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Serum and Milk Procalcitonin levels as Early Diagnostic Indicators for Critical, Subclinical, and Clinical Mastitis in Egyptian Nubian Goats

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Abstract

CAPRINE MASTITIS is a common and costly disease in dairy goats, especially in Egypt, where ≈ 4.2 million goats are raised. Early detection is critical as the subclinical form is the most common. Procalcitonin is a sepsis-associated prohormone that rises in bacterial infections. It is unfortunately, goats haven't been extensively investigated. In this study, we measured Procalcitonin in both serum and milk along with somatic cell count (SCC) and calcium in fifty adult female lactating Egyptian Nubian goats, with an average body weight of (45± 5) kg and an age range of 6–7 years which were grouped into four: control (n=8), critical (n=5), subclinical (n=8), and clinical mastitis (n=29). Our results indicated an increase in vital signs such as temperature, pulse, and respiratory rate in the mastitis group. In addition to the mean corpuscular volume, white blood cell counts, monocytes, and eosinophils were elevated in the clinical group, and monocytes in the subclinical group. There was a decrease in red blood cell, and packed cell volume (PCV) in the subclinical group and in the RBC, hemoglobin, PCV, and platelet in the clinical group. The critical group showed a significant drop in the platelet count. Results showed a stepwise rise in both serum and milk procalcitonin with increasing mastitis severity, while SCC is highly elevated in the clinical cases. We conclude that measuring Procalcitonin levels in serum or milk may contribute in the early detection of critical and subclinical mastitis and aid in the prognosis of severe mastitis in Nubian goats.

Keywords: Mastitis, Procalcitonin, SCC, Nubian goats, Serum, Milk.

Introduction

Mastitis is the most common infection of the goat mammary gland and a major welfare and production issue worldwide [1]. Clinical cases occur in a minority of goats (<5% annual incidence) [2], whereas subclinical infections are far more prevalent [2, 3]. For example, in Egyptian dairy goats, clinical mastitis occurred in 33.7% of animals, while 52.6% of "apparently healthy" udder-halves had SCC ≥106 cells/mL (indicative of subclinical mastitis) [4]. These 8 numbers are variable and were found to be 5% clinical incidence versus ~30% subclinical in Egyptian herds in different sources [5]. These high rates reflect global trends: pooled prevalence studies estimate subclinical goat mastitis in the range of 30–50%, far exceeding clinical cases [3].

Etiologically, *Staphylococcus species* dominate caprine mastitis. Reviews note that S. aureus is the leading agent of clinical cases, with coagulase-negative staphylococci common in subclinical infections [2]. Coagulase-negative staphylococci were found to be the cause in 58.8% of cases and S. aureus in 24.4% of isolates from Egyptian mastitic goat milk [4]. Other bacteria (streptococci, coliforms,

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etc.) are also involved but less frequently in goats [1, 5]. These pathogens spread primarily via contaminated milking equipment or environment, and infected does often remain chronically colonized [2].

Standard mastitis screening relies on SCC and the California Mastitis Test (CMT). However, goat milk naturally has higher SCC due to apocrine secretion of epithelial cells [6]. Thresholds vary widely. Some authorities use 106 cells/mL as a benchmark for subclinical mastitis, while action limits of 5×10^5–3×10^6 have been proposed depending on parity [7]. Critically, many studies report that SCC in goats is strongly influenced by non-infectious factors (lactation stage, breed, and season) [7, 8], reducing its diagnostic reliability. SCC is usually not regarded as a proper indicator for intramammary infection in goats, and bacteriology remains the gold standard [4]. Thus, there is a pressing need for better biomarkers to detect early and subclinical mastitis.

Procalcitonin (PCT) is a prohormone of calcitonin that normally circulates at very low levels but rises sharply in bacterial infections and sepsis [9]. In human medicine, the PCT is used to distinguish bacterial from viral infections. In veterinary research, elevated serum PCT has been reported in mastitic cows [10,11], and was regarded as having 100% sensitivity and specificity in distinguishing cows with clinical staphylococcal mastitis from healthy cows [10]. In subclinical bovine mastitis, a serum PCT cutoff of ≈ 90 pg/mL could discriminate infected from healthy cows (AUC≈0.695) [12]. In small ruminants, PCT increases in response to bacterial inflammation in sheep mastitis [9]. Regarding other infections, goats with acute pneumonia had greatly elevated serum PCT compared to healthy controls, with PCT perfectly discriminating disease (AUC=1.0) [13]. These findings suggest PCT could be a sensitive mastitis indicator.

To our knowledge, however, no study has measured PCT in caprine mastitis. We hypothesized that combining PCT (in serum and milk) with SCC would improve early detection of critical, subclinical, and clinical mastitis in goats. Our objective is to assess the potential of measuring PCT level as an early biomarker for detecting of mastitis in dairy goats.

Material and Methods

. All procedures were performed in accordance with established animal welfare guidelines. This study included a total of fifty adult female lactating Egyptian Nubian goats, with an average body weight of (45± 5) kg and an age range of 6–7 years. Each animal underwent a complete physical and clinical examination prior to inclusion. Based on clinical manifestations, somatic cell count (SCC), and complete blood pictures (CBC), the goats were assigned to four experimental groups: healthy control group (n = 8); goats with SCC < 150,000 cfu/ml, critical group (n = 5); goats with SCC between 150,000 - 250,000 cfu/ml, subclinical group (n = 8); goats with SCC between 250,000 - 400,000 cfu/ml, & clinical group (n = 29); goats with SCC ≥ 400,000 cfu/ml.

The animals were obtained from multiple goat farms in El-Fayoum Governorate, Egypt, and from the Faculty of Veterinary Medicine farm at Cairo University, Giza. They were maintained under standard housing conditions, received their daily rations twice, and had access to drinking water. Sampling was conducted between August 10, 2024, and January 12, 2025.

Sampling

Blood sampling

Blood was drawn aseptically from the jugular vein using sterile syringes following proper cleaning and disinfection of the venipuncture site. Each sample was divided into three types of collection tubes: EDTA tubes, heparinized tubes, & plain tubes.

EDTA tubes were used for complete blood count (CBC) analysis on an automated hematology analyzer (Sysmex XT 2000 iV, Japan Veterinary Automatic Cell Counter). Heparinized tubes were centrifuged at 4000 rpm for 10 minutes to obtain plasma, which was stored in Eppendorf tubes at -20°C until analyzed for calcium levels in a veterinary diagnostic laboratory. Plain tubes were centrifuged at 3000 rpm for 10 minutes to separate serum, which was aliquoted into Eppendorf tubes and stored at -80°C for procalcitonin (PCT) assessment using a goat-specific enzyme-linked immunosorbent assay (ELISA) kit (Sunlong, China).

Milk Sampling and Processing

Milk samples were collected aseptically from two udder halves into sterile 15 mL Falcon tubes after thorough cleaning and disinfection of the teats. Each sample was divided into two portions: The first portion (7 mL) was analyzed for somatic cell count (SCC) using an automated counter (BacSomatic, Denmark), & the second portion was centrifuged at 2000–3000 rpm for 20 minutes to remove the cream layer. The skimmed supernatant was collected into sterile Eppendorf tubes and stored at -80°C for PCT quantification using the goat ELISA kit (Sunlong, China).

Statistical analysis

All data were expressed as mean ± standard error (SE). Comparisons between groups were performed using the independent-samples t-test, with statistical significance considered at $P < 0.05$. Data analyses were carried out using SPSS version 27 (IBM, NY, USA).

Results

In cases of clinical mastitis, the predominant findings were both local alterations in the udder and systemic disturbances. Locally, the affected udder exhibited heat, swelling, firmness, marked sensitivity, and enlargement of the retro-mammary lymph nodes. The milk also showed pathological changes, including diminished flow and the presence of blood, clots, or purulent material.

Vital signs in goats

The clinical evaluation of vital signs in the examined goats is presented in Table 1. Goats with clinical mastitis showed a marked increase in rectal temperature, with mean values reaching 40.68 ± 0.11°C, significantly higher than the control group. Similarly, heart rate rose progressively across mastitic groups, peaking in clinical mastitis compared with controls. Respiratory rate followed the same trend, being markedly elevated in clinical goats relative to healthy controls. Critical and subclinical groups showed modest but non-significant increases compared with controls.

Hematological profile in goats

Hematological profiles (Table 2) showed typical changes of infection. Clinical mastitis goats had significantly lower RBC counts, hemoglobin concentrations, packed cell volume (PCV), and platelet count with a significant increase in MCV compared with the control group. In the subclinical group, RBC counts were significantly reduced, and packed cell volume compared with the control goats. In addition, platelet counts were significantly lowered in the critical group when compared with the control group.

Total leukocyte count was significantly elevated in clinical mastitis relative to healthy goats. There was monocytosis in both subclinical and clinical mastitis goats compared with controls. Eosinophil counts also rose in clinical mastitis.

Inflammatory biomarkers in goats

Inflammatory biomarkers in milk and serum are presented in Table 3. Milk somatic cell count (SCC) was markedly elevated in mastitic groups. Healthy goats had a low SCC which significantly increased in critical, and subclinical mastitis goats, while clinical mastitis showed a dramatic rise. Milk procalcitonin (PCT) mirrored the SCC trend where healthy goats showed minimal milk PCT, while critical, subclinical, and clinical groups exhibited significantly higher concentrations.

Serum PCT followed a similar pattern. Control goats had a baseline value which increased significantly in critical, subclinical, and clinical mastitis goats. Notably, serum PCT in clinical mastitis was more than tenfold higher compared with healthy controls. Serum calcium levels did not differ significantly between groups.

Discussion

In this study, goats with severe clinical mastitis exhibited modest elevations in systemic vital signs, whereas subclinical cases showed little to no change. A similar study found mastitic goats had a mean rectal temperature of 104.09°F (~40.1°C), pulse rate 94.2 beats/min, and respiratory rate 27.8 breaths/min [14], similar to our findings of 40.68 ± 0.11°C and 96.87 ± 0.92 bpm in clinical cases. Likewise, another paper experimentally infected goats and observed rectal temperature peaks around 40.2°C in severe mastitis [15]. In contrast, goats with subclinical mastitis in our study showed essentially normal temperatures (39.40 ± 0.22°C) and heart rates (82.10 ± 1.95 bpm), supporting the conventional view that subclinical mastitis often lacks overt fever or systemic signs. Only non-significant increases in temperature and pulse in subclinical cases was noted [14].

We also observed that heart rate and respiratory rate increased progressively with mastitis severity (e.g., from 78.45 ± 2.10 bpm and 15.73 ± 0.64 breaths/min in controls to 96.87 ± 0.92 bpm and 25.34 ± 0.46 breaths/min in clinical goats), although changes were not as pronounced as temperature. In sum, our clinical mastitis group showed mild pyrexia and tachycardia consistent with inflammation, whereas subclinical animals remained largely within normal limits. These patterns are in line with goat-specific studies reporting only mild systemic changes in mastitis unless severe (e.g., gangrenous) infection is present [14, 15]. Thus, vital signs alone are relatively insensitive for detecting subclinical mastitis in goats, but moderate elevations (40.0–40.5°C) may accompany severe clinical cases.

Hematological profiles in our mastitic goats reflected inflammation and lower RBC, Hb and PCV counts in severe cases. In contrast, total leukocyte count was substantially higher in clinical cases than in controls. Differential counts revealed increased neutrophils, and lymphocytes in clinical mastitis goats. Subclinical cases had intermediate values. These findings suggest that severe mastitis elicited acute leukocyte mobilization, whereas changes related to RBCs were within normal range.

Our results align with some goat-specific studies and differ from others, underscoring variable hematologic responses. A study found no significant differences in most hematological parameters between mastitic and healthy Red Sokoto goats [16], differing from our observation that differences in RBC and Hb were statistically significant. By contrast, a detailed Egyptian field study reported significant anemia in mastitic goats. Mean hemoglobin, RBC count, and hematocrit were significantly lower in mastitic goats, while neutrophil

and lymphocyte counts were actually lower (rather than higher) in clinical mastitis. In that study, 70% of goats had clinical mastitis, and they observed that mean values of Hb, RBCs, and WBCs were decreased significantly, with lymphocytes, neutrophils being decreased significantly [17].

This contrasts with our leukocytosis, perhaps because we sampled animals with active infection (causing leukocyte recruitment), whereas Abd El-Latif's sample may have included chronic or partially resolved cases. A recent study found significant leukocytosis and neutrophilia in Shami breed goats with mastitis, along with reduced RBC, PCV, and lymphocytes [18]. Similarly, another study observed marked leukocytosis with neutrophilia and lymphopenia in clinically mastitic goats [14].

Thus, our elevation of WBC and neutrophils in severe cases is well supported by recent goat studies [14, 18], whereas the extent of anemia appears mild (perhaps secondary to inflammation and edema). Overall, our hematology results indicate an inflammatory leukocyte response (likely driven by neutrophil mobilization) and slight anemia in clinical mastitis, broadly consistent with other caprine reports. Such changes support the idea that severe mastitis can trigger systemic inflammation in goats, albeit blood parameter shifts may be more subtle than in cattle.

The mean platelet count was lowered in goats critical and clinical mastitis, which agreed with the study of Red Sokoto does, where mastitic value was slightly lower than the non-mastitic does [16]. The pattern of platelet count showed a reduction then an increased, and finally decreased again in severe mastitis form. These effects are mainly attributed to in the beginning of the inflammatory process during the critical form; the platelet count lowered due to inflammation, then the body tries to increase the platelet count in subclinical mastitis, and finally it decreased again due to widespread consumption of platelet as part of the clotting and inflammatory response. A decrease in platelet count can be a marker of the severity of the infection [19].

Somatic cell count (SCC) in goat milk rose markedly with mastitis severity, paralleling trends reported in caprine literature. In current study, healthy goats had a mean SCC of 89×10^3 cfu/mL, subclinical cases 303×10^3 cfu/mL, and clinical mastitis goats 6365×10^3 cfu/mL. This gradient reflects activation of udder inflammation: subclinical infection produced elevated SCC above normal, while clinical infection drove SCC into the millions. These values are within ranges reported by other researcher studying goats researchers. For example, a study found repeatedly healthy goats averaged only 277×10^3 cfu/mL, and a worldwide review noted average physiological SCC $\approx764\times10^3$ cfu (range 200-$1{,}500\times10^3$) in healthy goats. An SCC threshold of 500×10^3 cfu/mL to distinguish infected quarters was previously proposed [20]. Accordingly, our subclinical average (303×10^3) and clinical average (6365×10^3) well exceed this, compared to healthy goats (89×10^3).

Consistent with our findings, a study analyzed Norwegian dairy goats and likewise recommended relatively high SCC action limits: 500×10^3 cfu/mL for first-lactation goats, rising to 3000×10^3 in older goats at risk of Staphylococcus aureus infection [7]. This mirrors our clinic group (6365×10^3). A similar study reported mean SCC $\approx$ 2,000–4,000$\times10^3$ cfu/mL in infected goats [21], aligning with our clinical values.

Conversely, a study found no significant difference in mean SCC between bacteriologically positive and negative goat milk samples in Egypt [4]. They caution that SCC is an unreliable mastitis indicator in goats, especially given physiological fluctuations. Indeed, goats naturally have higher and more variable SCC than cows (due to apocrine secretion), so absolute thresholds are debated.

In our analysis, milk PCT was low in all healthy goats, modestly elevated in subclinical mastitis, and markedly elevated in clinical cases. In other words, milk PCT rose several-fold with infection severity, paralleling but rising earlier than SCC increases. Although goat-specific milk PCT data do not exist, analogous research in other contexts suggests local inflammatory proteins appear early in milk. For instance, a study detected cathelicidin proteins in goat milk at early stages of mastitis, often before somatic cell increases, and achieved ~82% overall diagnostic accuracy [22]. This indicates that goat mammary glands release innate immune markers into milk promptly upon infection. By analogy, our finding that milk PCT was elevated even in subclinical mastitis (with just slight increases in SCC) suggests it may similarly signal early inflammation.

Procalcitonin is produced by many tissues in response to infection, and it may enter mammary secretions during mastitis. Our data indicate that measuring milk PCT could potentially complement SCC for mastitis detection. Further research is needed to validate diagnostic thresholds. As an exploratory result, our evidence of significantly higher milk PCT in infected goats implies that PCT merits consideration as a milk biomarker for caprine mastitis, akin to the role of milk cathelicidins [22].

Serum procalcitonin levels in our goats strongly reflected mastitis severity, consistent with PCT's role as a systemic inflammation marker. In healthy controls, serum PCT averaged only 17.79 pg/mL but it rose to 35.61 pg/mL in critical mastitis, 55.47 pg/mL in subclinical mastitis, and 210.44 pg/mL in clinical mastitis ($p<0.01$ vs control). Thus, systemic PCT was fourfold higher in clinical than in

subclinical cases, and in order of magnitude above normal. These goat results are similar in magnitude to those reported for other caprine infections. A study measured PCT in goats with bacterial pneumonia and found a mean serum PCT of ~55.39 pg/mL in healthy goats versus 229.88 pg/mL in diseased goats [13]. Our values (17.8 and 210 pg/mL) align closely with those findings (noting differences in disease type and assay) [13], reflecting the acute inflammatory response.

PCT elevations were most dramatic in goats with overt clinical signs, as seen in cattle studies as well. For example, a study in dairy cows found serum PCT and cytokines markedly increased in Staph aureus mastitis [10], although species differences preclude direct quantitative comparison. Importantly, our goat-specific evidence suggests that PCT behaves as an acute-phase indicator in caprine mastitis, rising tens to hundreds of pg/mL above baseline in infection. These results imply that combining serum PCT measurement with clinical evaluation could improve mastitis detection.

In severely mastitic goats, serum PCT was >10-fold the normal level. In practical terms, this degree of elevation is likely to be diagnostically useful. While SCC and milk PCT indicate local udder infection, serum PCT and other acute-phase reactants reflect systemic involvement and could help differentiate severe mastitis. For instance, goats with systemic illness (e.g., fever, anorexia) should have markedly higher serum PCT than those with only occult infection. By analogy to Nair et al.'s pneumonia study, where PCT correlated with temperature and respiratory rate, we predict that mastitic goats' PCT would correlate with fever and other signs; our data are consistent with this expectation [23].

Conclusion

Measuring procalcitonin in serum and milk shows clear promise as an early detector of subclinical mastitis in Egyptian Nubian goats. In this study of Egyptian dairy goats, PCT levels rose markedly even in subclinical infections, whereas SCC was only dramatically elevated in clinically overt cases. Such early detection may allow timelier treatment and better control of goat mastitis, reducing economic losses. We recommend further validation of PCT assays in larger goat populations and development of field-usable tests. If confirmed, routine PCT testing (especially in milk) together with SCC could become a valuable tool for udder health management in goat-herds

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Declaration of Conflict of Interest

The authors declare that there is no conflict of interest.

Authors' Contribution

All authors participated in the study's conception and design. Data collection, clinical examination, and experimental study were performed by YNA. All biochemical analysis was performed by YNA and AHG. Data analysis was performed by YNA and AHG. YNA, NEE and FAS drafted and corrected the manuscript; NEE and FAS revised the manuscript. All authors read and approved the final manuscript.

Ethical of approval

The present research protocol was reviewed and approved by the Institutional Animal Care and Use Committee of Cairo University (IACUC) under approval number Vet CU 131020241048

TABLE 1. Vital signs in Critical, Subclinical, and Clinical Mastitic Nubian Goats Compared to the Healthy Control Group

Groups Parameters	Control (n=8) Mean ± SE	Critical (n=5) Mean ± SE	Subclinical (n=8) Mean ± SE	Clinical (n=29) Mean ± SE
Temperature (°C)	39.05 ± 0.18	39.62 ± 0.35	39.40 ± 0.22	40.68 ± 0.11[a]
Heart Rate (bpm)	78.45 ± 2.10	84.20 ± 3.55	82.10 ± 1.95	96.87 ± 0.92 [a]
Respiratory Rate (breaths/min)	15.73 ± 0.64	19.28 ± 1.20	18.90 ± 0.78	25.34 ± 0.46 [a]

a: p≤ 0.001 , b: p≤ 0.01 , c: p≤0.05

TABLE 2. Hematological Profile in Critical, Subclinical, and Clinical Mastitic Nubian Goats Compared to the Healthy Control Group

Groups Parameters	Control (n=8) Mean ± SE	Critical (n=5) Mean ± SE	Subclinical (n=8) Mean ± SE	Clinical (n=29) Mean ± SE
RBCs x106 (u/L)	9.98 ± 0.13	9.62 ± 0.27	9.22 ± 0.21 [b]	8.38 ± 0.24 [a]
Hb (g/dl)	10.77 ± 0.20	11.12 ± 0.41	10.56 ± 0.40	9.38 ± 0.22 [a]
PCV%	37.38 ± 0.64	37.48 ± 1.02	35.11 ± 0.51 [c]	34.51 ± 0.45 [b]
Plts $X10^3$ (u/L)	388.00 ± 20.91	307.00 ± 24.15 [c]	314.75 ± 29.02	287.37 ± 12.31 [a]
MCV (fl)	38.23 ± 0.43	38.94 ± 0.51	38.44 ± 1.02	42.23 ± 1.04 [a]
MCH (pg)	10.80 ± 0.22	11.54 ± 0.23	11.51 ± 0.59	11.39 ± 0.24
MCHC (g/dl)	28.25 ± 0.38	29.67 ± 0.82	30.08 ± 1.07	28.37 ± 1.29
WBCs$X10^3$ (u/L)	9.81 ± 0.41	10.04 ± 1.01	10.50 ± 0.53	13.60 ± 0.61 [a]
Lymphocytes (%)	4.95 ± 0.41	5.28 ± 0.45	5.14 ± 0.26	5.86 ± 0.22
Neutrophils (%)	4.84 ± 0.66	4.76 ± 1.18	6.19 ± 0.81	5.86 ± 0.42
Monocytes (%)	0.41 ± 0.01	0.45 ± 0.07	0.53 ± 0.04 [c]	0.75 ± 0.04 [a]
Eosinophils (%)	0.31 ± 0.009	0.30 ± 0.02	0.33 ± 0.02	0.46 ± 0.02 [a]
Basophils(%)	0.17 ± 0.01	0.20 ± 0.02	0.17 ± 0.01	0.20 ± 0.01

a: p≤ 0.001 , b: p≤ 0.01 , c: p≤0.05, RBCs: red blood cells, Hb: hemoglobin, PCV: packed cell volume, Plts: platelets, MCV: mean corpuscular volume, MCH: mean corpuscular hemoglobin, MCHC: mean corpuscular hemoglobin concentration, and WBCs: white blood cells.

TABLE 3. Inflammatory Biomarkers in Milk and Serum of Critical, Subclinical, and Clinical Mastitic Nubian Goats Compared to the Control Group.

Groups Parameters	Control (n=8) Mean ± SE	Critical (n=5) Mean ± SE	Subclinical (n=8) Mean ± SE	Clinical (n=29) Mean ± SE
Milk inflammatory biomarkers				
SCC x103 (cfu/ml)	89.00 ± 14.07	202.20 ± 12.30 [a]	303.12 ± 11.33 [a]	6365.41 ± 1777.62 [a]
PCT-Milk (pg/ml)	5.53 ± 1.87	15.02 ± 0.45 [b]	25.23 ± 1.93 [a]	124.16 ± 40.62 [b]
Serum inflammatory biomarkers				
PCT- Serum (pg/ml)	17.79 ± 3.19	35.61 ± 2.85 [b]	55.47 ± 1.63 [a]	210.44 ± 41.47 [a]
Calcium (mg/dl)	8.45 ± 0.21	9.40 ± 0.74	8.97 ± 0.60	9.26 ± 0.37

a: p≤ 0.001 , b: p≤ 0.01 , c: p≤0.05, SCC: somatic cell count, and PCT: procalcitonin

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مستويات البروكالسيتونين في المصل والحليب كمؤشرات تشخيصية مبكرة لالتهاب الضرع الحرج وتحت السريري والسريري في الماعز النوبي المصري

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الملخص

التهاب الضرع في الماعز مرض شائع ومكلف في الماعز الحلوب، وخاصة في مصر، حيث يتم تربية حوالي 4.2 مليون ماعز. يسود الشكل دون السريري مما يجعل الكشف المبكر أمرًا بالغ الأهمية. البروكالسيتونين هو هرمون مرتبط بتسمم الدم ويرتفع في حالات العدوى البكتيرية. وللأسف، لم يتم إجراء دراسات موسعة على الماعز. في هذه الدراسة، قمنا بقياس البروكالسيتونين في كل من المصل والحليب إلى جانب عدد الخلايا الجسدية (SCC) والكالسيوم في خمسين من الماعز النوبي المصري المرضعة البالغة، بمتوسط وزن الجسم (45 ± 5) كجم وفئة عمرية تتراوح بين 6 و7 سنوات، والتي تم تصنيفها إلى أربعة: المجموعة الضابطة (ن = 8)، والحرجة (ن = 5)، ودون السريرية (ن = 8)، والتهاب الضرع السريري (ن = 29). أشارت نتائجنا إلى زيادة في العلامات الحيوية مثل درجة الحرارة والنبض ومعدل التنفس في مجموعة التهاب الضرع. بالإضافة إلى متوسط حجم الكريات، ارتفع عدد خلايا الدم البيضاء والوحيدات والحمضات في المجموعة السريرية، والوحيدات في المجموعة دون السريرية. كان هناك انخفاض في خلايا الدم الحمراء وحجم الخلايا المكدسة (PCV) في المجموعة دون السريرية وفي خلايا الدم الحمراء والهيموغلوبين وحجم الخلايا المكدسة والصفائح الدموية في المجموعة السريرية. أظهرت المجموعة الحرجة انخفاضًا كبيرًا في عدد الصفائح الدموية. أظهرت النتائج ارتفاعًا تدريجيًا في كل من البروكالسيتونين في المصل والحليب مع زيادة شدة التهاب الضرع، بينما كان SCC مرتفعًا للغاية في الحالات السريرية. نستنتج أن قياس مستويات البروكالسيتونين في المصل أو الحليب قد يشارك في الكشف المبكر عن التهاب الضرع الحرج وتحت السريري ويساعد في تشخيص التهاب الضرع الشديد في الماعز النوبي.

الكلمات الدالة: التهاب الضرع، البروكالسيتونين، SCC، الماعز النوبي، المصل والحليب.