagnosed with ketosis following parturition and during early lactation. Within this group, cases of both primary and recurrent mastitis were identified throughout the lactation period. Data on milk yield and its variations were recorded over the entire lactation cycle. The second group (N = 52; N = 31 HF, N = 21 S) consisted of cows that did not experience metabolic disorders post-calving or during early lactation but developed primary and recurrent mastitis at different stages of lactation. For this group, milk yield data and fluctuations during mastitis episodes were collected. The third group served as a control group and included healthy cows (N = 52; N = 31 HF, N = 21 S) that remained free from both metabolic disorders and mastitis throughout the lactation period. For these cows, daily milk yield data were recorded across the entire lactation. To diagnose ketosis, blood, milk, and urine samples were collected from all cows (N = 156) between the third and the twentieth day postpartum, with the sixth day post-calving identified as the most appropriate period for diagnosis due to the peak concentration of ketone bodies. Blood samples were analyzed for ketosis using an enzymatic catalysis method with a biochemical analyzer to measure beta-hydroxybutyric acid (BHB) concentration in serum. Blood was collected via puncture of the coccygeal vein (v. Coccygea), with subclinical ketosis defined as $\geqslant$ 1.2 mmol/L BHB and clinical ketosis as $\geqslant$ 2.8 mmol/L BHB (Seifi et al., 2011). Ketosis in milk was detected using the Rother-Ross test, which identifies the presence of acetone and acetoacetate by adding an alkaline sodium nitroprusside solution to milk. A positive reaction results in a color change to red or violet, allowing the detection of acetoacetic acid (1–5 mg/dL) and acetone (10–20 mg/dL) (Carrier et al., 2004; Seifi et al., 2011; Serenho et al., 2022). Ketosis in urine was confirmed using Lestradet's test, which detects ketone bodies (acetone and acetoacetate) through the addition of sodium nitroprusside, leading to a color change to red or purple in a positive reaction. This method can detect acetoacetic acid concentrations of 1–5 mg/dL and acetone concentrations of 10–20 mg/dL (Carrier et al., 2004; Serenho et al., 2022). Mastitis diagnosis and mammary gland health assessment in cows previously diagnosed with ketosis were conducted through standard clinical examination methods, including evaluation of visible changes in milk consistency. Cows displaying abnormal milk secretion without visible signs of mammary gland inflammation were further screened using the California Mastitis Test (CMT), which involved mixing 3 mL of milk with 3 mL of CMT reagent (containing alkyl sulfonate as the active component). The test results were determined based on gel viscosity and the color change of the indicator (Bartlett et al., 2001; Karimuribo et al., 2006; Heikkilä et al., 2012). For cows with a positive CMT reaction, sterile milk samples were collected for microbiological analysis. The laboratory procedure for isolating and identifying pathogenic microorganisms involved serial dilution in physiological saline (0.85% NaCl) followed by inoculation onto a solid nutrient medium. The samples were incubated at 37°C for 24 hours (Gezhagn et al., 2020; Kasravi et al., 2010; Bradley et al., 2005). Morphological characteristics of the resulting bacterial colonies (color, size, shape) were analyzed, alongside physiological properties such as enzymatic activity on proteins and carbohydrates and biochemical characteristics, including enzyme expression and pathogen susceptibility to specific substances. Cows diagnosed with additional health disorders during lactation, aside from ketosis as the primary condition and mastitis as the secondary condition (including mastitis recurrences), were excluded from further research. Ethical approval for this study was obtained from an ethics committee, which authorized sample collection and animal handling (ethics approval number: 001328298 2024 14841 002 001 323 022).

Statistical analysis of the collected data was performed using SPSS software version 19.0 for Microsoft Windows. Descriptive and inferential statistical methods were applied, including analysis of variance (ANOVA) with post-hoc Tukey HSD tests, as well as t-tests for comparing mean milk yields between breeds.

Results

Fig. 1 illustrates that 50% of the cows diagnosed with metabolic ketosis, equivalent to 26 of a total of 52 cows, also developed mastitis.

Based on the presentation of Fig. 2, it can be seen that of 26 cows with primary mastitis, in 50% or 13 cows, the pathogenic agent of mastitis was *Staphylococcus aureus*, in 15% or 4 cows, the pathogenic agent was *Streptococcus agalactiae*, in 11% or 3 cows, the pathogenic agent was *Streptococcus uberis*, while the pathogenic agents *Pseudomonas aeroginosa, Escherichia coli, Streptococcus dysgalactiae, Klebsiella* spp., *Streptococcus pyogenes* and *Enterobacter* spp. each separately caused mastitis in one cow and each represented 4% of the total number of primary mastitis.

From Fig. 3, it can be seen that of a total of 14 cows with recurrent mastitis, in 93% or 13 cows with recurrent mastitis, the pathological cause of mastitis was *Staphylococcus aureus*, while in 7% or one recurrent mastitis, the pathological cause was *Streptococcus agalactiae*.

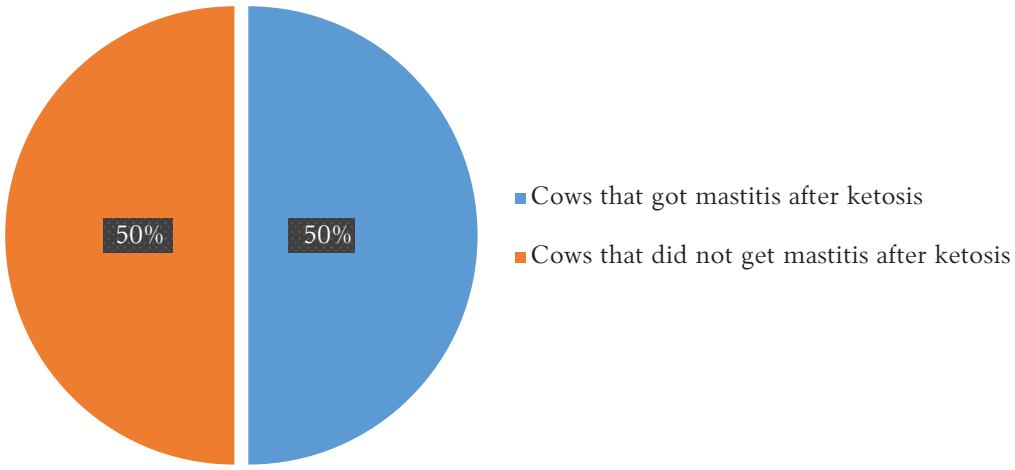

Fig. 1. Percentage of cows diagnosed with mastitis and cows with no mastitis after ketosis (%)

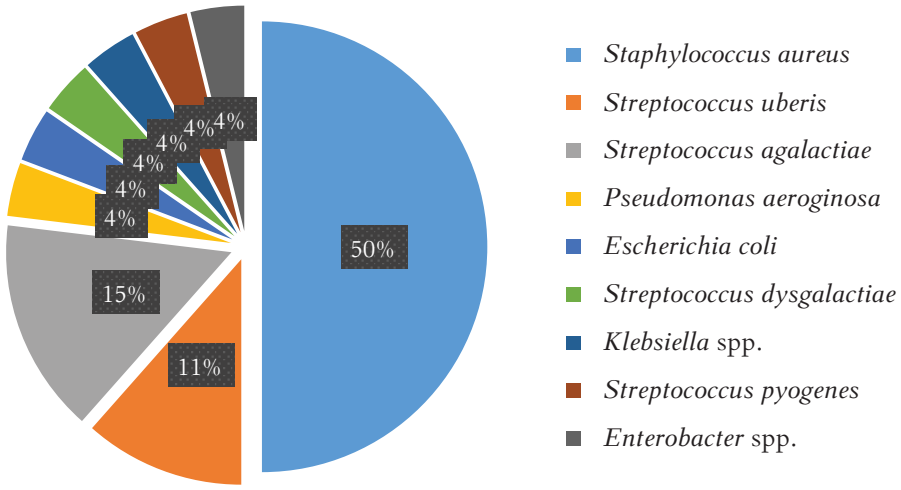

Fig. 2. Pathogenic agents of mastitis in cows that got mastitis after ketosis (%)

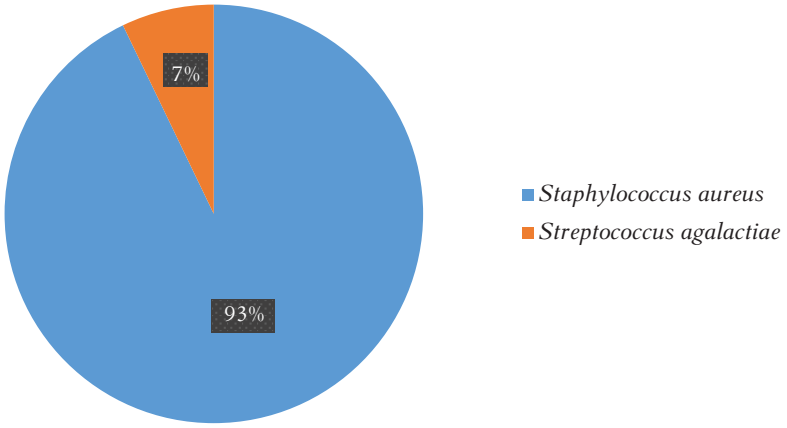

Fig. 3. Pathogenic agents of recurrent mastitis (%)

Fig. 4 indicates that among the 52 cows diagnosed with mastitis without a prior history of metabolic disorders, *Staphylococcus aureus* was identified as the causative agent in 56% of cases (29 cows). *Streptococcus uberis* was responsible for 13% of cases (7 cows), while *Streptococcus agalactiae* accounted for 11% (6 cows). Additionally, *Pseudomonas aeruginosa* was detected in 8% of cases (4 cows), and *Escherichia coli* was identified in 6% (3 cows). The remaining 2% of cases (one cow each) were attributed to *Streptococcus dysgalactiae*, *Klebsiella* spp.., and *Streptococcus pyogenes*.

In Fig. 5, it can be seen that of 10 cows with recurrent mastitis, in 80% or 8 cows, the main factor for mastitis was the bacterium *Staphylococcus aureus*, in 10% or in one cow, the bacterium isolated was *Streptococcus uberis*, and in 10% or in one cow, the

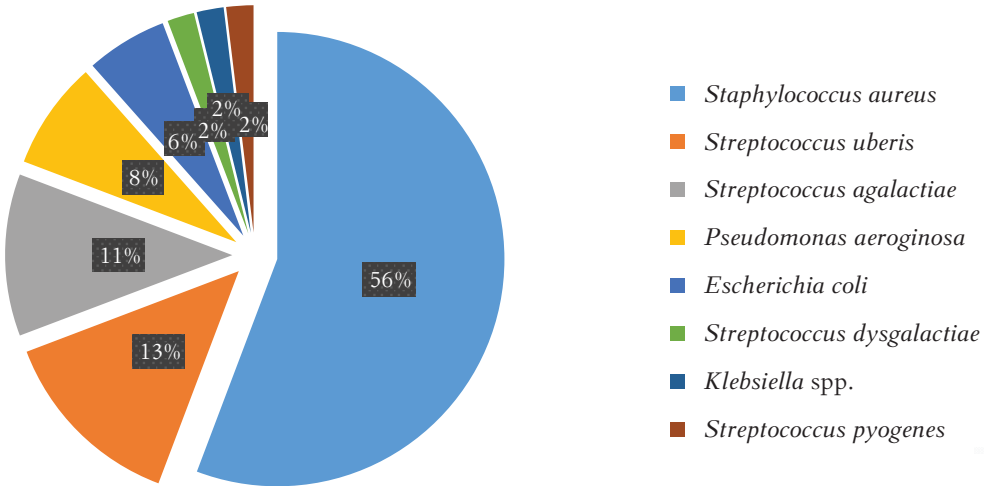

Fig. 4 Pathogenic agents of primary mastitis in cows without metabolic disease (%)

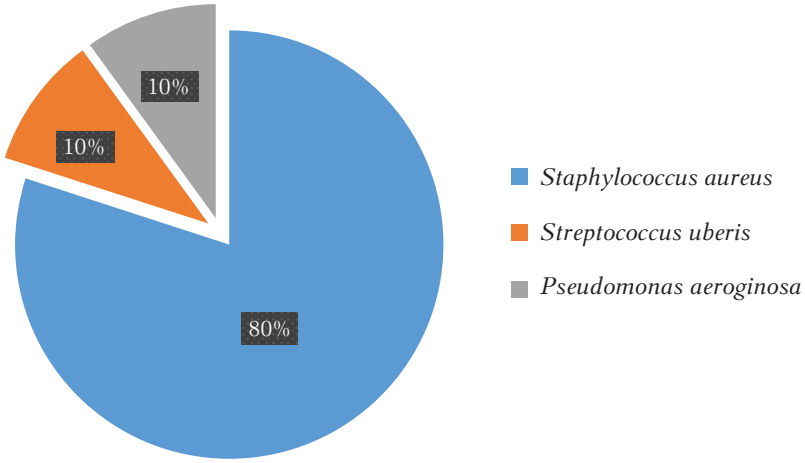

Fig. 5. Pathogenic agents of recurrent mastitis

cause was the bacterium *Pseudomonas aeruginosa*.

Table number 1 shows the results of the descriptive statistics for milk yield in the three groups of cows, where it is noted that the control group had the highest average milk yield (29.29 L/day), while the average milk yield in the group with mastitis and metabolic disease was 23.89 L/day and the average milk yield in the group with mastitis without metabolic diseases was 22.89 L/day.

From the values of standard deviations and coefficients of variation, it can be observed that the highest variability of milk yield was in the group that had mastitis and metabolic diseases (27.75%), and the lowest (17.92%) was in the control group of cows.

The results of the one-way analysis of variance revealed statistically significant differences ($P \leqslant 0.05$) in milk yield among the three observed groups of cows. To identify specific groups with significant differences in milk yield, a post hoc Tukey HSD test was conducted (Table 3).

The findings indicate a statistically significant difference in average milk yield between the control group and the group affected by both mastitis and metabolic disorders ($P \leqslant 0.05$), as well as between the control group and the mastitis-only group ($P \leqslant 0.05$). However, no statistically significant difference was observed in the average daily milk yield between the mastitis-only group and the group with both mastitis and metabolic disorders ($P > 0.05$).

According to the results presented in Table 4, the control group, which remained free of disease, exhibited the highest average milk yield on the final day of measurement, reaching 29.21 liters per day. In comparison, the group with both mastitis and metabolic disorders demonstrated a slightly lower average yield of 25.15 liters per day, while the mastitis-only group had the lowest average yield at 19.38 liters per day.

The lowest recorded milk yield on the final day was 6 liters, observed in a cow from the mastitis and metabolic disorder group, whereas the highest yield within the same group reached 41 liters. When examining standard deviations and coefficients of variation, the greatest variability in milk yield on the last measurement day was observed in the group affected by both mastitis and metabolic disorders (20.61%), while the lowest variability (14.31%) was recorded in the mastitis-only group.

Table 1. Descriptive statistics of the average milk yield of the studied cows

Group of cows	%	Min	Max	Standard deviation	Coefficient of variation (%)
Control group	29.29	18.00	47.00	5.25	17.92
Cows with mastitis and metabolic	23.89	1.00	45.00	6.63	27.75
Cows with mastitis	22.89	6.00	43.00	5.88	25.68

Table 2. ANOVA test for the average daily milk yield in the studied groups of cows

Effect	SS	df	MS	F	*P* value
Intercept	100 297.3	1	100 297.3	5952.691	0.000
Tretman	1231.0	1	615.5	36.532	0.000

Table 3. Results of the post-hoc Tukey HSD test for the average milk yield of the studied groups of cows

Treatment	Control group of cows	Cows with mastitis and metabolic disturbances (ketosis)	Cows with mastitis
Control group of cows		0.000	0.000
Cows with mastitis and metabolic disturbances (ketosis)	0.000		0.424
Cows with mastitis	0.000	0.424	

Table 4. Descriptive statistics of daily milk yield in the studied cows on the last day of measurement

Group of cows	Average	Min	Max	Standard deviation	Coefficient of variation (%)
Control group of cows	29.21	21.00	40.00	4.47	15.31
Cows with mastitis and metabolic disturbances (ketosis)	25.15	6.00	41.00	5.18	20.61
Cows with mastitis	19.38	11.00	24.00	2.77	14.31

Table 5. ANOVA test for average daily milk yield in the studied groups of cows

Effect	SS	df	MS	F	*P* value
Intercept	94 277.08	1	94 277.08	5181.641	0.000
Treatment	2536.17	1	1268.08	693 696	0.000

Table 6. Results of the post-hoc Tukey HSD test for the average milk yield of the studied groups of cows

Treatment	Control group of cows	Cows with mastitis and metabolic disturbances (ketosis)	Cows with mastitis
Control group of cows		0.000	0.000
Cows with mastitis and metabolic disturbances (ketosis)	0.000		0.000
Cows with mastitis	0.000	0.000	

The results of the one-factor analysis of variance showed the existence of statistically significant differences ($P \leqslant 0.05$) in milk yield on the last day of measurement in the observed three groups of cows. To determine exactly which cows had a statistically significant difference in milk yield on the last day, the post-hoc Tukey HSD test was applied, as in the previous case (Table 6).

Based on the results of the Tukey HSD test, it can be noted that there is a statistically significant difference in milk yield on the last day of measurement between all three groups of cows ($P < 0.05$).

Discussion

The statistical analysis of the research findings confirmed the thesis proposed by Fleischer et al.

(2001), which suggests an intrinsic correlation between ketosis and mastitis. This relationship was also validated in the present study, as 50% (26 of 52) of the cows diagnosed with ketosis subsequently developed mastitis at a certain stage of lactation. Mastitis was attributed to various bacterial strains, with *Staphylococcus aureus* being the predominant causative agent in both mastitis-affected groups. Specifically, *Staphylococcus aureus* accounted for 50% of cases in the first group and 56% in the second group, aligning with the findings of Saidi (2013), where *Staphylococcus aureus* comprised 40% of the total isolated mastitis pathogens. Similarly, Abrahamsen (2013) reported that *staphylococci* were responsible for 54.7% of mastitis cases.

Regarding recurrent mastitis (N = 24), *Staphylococcus aureus* was identified as the causative agent in 87.5% (N = 21) of cases, leading to the conclusion that this pathogen represents a primary risk factor for recurrent mastitis. These findings are consistent with studies by Bradley et al. (2007), Roesch et al. (2007), and Kalmus et al. (2011), which identified *staphylococci* and *streptococci* as the most frequently isolated pathogens in subclinical mastitis. Additionally, research by Dingwell et al. (2003) highlighted *Staphylococcus aureus*, *Streptococcus* spp., and *Escherichia coli* as the predominant pathogens during early lactation.

The impact of ketosis and mastitis on milk yield, as demonstrated through statistical analysis, closely aligns with the research of Dohoo and Martin (1983), who documented a reduction in milk yield ranging from 1 to 1.4 liters per day. In the present study, similar findings were observed concerning standard deviation values and coefficients of variation. The highest variability in milk yield (27.75%) was observed in the group affected by both mastitis and metabolic disorders, while the lowest variability (17.92%) was recorded in the control group. The group with mastitis but no metabolic disease exhibited a milk yield variability of 25.68%. These findings further support the mild influence of ketosis on milk production, as indicated by Oetzel (2015) and Dohoo and Martin (1983), while also confirming the significant impact of mastitis on milk yield variability.

The one-way analysis of variance revealed statistically significant differences ($P \leqslant 0.05$) in milk yield among the three observed groups. The application of the Tukey HSD test confirmed a statistically significant difference in average milk yield between the control group and the group with both mastitis and metabolic disorders ($P \leqslant 0.05$), as well as between the control group and the mastitis-only group ($P \leqslant 0.05$). However, no statistically significant difference was detected in average daily milk yield between the mastitis-only group and the group with both mastitis and metabolic disorders ($P > 0.05$), partially aligning with the findings of Oetzel (2015) and Dohoo and Martin (1983), who reported a minor impact of ketosis and a significant influence of mastitis on milk yield.

Based on the final measurement of average milk yield, the lowest production was recorded in the mastitis-only group, with an average of 19.38 liters per day. This suggests that mastitis, when not preceded by metabolic disorders, has the most pronounced effect on the physiological condition of the mammary gland and milk yield, consistent with the findings of Sanford et al. (2006). Conversely, the group affected by both ketosis and mastitis had a higher average milk yield of 25.15 liters per day. This result indicates that milk production in this group may be attributed to the correlation between mastitis and ketosis-induced immunosuppression, leading to a higher incidence of mastitis in the postpartum period, as described by Spears and Weiss (2008) and Sordillo and Aitken (2009).

Lastly, the one-way analysis of variance and Tukey HSD test confirmed statistically significant differences ($P \leqslant 0.05$) in milk yield among all three groups of cows on the final day of measurement.

Conclusion

The findings of this study confirm a significant correlation between ketosis and mastitis, supporting the hypothesis proposed by Fleischer et al. (2001). The results indicate that ketosis is a predisposing factor for the onset of mastitis, with *Staphylococcus aureus* emerging as the predominant pathogenic agent in both mastitis-affected groups. This aligns with previous research, reinforcing the role of *Staphylococcus aureus* as a major contributor to both primary and recurrent mastitis.

Furthermore, the study highlights the impact of ketosis and mastitis on milk yield, demonstrating that mastitis exerts a stronger influence on milk production variability compared with ketosis. The statistical analysis revealed significant differences in milk yield among the three groups, with the lowest production recorded in cows with mastitis alone. These findings suggest that mastitis, particularly in the absence of prior metabolic disorders, has severe consequences for the physiological condition of the mammary gland. The observed lowering of milk yield in cows affected by both ketosis and mastitis compared with the control group suggests that immunosuppressive effects associated with ketosis may contribute to a higher incidence of mastitis, particularly in the postpartum period. This aligns with previous studies on the relationship between metabolic disorders and immune function. Overall, this study provides valuable insights into the interplay between ketosis, mastitis, and milk yield, emphasizing the need for effective management strategies to mitigate the economic and physiological impacts of these conditions. Future research should further investigate the underlying mechanisms linking metabolic diseases with infectious conditions to develop targeted prevention and treatment strategies.

References

1. Abrahmsén M., Persson Y., Kanyima B.M., Båge R. Prevalence of subclinical mastitis in dairy farms in urban and periurban areas of Kampala, Uganda. Trop Anim Health Prod. 2013. T. 46. P. 99-105.
2. Ahmadzadeh A., Frago F., Shafii B., Dalton JC., Price W.J., McGuire MA. Effect of clinical mastitis and other diseases on reproductive performance of Holstein cows. Animal Reproduction Science 2009. T. 112 P. 273-282.
3. Almaw G., Zerihun A., Asfaw Y. Bovine mastitis and its association with selected risk factors in smallholder dairy farms in and around Bahir Dar, Ethiopia. Trop Anim Health Prod. 2008. T. 40. P.427–432.
4. Ayano A.A, Hiriko F., Simyalew A.M., Yohannes, A. Prevalence of subclinical mastitis in lactating cows in selected commercial dairy farms of Holeta district. Journal of Veterinary Medicine and Animal Health 2013. T. 5. P. 67-72.
5. Bartlett P.C., Agger J.F., Houe H., Lawson L.G. Incidence of clinical mastitis in Danish dairy cattle and screening for nonreporting in a passively collected national surveillance system. Prev. Vet. Med. T. 2001. 48. P. 73–83.
6. Bernabucci U., Ronchi B., Lacetera N., Nardone A. Influence of body condition score on relationships between metabolic status and oxidative stress in periparturient dairy cows. J. Dairy Sci. T. 2005. 88. P. 2017-2026.
7. Bradley A.J., Newton H., Green M.J. Use and interpretation of bacteriology in the diagnosis of bovine intramammary infection. In: Proceedings of the 4th IDF International Mastitis Conference. 2005. T. 83. P. 409-415.
8. Carrier J., Steward S., Godden S., Fetrow J., Rapnicki, P. Evaluation and use of three cowside tests for detection of subclinical ketosis in early postpartum cows. Journal of Dairy Science. 2004. T.87. P. 3725-3735.
9. Dingwell RT, Kelton DF, Leslie KE. Management of the dry cow in control of peripartum disease and mastitis. Veterinary Clinics: Food Animal Practice. 2003 Mar 1;19(1):235-65.
10. Dohoo IR, Martin SW. Subclinical ketosis: prevalence and associations with production and disease. Canadian Journal of Comparative Medicine. 1984 Jan;48(1):1.
11. Fleischer P., Metzner M., Beyerbach M., Hoedemaker M., Klee W.The relationship between milk yield and the incidence of some diseases in dairy cows, J Dairy Sci. 2001. T. 84. P. 2025-2035.
12. Frey Y., Rodriguez J.P, Thomann A., Schwendener S., Perreten V. Genetic characterization of antimicrobial resistance in coagulase-negative staphylococci from bovine mastitis milk, Journal of Dairy Science. 2013. T. 96. P. 2247-257.
13. Gezehagn K., Betelihem T., Belege T. Isolation and Identification of Major Pathogenic Bacteria from Clinical Mastitic Cows in Asella Town, Ethiopia. 2020 T.20. P.1-6.
14. Heikkila, A.M., Nousiainen, J.I., Pyorala, S. Costs of clinical mastitis with special reference to premature culling. J Dairy Sci. 2012 T. 99. P 8365-8374.
15. Jamali H., Herman W.B., Jacques M., Lavalle – Bourget E.M., Maloun F., Saini V., Dufour S. Invited review: Incidence, risk factors, and efects of clinical mastitis recurrence in dairy cows. J. Dairy SCI. 2018. T.101.
16. Kalmus P., Aasmäe B., Kärssin A., Orro T., Kask K. Udder pathogens and their resistance to antimicrobial agents in dairy cows in Estonia. Acta Veterinaria Scandinavica. 2011 Dec; 53:1-7.
17. Karimuribo E.D,, Fitzpatrick J.L., Bell C.E., Swai E.S., Kambarage D.M., Ogden N.H. Clinical and subclinical mastitis in smallholder dairy farms in Tanzania: risk, intervention and knowledge transfer. Prev Vet Med. 2006. T. 74. P. 84 –98.
18. Kasravi R., Bolourchi M., Farzaneh N., Seifi H. A., Barin A., Hovareshti P., Gharagozlou F Relationship between in vitro antimicrobial sensitivity of bovine subclinical mastitis isolates and treatment outcome in lactating dairy cows, Iranian Journal of Veterinary Research, Shiraz University. 2010 T. 11. P. 249 – 253.
19. Madeleine F., Serge M., Julie P., Robert H. Identification of catalase-negative, non-beta-hemolytic, gram-positive cocci isolated from milk samples,J Clin Microbiol, 2003.T.41. P. 106-109.
20. Oetzel G.R., Herd-level ketosis-diagnosis and risk factors. Zbornik radova. 40th Annual Conference, American Assocation of bovine practitioners. Vancouver, Canada, 2007. P. 67-91.
21. Oetzel G.R., Uderstanding the impact of subclinical ketosis. Department of Medical Sciences, University of Wisconsin, Madison 2015. T.1. P.15-25.
22. Riekerink R.G.M., Barkema H.W., Kelton D.F., Scholl D.T. Incidence rate of clinical mastitis on Canadian dairy farms. J Dairy Sci. 2008. T. 91. P.66 –77.
23. Roesch M., Doherr M.G., Schären W., Schällibaum M., Blum J.W. Subclinical mastitis in dairy cows in Swiss organic and conventional production systems. Journal of Dairy Research. 2007 Feb;74(1):86-92.
24. Ruegg P.L., Mastitis detection, management, and prevention 100-Year Review: J. Dairy Sci. 2017. T. 100. P. 10381-10397.
25. Sanford C.J., Keefe G.P., Sanchez J., Dingwell R.T., Barkema H.W., Leslie K.E., Dohoo I.R. Test characteristics from latent-class models of the California Mastitis Test. Preventive Veterinary Medicine. 2006 Nov 17;77(1-2):96-108.
26. Seifi H., LeBlanc S., Leslie K., Duffield T. Metabolic predictors of post-partum disease and culling risk in dairy cattle. 2011.T. 188. P. 216-20.
27. Serenho R.C., Williamson M., Berke O., LeBlanc S.J., DeVries T.J., McBride B.W., Duffiel TF An investigation of blood, milk, and urine test patterns for the diagnosis of ketosis in dairy cows in early lactation. 2022. T.105. P. 7719-7727.
28. Spears J.W., Weiss, W. P. Role of antioxidants and trace elements in health and immunity of transition dairy cows. 2008. The Veterinary Journal, 176(1), 70-76.
29. Sumathi B.R., Veeregowda B.M., Amitha R.G. Prevalence and antibiogram profile of bacterial Isolates from clinical bovine mastitis Department of Veterinary Microbiology, Karnataka Veterinary, Animal and Fisheries Sciences University, Hebbal, Bangalore. 2008. T. 1. P. 237-238.
30. Vikova H., Babak V., Vrtkova I., Cervinkova D., Marosevic D., Moravkova M., Jaglic Z. Epidemiology of intramammary infections with Staphylococcus aureus and mastitis streptococci in a dairy cattle herd with a history of recurrent clinical mastitis. Polish Journal of Veterinary Sciences. 2017. T. 20. P.133–139.