# Serological diagnosis of persistent infection with *Anaplasma marginale* bacteria in cattle

**Hasanain AJ. Al-gharban[1] and Salim H. Dhahir[2]**

[1]Department of Internal and Preventive medicine, College of Veterinary Medicine, Wasit University, [2]Department of Internal and Preventive Medicine, College of Veterinary Medicine, Baghdad University, Iraq.

E-mail: hassluq@yahoo.com

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## Summary

Bovine anaplasmosis is one of the tick-borne diseases caused by *Anaplasma marginale* bacteria which can cause high economic losses to livestock. Cattle that recovered from acute infection become carriers without clinical signs related to the disease and these bacteria can persist for lifetime in the blood. The present study was conducted to detect antibodies of persistently infected cattle with *A. marginale* in Wasit province/ Iraq. A total of 100 blood samples were collected randomly from cattle over one year old. Blood smears were prepared, stained with Giemsa's stain and subjected to microscopic examination for detection of *Anaplasma marginale* bacteria within an infected RBCs, while serum samples were tested by a competitive enzyme - linked immunosorbent assay test (cELISA) for detection of antibodies in persistently infected cases . Mythic 18 Vet system was used as blood analysis for blood parameters measurement. Results of blood smear examination revealed 13 acute cases, while ELISA detected 35% of carriers. Whereas, the hematological parameters showed that the acutely infected cattle had the macrocytic hypochromic anemia, the persistently infected cattle displaed the microcytic hypochromic and the normocytic hypochromic anemia. Results of blood analysis revealed a significant difference ($P<0.05$) in hematological parameters of acute and chronic cases. It has been concluded that cELISA is a reliable screening test for detection of antibodies specific to *Anaplasma marginale* bacteria.

**Keywords: *Anaplasma marginale*, Anaplasmosis, Bovine persistent infection, cELISA.**

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## Introduction

Bovine anaplasmosis is an infectious non contagious, transmissible arthropod borne hemoparasitic disease of cattle caused by an obligate intraerythrocytic rickettsia (1 - 3). Clinical anaplasmosis is more commonly encountered in cattle older than 1 year of age (4). Animals that recovered clinically are lifelong carriers of agent (5). Diagnosis of bovine anaplasmosis is usually based on signalment and presenting clinical signs as well as laboratory tests such as light microscopic examination of Giemsa - stained blood smears or serological/ molecular diagnostic procedures (6). In carrier animals, microscopic diagnosis can be difficult, owing to variable rickettsemia, and thus, a variety of serologic tests or genetic material of the agent are used to detect the specific antibodies. A competitive ELISA has been used for diagnosis of *A. marginale* infection in various ruminants including cattle, sheep and deer (7). Currently cELISA test used for diagnosis of bovine anaplasmosis is based on use of a monoclonal antibody that recognizes MSP5 of *Anaplasma marginale* (8). This antigen is conserved among all known species of *Anaplasma* (9). The test is in accordance with PCR for diagnosis *A.marginale* infection in cattle (10). In Iraq, *A. marginale* infection was recorded in northern, middle and southern parts. Microscopic blood smear examination and cELISA were used and compared for diagnosis of acute infection in all these studies (11 - 17). The aim of this study is to diagnose the carriers of *Anaplasma marginale* infection in cattle by cELISA test and to demonstrate types of anemia in acute and persistent infections.

## Materials and Methods

A total of 100 local breed cattle of different age groups over one year old were selected randomly in Al-Aziziyah/ wasit Province/Iraq. Case history, clinical examination and clinical signs were recorded for each animal. Blood samples were collected for hematological and serological examination. Thin blood smears were prepared from each unclotted blood

sample and fixed with absolute methanol examined under oil immersion objective lens for detection of infected erythrocytes (18). Blood analysis system (Mythic 18 Vet/ Orphee, Switzerland) was used for determination of other blood parameters (PCV, Hb, total RBCs, MCV, MCH and MCHC) (19). Competitive Enzyme - Linked Immuno - sorbent Assay test (cELISA) was performed according to manufacturer's instructions (95%) stained with Giemsa's stain and (SVANOVA, Sweden). Statistical software program (SPSS20.0) was used for statistical analysis (20).

## Results and Discussion

The results of hundred examined cows of different age groups, revealed 48 infected cattle, 35 (72.9 %) were persistently infected cases or chronic carriers and 13 (27.1%) were acutely infected cases (Table, 1).

**Table, 1: Infected cases of bovine anaplasmosis; age, number and percentage.**

| Age / year | Total tested number | Infected cases | | | | Total Infected number |
|---|---|---|---|---|---|---|
| | | Acute | | Chronic ( Carrier ) | | |
| | | No. | % | No. | % | |
| > 1 - 2 | 13 | - | - | 3 | 6.25 | 3 |
| > 2 - 3 | 27 | 2 | 4.17 | 10 | 20.8 | 12 |
| > 3 - 4 | 39 | 9 | 18.75 | 8 | 16.7 | 17 |
| > 4 | 21 | 2 | 4.17 | 14 | 29.17 | 16 |
| Total | 100 | 13 | 27.1 | 35 | 72.9 | 48 |

As presented in (Table, 1), the acute stage is common in adult cattle over 2 years old, while persistent infection is prevalent in all age groups. This may be attributed to that calves and yearlings may display subclinical signs of anaplasmosis. This fact was reported by many authors, who stated that *Anaplasma* infection is mild or subclinical in calves under 9 months, yearlings and recovered cattle or those under one year (6 and 21). Calves from immune mothers receive temporary protection from the colostrum which prevents anaplasmosis. This protection lasts about 3 months, and in most cases , is followed by an age resistance, which lasts until the animals are about 9 to 12 months of age (13 and 22). Although, (23) suggested that in animal < 1 year old, Anaplasmosis is usually subclinical, in yearlings and 2 years old, it is moderately severe and in older cattle, it is severe and often fatal. While (24) concluded that clinical infections were significantly observed in cattle of all ages.

The age resistance in calves gradually wanes after one year of age and these animals become increasingly susceptible to the disease in the regions which have no endemic stability (25 and 26). The results of this study was also compatible with those of (27) who recorded that the highest seroprevalence of *Anaplasma marginale* was found in > 4 years old cattle compared to other age cohorts (< 1, 1 - 2 and > 2-3 years). Whereas, (28) considered higher sero-prevalence in specific age might be associated with size of animal groups. The results of clinical signs (Range and Mean ± SE) were as follows: In acute infection, temperature 37.9 – 41.4°C and mean 40.15 ± 0.24°C, pulse rate 62 – 92/ minute and 74.07 ± 2.66/ minute, respiratory rate 25 – 41/ minute and 33.46 ± 1.5/ minute, while in carriers, temperature 37.6 – 40.5°C and mean 39.05 ± 0.13°C, pulse rate 48 – 95 minute and 67.14 ± 1.8/ minute, respiratory rate 22 – 38/ minute and 30.14 ± 0.66/ minute.

There is a significant increase ($P<0.05$) in temperature, pulse rate and respiratory rate between acute cases and carriers (Table, 2). There is a significant increase in body temperature, pulse and respiratory rate in acute cases compared to chronic carriers, while these signs were within the normal range in persistently infected cases or chronic carriers with minimal variation according to age, genetic factors, country and season (29). The elevation noticed in acute infection may be associated with peak rickettsemia and anemia, where a transient febrile response occurs concurrently with increased pulse and respiratory rate (23). The febrile crisis may be a result of pyrogens releasing from destruction

of WBCs, these pyrogens effect on the hypothalamus, causing elevation of the body temperature. The increasing in body temperature causes increasing in respiratory rate. While the increasing in pulse rate resulting from anemia and dehydration. Furthermore, the increased respiratory rate may be ascribed to hypoxemia and subsequent tissue hypoxia. Anemia is usually accompanied by increase cardiac output, pulse rate and respiratory rate (6). These signs may also be affected by several factors, including stress, environment, species, age, sex, pregnancy, lactation and trace mineral deficient diet (21 and 23).

Results of hematological parameters (Table, 3) revealed that there is a significant decrease ($P<0.05$) in means of PCV, Hb, RBCs and MCHC in acute cases compared to chronic carriers. While there is a significant increase in MCV and MCH in acute cases compared to carriers. The decrease in blood indices may refer to anemia which may be ascribed to fact that high erythrocytes ricketts emia occurs during acute infection with *A. marginale* resulting in splenic and hepatic macrophage-mediated phagocytosis constitute definitive diagnosis, so classification schemes are used for definitive diagnosis ,as a single classification may not be entirely satisfactory (4). The diversity of mean values and ranges of blood indices may be associated with a dose and virulence of the causative strain, host, susceptibility or genetic factors, nutrition, geographic distribution, physiologic status, stage of infection, age, breed, coincident infection and development of cyclic rickettsemia (21 - 32).

**Table, 2: Vital clinical signs of acute and carrier cases of bovine anaplasmosis; Range and Mean ± SE**

| Signs | Acute ( 13 ) | Carrier ( 35 ) |
|---|---|---|
| Temperature ( °C ) | 37.9 – 41.1<br>40.15 ± 0.24 [a] | 38 – 40.5<br>39.1 ± 0.12 [b] |
| Pulse / minute | 62 - 92<br>74.07 ± 2.66 [a] | 48 – 95<br>67.14 ± 1.8 [b] |
| Respiratory rate / minute | 25 – 41<br>33.46 ± 1.5 [a] | 22 – 38<br>30.14 ± 0.66 [b] |

**The difference in small letters horizontally refers to significant differences at level P< 0.05.**

**Table,3: Hematological parameters of acute and carrier bovine anaplasmosis; Range and Mean ± SE**

| Hematological parameters | Acute | Carrier |
|---|---|---|
| PCV % | 21.1 – 39.5<br>27.46 ± 1.54 a | 19.8 – 40.2<br>30.37 ± 0.85 b |
| Hb. g / dl | 5.3 –11.4<br>7.55 ± 0.53 a | 4.92 – 12.7<br>9.7 ± 0.35 b |
| RBCs × 106 / µl | 3.45 – 6.3<br>4.37 ± 0.25 a | 3.51 – 9.1<br>7.17 ± 0.24 b |
| MCV fl | 60.2 – 67.7<br>62.9 ± 0.6 a | 37 – 61.2<br>43.12 ± 1 b |
| MCH pg | 15.1 – 18.7<br>17.13 ± 0.35 a | 10.6 – 16.5<br>13.56 ± 0.23 b |
| MCHC g / dl | 23.6 – 29.5<br>27.26 ± 0.49 a | 21.7 – 36.2<br>31.7 ± 0.57 b |

Results of anemia in bovine anaplasmosis (Table, 4) showed that acutely infected cattle 13 cows displayed macrocytic hypochromic anemia (56.5%).While out of 35 persistently infected cattle, 7 cows revealed microcytic hypochromic anemia (30.4%) and 3 cows presented normocytic hypochromic anemia (13% ).

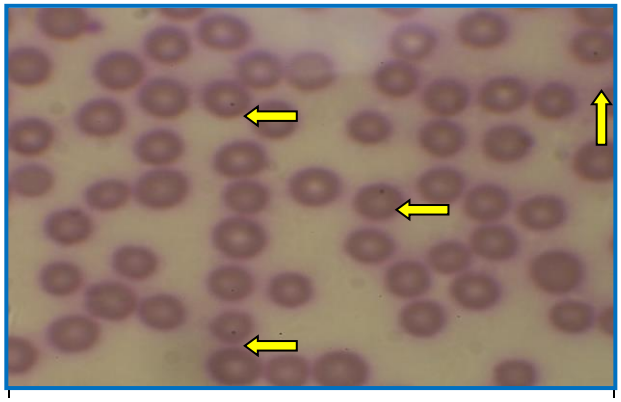
**Table, 4: Type of anemia in acute and carrier bovine anaplasmosis.**

| Infection stage | Type of anemia ( % ) | | | Total |
|---|---|---|---|---|
| | Macrocytic hypochromic | Microcytic hypochromic | Normocytic Hypochromic | |
| Acute | 13 | - | - | 13 |
| Chronic ( Carrier ) | - | 7 | 3 | 10 |
| Total | 13 ( 56.5 % ) | 7 ( 30.4 % ) | 3 ( 13 % ) | 23 |

Anemia is usually classified according to size (MCV) and Hb concentration (MCHC) of the erythrocytes (19). The results revealed macrocytic hypochromic anemia (56.5%) in acutely infected cases. While persistently infected cases displayed microcytic hypochromic anemia (30.4%) and normocytic hypochromic anemia (13%). These results were similar to those reported by many researchers in Iraq and other countries, where different types of anemia has been described as a result of acute infection with anaplasmosis

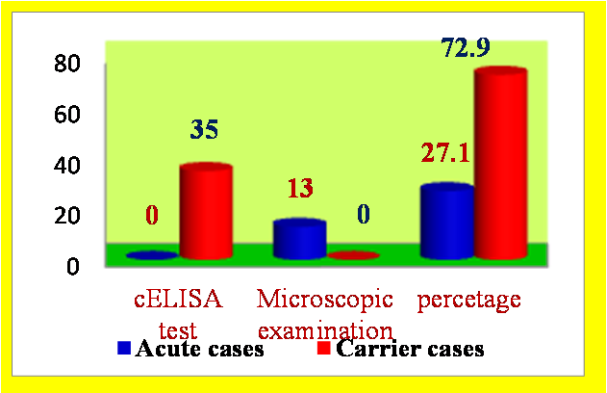
(13 and 33-35). Anemia was drastic in acute cases compared to chronic carriers that experienced transitory anemia, this may be attributed to that rickettsemia could subside gradually with disease progression to subclinical stage, where the parasitic erythrocytes decrease to undetectable level (below 1%). This fact was demonstrated in different geographical distribution of anaplasmosis (36 - 38).

The anisocytosis, poikilocytosis, reticulo-cytosis and polychromasia noticed in this study were in accordance with results of (13 and 39 - 41). The result of cELISA was higher than those reported in neighboring countries, where, (42) detected 50 % of perpetually infected cases of anaplasmosis by ELISA in Iran. While (43) discovered 11 % of persistently infected cases by ELISA in Turkish cattle. Moreover, (44) recorded 1% of *Anaplasma* carrier cases by this test in Saudi Arabia. The higher incidence of bovine anaplasmosis in Iraq compared to neighboring countries may be associated with Poor management, lack of tick control practices and inadequate economic sustainability of poor resource small holder farmers for the implementation of proper management and animal health practices in Iraq versus neighboring countries. However, anaplasmosis is a worldwide distributed disease, the diversity in its incidence among countries is due to differences in geographical distribution, availability of vectors, host susceptibility, age, breed, virulence of different strains of *A.marginale*, grazing practice, management and sanitary practice (21 and 28-32).



**Figure, 1: Microscopic picture of infected RBCs with *A.marginale* under oil lens x 100.**

Results of microscopical and serological examination revealed 13 (27.1) acutely infected cases by microscopic examination of stained blood smears (Fig.1). While cELISA detected 35 (72.9 %) of chronic carrier cases (Fig. 2).




**Figure, 2: Microscopical and serological diagnosis of bovine anaplasmsis**

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## References

1. Dumler, J. S.; Barbet, A. F.; Bekker, C. P.; Dasch, G. A.; Palmer, G. H.; Ray, S. C.; Rikihisa, Y. and Rurangirwa, F. R. (2001). Reorganization of genera in the family's Rickettsiaceae and Anaplasmataceae in the order Rickettsiales: unification of some species of *Ehrlichia* with *Anaplasma*, *Cowdria* with *Ehrlichia* and *Ehrlichia* with *Neorickettsia*, descriptions of six new species combinations and designation of *Ehrlichia equi* and 'HGE agent' as subjective synonyms of *Ehrlichia phagocytophila*. Int. J. Syst. Evol. Microbiol., 51: 2145-2165.
2. Carreño, A. D.; Alleman, A. R.; Barbet, A. F.; Palmer, G. H.; Noh, S. M. and Johnson, C. M. (2007). In vivo endothelial cell infection by *Anaplasma marginale*. Vet. Pathol. Online, 44(1):116-118.
3. Andrews, A. H.; Blowey, R. W.; Boyd, H. and Eddy, R. G. (2008). Bovine Medicine:

diseases and husbandry of cattle. Wiley-Blackwell,

4. Zivkovic, Z. (2010). Tick-pathogen interactions in bovine anaplasmosis. A talanta Drukw - erkbe middeling, Houten. ISBN 978 -90 – 393 -5298 - 4.
5. Kocan, K. M.; de la Fuente, J.; Blouin, E. F. and Garcia-Garcia, J. C. (2004). *Anaplasma marginale* (Rickettsiales: Anaplasmataceae): Recent advances in defining host-pathogen adaptations of a tick-borne rickettsia. Parasitology. 129: 285-300.
6. Radostits, O. M.; Gay, C. C.; Hinchcliff, K. W. and Constable, P. D. (2007). Veterinary Medicine. A textbook of the diseases of cattle, horses, sheep, pigs and goats. W.B. Saunders Company Ltd, London, Pp: 1454 - 1459.
7. Kocan, K. M.; de la Fuente, J.; Blouin, E. F.; Coetzee, J. F. and Ewing, S. A. (2010).The natural history of *Anaplasma marginale* . Vet. Parasitol., 167(2): 95-107.
8. de la Fuente, J.; Vicente, J.; Hofle, U.; Ruiz-Fons, F.; Fernandez de Mera, I. G.;Van Den Bussche, R. A.; Kocan, K. M. and Gortazar, C. (2004). *Anaplasma marginale* infection in free-ranging Iberian red deer in the region of Castilla La Mancha, Spain. Vet. Microbiol., 100: 163-173.
9. Salih, D. A.; Rahman, M. A.; Mohammed, A. S.; Ahmed, R.; Kamal, S. and Hussein, A. M. (2009). Seroprevalence of tick-borne diseases among cattle in the Sudan. Parasitol. Res., 104(4): 845-850.
10. de Echaide, S. T.; Knowles, D. P.; McGuir, T. C.; Palmer, G. H.; Suarez, C. E. and McElwain, T. F. (1998). Detection of cattle naturally infected with *Anaplasma marginale* in a region of endemicity by nested PCR and a competitive enzyme-linked immunosorbent assay using recombinant major surface protein 5. J. of clinical microbiol, 36(3): 777-782.
11. Al-Shukur, A. A. (1989). A clinical study of the incidence of bloody theileriosis and anaplamosis in chronic phase in the local cows. Report Higher Diploma of the College of Veterinary Medicine - University of Mosul.
12. Latif, B. M. A. and Ali, S. R. (1989). Prevalence of Bovine anaplasmosis in Iraq. Iraqi. J. Vet. Med.13: 91- 95.
13. Alsaad, K. M. (1990). Clinical, hematological and Biochemical study for local bovine anaplasmosis. Master Thesis, college of Veterinary Medicine, University of Mosul.
14. Al-Robaiy, M. A. (1994). A study of hematological and biochemical change in sheep experimentally infected with *Theileria hirci*. M. Sc. Thesis, College of Veterinary Medicine, Baghdad University. (In Arabic). Pp: 4- 67.
15. Saoud, H. A. (1997). Epidemiological and diagnostic study on mixed infection of major diseases in cattle .Doctoral Thesis, College of Veterinary Medicine, Baghdad University. (In Arabic). Pp: 83 - 99.
16. Ameen, K. A.; Abdullah, B. A. and Abdul-Razaq, R. A. (2012). Seroprevalence of *Babesia bigemina* and *Anaplasma marginale* in domestic animals in Erbil, Iraq. Iraqi J. Vet. Sci., 26(3): 109-114.
17. Gati, J. A. (2012). Seroprevalence of *Anaplasma marginale* among cows in Nasiriya city- South of Iraq- by competitive ELISA, Al Qadisiya J. Vet. Med. Sci., 11(1): 105- 108.
18. Singla, A. A.; Balwinder, P. K. and Juyal, K. B. (2013). Prevalence and haemato-biochemical profile of *Anaplasma marginale* infection in dairy animals of Punjab (India). Asian Pacific J. Tropical Medicine, 15: 139-144.
19. Harold, T. (2010). Laboratory and Clinical Diagnosis of Anemia, In, Weiss, D. J.; Wardrop, K. J.; Schalm's Veterinary Hematology. $^{6}$th ed., Blackwell Publishing Ltd. Pp: 199-210.
20. Onwuegbuzie, A. J. and Leech, N. L. (2007). Sampling Designs in Qualitative Research: Making the Sampling Process More Public. Qualitative Report, 12(2): 238-254.
21. Smith, B. P. (2004). Large animal internal medicine, 4th Edition, New York, Mosby. Pp: 1155-1156.
22. Tassi, P.; Carelli, G. and Ceci, L. (2002). Tick - borne diseases (TBDs) of dairy cows in a Mediterranean environment: a clinical, serological, and hematological study. Ann. N. Y. Acad. Sci., 969: 314-317.
23. Kahn, C. M.; Scott, L. and Aiello, S. E. (2005). The Merck veterinary manual 9th ed. Copyright (C) by Merck Co., Inc printed in

the USA by National publishing. Inc. Philadelphia, Pensylvania, Pp: 146-148.

**24.** Birdane, F. M.; Sevinc, F. and Derinbay, O. (2006). *Anaplasma marginale* infection in dairy cattle: clinical disease. Bull Vet Inst Pulawy, 50: 467- 470.

**25.** Gale, K. R.; Dimmock, C. M.; Gartside, M. and Leatch, G. (1996). *Anaplasma marginale*: detection of carrier cattle by PCR and ELISA. Int. J. Parasitol, 26: 1103-1109.

**26.** Muraleedharan, K.; Ziauddin, K. S.; Hussain, P. M.; Pattabyatappa, B.; Mallikarjun, G. B. and Seshardi, S. J. (2005). Incidence of Anaplasma spp., Babesia sp. and Trypanosoma sp. in cattle of Karnataka. J. Vet. Parasitol. 19: 135-137.

**27.** Atif, F. A.; Khan, M. S.; Khan, M. A.; Ashraf, M. and Avais, M. (2012). Chemotherapeutic efficacy of oxytetracycline, enrofloxacin and imidocarb for the elimination of persistent *Anaplasma marginale* infection in naturally infected Sahiwal cattle. Pakistan J. of Zoology, 44 (2): 449 - 456.

**28.** Swai, E. S.; Karimuribo, E. D.; Ogden, N. H.; French, N. P.; Fitzpatrick, J. L.; Bryant, M. J. and Kambarage, D. M. (2005). Seroprevalence estimation and risk factors for *A. marginale* on smallholder dairy farms in Tanzania. Trop. Anim. Health Prod. 37: 599-610.

**29.** Mtshali, M. S.; De la Fuente, J.; Ruybal, P.; Kocan, K. M.; Vicente, J.; Mbati, P. A. and Latif, A. A. (2007). Prevalence and genetic diversity of *Anaplasma marginale* strains in cattle in South Africa. Zoonoses and public health, 54(1): 23-30.

**30.** Weiss, D. J. and Wardrop, K. J. (2010). Normal Hematology of Cattle. Schalm's Veterinary Hematology. 2121 State Avenue, Ames, Iowa 50014-8300, USA. ISBN 978-0-8138-1798-9. Pp: 829 - 835.

**31.** Duncan, J. R. and Prasse, K. W. (2003). Veterinary Laboratory Medicine: Clinical Pathology, 4th ed. Ames: Blackwell: 3 - 45.

**32.** Alonso, A. J.; Teresa, R. D.; Garcia, M.; Gonzalez, J. R. and Vallejo, M. (1997). The effects of age and reproductive status on serum and blood parameters in Merino breed sheep. J. Vet. Med. 44: 223-231.

**33.** Swenson, C. and Jacobs, A. (1986). Spherocytosis associated with anaplasmosis in two cows. JAVAMA.188:1061- 1063.

**34.** Egbe Nwiyi, T. N.; Salako, M. A. and Otukonyong, E. E. (1997). Observations on naturally occurring bovine anaplasmosis in arid zone of North-Eastern Nigeria: prevalence, haematological and pathological changes. Studies and Researches in Veterinary Medicine, 5: 95-99.

**35.** Nazifi, S.; Razavi, S. M.; Mansourian, M.; Nikahval, B. and Moghaddam, M. (2008). Studies on correlations among parasitaemia and some hemolytic indices in two tropical diseases (theileriosis and anaplasmosis) in Fars province of Iran. Trop. Anim. Health. Prod., 40: 47–53.

**36.** Allison, R.W. and Meınkoth, J. H. (2010). Anemia caused by *Rickettsia*, *Mycoplasma*, and Protozoa erythrocytes. In, Weiss DJ, Wardrop KJ (Eds): Schalm's Veterinary Hematology. 6th ed., Blackwell Publishing Ltd. Pp: 199-210.

**37.** Krista, J. H. and Dorothy, W. G. (2010). An update on bovine anaplasmosis (*Anaplasma marginale*) in Canada. Can. Vet. J. 51(8): 837–840.

**38.** Coşkun, A.; Derınbay Ekıcı, Ö.; Güzelbekteş, H.; Aydoğdu, U. and Şen, İ. (2012). Acute Phase Proteins, Clinical, Hematological and Biochemical Parameters in Dairy Cows Naturally Infected with *Anaplasma Marginale*. Kafkas Univ. Vet. Fak. Derg., 18 (3): 497-502.

**39.** Prchal, J. (2005). Clinical manifestations and classification of erythrocyte disorders. In: Williams hematology. Lichtman, M. A.; Williams, W. J.; Beutler, E.; Kaushansky, K.; Kipps, T. J.; Seligsohn, U. and Prchal, J. (eds.), (7thedition). Chicago, Illinois, USA; McGraw- Hill Professional. Pp: 411-418.

**40.** Hussain, B. H. (2012). Blood smear analysis for infected cows with blood parasites. M. Sc. Thesis, College of Veterinary Medicine - University of Mosul. (In Arabic). Pp: 67 - 78.

**41.** Thrall, M. A.; Weiser, G.; Allison, R. and Campbell, T. W. (2012). Veterinary hematology and clinical chemistry. John Wiley and Sons. Pp.187 – 188.

**42.** Noaman, V.; Shayan, P. and Amininia, N. (2009). Molecular diagnostic of *Anaplasma*

*marginale* in carrier cattle. Iranian J. Parasitol, 4(1): 26-33.

**43.** Aktas, M.; Altay, K. and Dumanli, N. (2011). Molecular detection and identification of *Anaplasma* and *Ehrlichia* species in cattle from Turkey. Ticks and tick-borne diseases, 2(1): 62-65.

**44.** Yoshihara, E.; Vidotto, O.; Yamamura, M. H.; Marana, E.; Pacheco, R. and Silviera, A. P. (2003). Studies of natural infection with *Anaplasma marginale* in nelore cattle in the Umuarama municipality, Paraná State, Brazil. Revista Brasileira de Parasitologia Veterinária, 12(1): 21-26.

## التشخيص المصلي للأصابة المزمنة بجراثيم *Anaplasma marginale* في الابقار

**حسنين عبد الحسين جعفر الغربان [1] و سالم حمد ظاهر [2]**

فرع الطب الباطني والوقائي، كلية الطب البيطري، [1]جامعة واسط، [2]جامعة بغداد، العراق.

E-mail: hassluq@yahoo.com

**الخلاصة**

يعد داء الانابلازما البقري احد الامراض التي تنتقل بواسطة القراد والذي تسببه جراثيم *Anaplasma marginale* مسببا خسائر اقتصادية كبيرة في قطعان الماشية. تبقى الابقار التي تشفى من الاصابة الحادة حاملة للاصابة بدون ظهور اي علامات سريرية تدل على الاصابة بالمرض ويبقى الحيوان حاملا للجراثيم في دمه طوال فترة حياته. هدفت الدراسة الحالية الى تحديد الاجسام المناعية الخاصة بالجراثيم في دم الابقار المصابة بالشكل المزمن للمرض في محافظة واسط / العراق. تم جمع 100 عينة دم من ابقار تم اختيارها عشوائيا بعمر اكثر من سنة وتم تحضير مسحات دموية لكل عينه وصبغها بصبغة الكيمزا لغرض فحصها تحت المجهر وتحديد الكريات الحمراء المصابة بجراثيم *Anaplasma marginale* . بينما تم فحص عينات مصل الدم باختبار الاليزا التنافسي لتحديد الحالات المصابة بالشكل المزمن للمرض. تم استخدام جهاز تحليل الدم Mythic 18 Vet لقياس المعايير الدموية المختلفة المرافقة للاصابة بهذا المرض. اظهرت نتائج فحص المسحات الدموية عن وجود 13 حالة مصابة بالشكل الحاد للمرض في حين اظهر فحص الاليزا التنافسي وجود 35 حالة مصابة بالشكل المزمن للمرض. بينما اظهرت المعايير الدموية حدوث فقر الدم من نوع كبيرة الكرية قليلة الصباغ في الحالات الحادة، وصغيرة الكرية قليلة الصباغ وسوية الكرية قليلة الصباغ في الحالات المزمنة. احصائيا، اظهرت نتائج تحليل الدم وجود اختلاف معنوي ($P < 0.05$) في قيم المعايير الدموية في الحالات الحادة مقارنة بالحالات المزمنة. تم الاستنتاج بان اختبار الاليزا التنافسي يعتبر اختبارا موثوقا به لتحديد الاجسام المناعية الخاصة بجراثيم *Anaplasma marginale*

**الكلمات المفتاحية: انابلازما، حامل جرثومة انابلازما، الابقار المصابة، ايلايزا.**