Open Access
Baghdad Science Journal Vol.13(1)2016

DOI: http://dx.doi.org/10.21123/bsj.2016.13.1.0066

Evaluating the Humoral Immunity and Interleukin 18 Receptor 1 in some Patients with *Molluscum Contagiosum* Infection

Mohammed Abdul-Daim Saleh

Department of Biology, College of Science, Diyala University, Iraq

Received 28, December, 2014
Accepted 29, April, 2015

Abstract:

The molluscum contagiosum virus (MCV) is a dermatotropic poxvirus. The causative agent of molluscum contagiosum (MC) is nonlethal, common and worldwide. Additionally, little inflammation is associated with MC papules. The present study aims to evaluate the immune status of MC patients by measuring the level of immunoglobulins IgG and IgM by using the radial immune diffusion assay (RIA) and the level of interleukin 18 receptor 1 (IL-18R1) by the Enzyme-linked immunosorbent assay (ELISA).The study is conducted during November 2013 to April, 2014 in outpatient clinic of Baquba Teaching Hospital. There are 75 patients, diagnosed with clinical lesions of MCV on different areas of the body, whose age is ranged between 2-50 years including 40(53.3%) males and 35(46.7%) females. The study includes 15 healthy persons age between 2-50 years. The level of IL 18R1 were significantly elevated in patients (677.15±874.22) compared with control (178.46±31.79 ng/ml). There is also a significant elevation in the mean level of serum IgM, where it is 1946.6±825.6 mg/dl while in control group is 140.1±68.7mg/dl. By contrast in patients with lower levels of IgG than the control, the mean serum IgG level in patient is 221.9±96.7 mg/dl while in the control is 1229.9±299.7 mg/dl. Finally, there is no significant difference between MC patients from rural area and urban area.

Key words: Molluscum Contagiosum , Interleukin 18 Receptor 1, IgG , IgM .

Introduction:

Molluscum Contagious Virus (MCV) is first described and later named by Bateman in the early nineteenth century [1]. Additionally , there are various types of MCV and they are most commonly seen on humans, but have been found to be on animals such as chickens, horses, oxen and cows [2].Furthermore, Molluscum contagiosum is caused by up to four closely related types of poxvirus, MCV-1 to 4 and their variants. However, MCV-2 causes the majority of infections, (60%) that is [3]. On other hand , humoral immunity and cellular immunity play an important role in the body's defense against molluscum infection. Most adults are more resistant to MCV infection than children because they have developed IgG antibodies against the viral antigen [4]. Moreover *Molluscum contagiosum* virus contains an IL-18 binding protein gene that it apparently acquire from humans. This blocks the host's initial effective Th-one immune response against the virus by reducing local IFN-gamma production [3],also IL-18, a recently described member of the IL-1 cytokine

super family ,is currently recognized as an important regulator of innate and adaptive immune responses. Furthermore, this little review will describe the basic biology of IL-18 and thereafter address its potential effector and regulatory role in several human disease states including autoimmunity and infection such as MCV infection. IL-18, formerly is known as interferon-gamma (IFN-γ)- inducing factor [5]. The study of the immune status of patients infected with the *Molluscum contagiosum* virus is the first study whether in Diyala governorates or in Iraq, so the present study aims at assessing the immune status of patients infected with *molluscum contagiosum* through the measurement of the level of immunoglobulin (IgG,IgM) by the radial immune diffusion assay and measuring the level of interleukin 18 by Elisa assay which immune responses are key for the eventual resolution of MC .

Material and Methods:

The present study is conducted during 1st November 2013 to 30 April 2014 in outpatient clinic of Baquba Teaching Hospital. There are 75 patients, diagnosed with clinical lesions of MCV on different areas of the body, whose age is ranged between 2-50 years including 40(53.3%) males and 35(46.7%) females. The study includes 15 healthy persons age between 2-50 years. Five milliliter of venous blood are taken from each patient by vein-puncture under the aseptic technique by disposable syringe, likewise from the control individuals .The blood is collected separately in plane tube with no anticoagulant, left to clot at room temperature then centrifuged and the serum is collected in two separated tubes and stored at (-20ºC) until used for investigation.

Human Interleukin 18 Receptor 1 (IL18 R1) Elisa Kit

Enzyme linked immunosorbent assay (ELISA) was used in determining the level of IL18 R1

Principle of the Test

In the first step , the micro titer plate provided in the kit has been pre-coated with an antibody specific to IL18R1.Standards or sample are then added to the appropriate micro titer plate wells a biotin –conjugated antibody preparation specific to IL18R1.Subsequently, avid in conjugated to Horseradish peroxidase (HRP) is added to each micro plate well and incubated .Next, tetramethylbinzidine (TMB) substrate solution is added, only those wells that contain IL18R1 ,biotin–conjugated antibody and enzyme-conjugated A vidin exhibit a change in color. The enzyme-substrate reaction is terminated by the addition of sulphuric acid solution and the color change is measured spectrophotometrically at a wavelength of 450nm± 10nm using Elisa reader. Finally, the concentration of IL18R1 in the samples is determined by comparing of the samples to the standard curve constructed from standard [6]. Moreover, the detailed procedure is carried out as suggested by the leaflet supplied with the test kit Mybiosource(U.S.A).

IgG and IgM Level in the Serum :

The IgG and IgM levels in serum are detected by single radial diffusion assay (SRIA). Principle of the test includes**:** The concentration of IgG and IgM is measured by a single radial immunodiffusion (SRID) method in which equal volumes of reference sera and test samples are added to wells in agarose containing mono specific antisera. After that, the sample diffuses radially through this gel and the substance being assayed form a precipitin ring with the mono specific antisera. Ring diameters are measured

and a reference curve is constructed on a graph paper. Unknown concentration is determined form the references standard curve [7, 8]. The detailed procedure is carried out as suggested by the leaflet supplied with the test kit Bussero (Milan) ITALY

Statistical Analysis: Data Analysis are computer aided and the statistical analysis is done by using SPSS version 20 computer software. Also, frequency distribution and percentage for the selected variable were done. The independent t- test is used and the P-Value (less than 0.05) is considered as the level of significance [9].

Results:

Demographic Data; Descriptive Statistics of Age in Both Groups

The mean ages± SD of patients are 26.92± 16.1 years (range from 2-53 years), 40 (53.3%) patients are males and 35 (46.7%) females with male to female ratio 1:1.14. Twenty (26.7%) of patients are equal or less than 16 years, 24 (32%) from 17-30 years, 19 (25.3%) from 31-45 years and 12 (16%) above 45 years (Tables 1 and 2). The mean ages of the control are 26.6±15.4 years (range from 2-50 years), 8 (53.3%) of them are males and 7 (46.7%) females with female to male ratio 1:1.14. Four (26.7%) of the control are equal or less than 16 years, 5 (33.3%) from 17-30 years, 3 (20%) from 31-45 years and 3 (20%) above 45 years (Tables 1 and 2).

Table -1: Descriptive Statistics of Age in Both Groups.

	No.	Minimum	Maximum	Mean	SD
Patients	75	2	53	26.92	16.105
Control	15	2	50	26.60	15.403

Table -2: Distribution of Age in Both Groups.

Age Group	Patients		Control		P value
	No.	%	No.	%	
≤ 16 years	20	26.7	4	26.7	**0.965**
17-30 years	24	32.0	5	33.3	
31-45 years	19	25.3	3	20.0	
> 45 years	12	16.0	3	20.0	
Total	75	100.0	15	100.0	

Residence of the Studied Subjects in Both Groups

Forty seven (62.7%) patients are from rural area while 28 (37.7%) are from urban. There is no statistical difference between both groups as the p value is equal to 0.567.

Table -3: Residence of the Studied Subjects in Both Groups.

Residence	patients		controls		P value
	No.	%	No.	%	
Rural	47	62.7	8	53.3	**0.567**
Urban	28	37.3	7	46.7	
Total	75	100.0	15	100.0	

Immunologic Study:-

Regarding the level of IL 18R1 in patients, the mean is 677.15±87.22 ng/ml while in the control it is 178.46±31.79 ng/ml. There is a significant statistical difference between both groups (p value= 0.0001) as patients with MC have a high level of IL18R1 than the control (Table -4, Table -5). The mean±SD of IgM in patients is 1946.6±825.6 mg/dl while in the control it is 140.1±68.7mg/dl. All patients have high IgM level and all the control have normal values. This result is highly significant which indicates that the patient with MC has a higher level of IgM than the control (p value= 0.0001) (Table -4, Table -5). By contrast, patients have lower levels of IgG than the control. The mean±SD of IgG in patients is 221.9±96.7 while in the control it is 1229.9±299.7. All patients have low IgG level and all the control have normal values. This

result is highly significant (p value= 0.0001).

Table -4: Levels of IL18R1, IgM and IgG in Both Groups.

		Min.	Max.	Mean	SD	Total No.
IL 18R1	Patients	153.70	5000.30	677.15	87.22	75
	control	110.80	220.30	178.46	31.79	15
IgM	Patients	773.4	2913.6	1946.6	825.6	75
	control	80.1	275.4	140.1	68.7	15
IgG	Patients	57.7	388.9	221.9	96.7	75
	control	810.4	1777.7	1229.9	299.7	15

Table -5:Values of IL18R1, IgM and IgG in Both Groups

		Patients		Control		P value
		No	%	No	%	
IL 18R1	Normal	44	58.7%	15	100.0%	0.0001
	High	31	41.3%	0	0.0%	
IgG	Low	75	100.0%	0	0.0%	0.0001
	Normal	0	0.0%	15	100.0%	
IgM	Normal	0	0.0%	15	100.0%	0.0001
	High	75	100.0%	0	0.0%	

Values of IL18R1, IgM and IgG Regarding Gender in Patients:-

The gender has no statistical relationship to the level of IL 18R1, IgM and IgG (Table-6).

Table -6:Values of IL18R1, IgM and IgG Regarding Gender in Patients

		Male		Female		Total		P value
		No	%	No	%	No	%	
IL 18R1	Normal	22	51.2%	21	48.8%	43	100.0%	#0.662
	High	18	56.2%	14	43.8%	32	100.0%	
IgM	High	40	53.3%	35	46.7%	75	100.0%	-
IgG	Low	40	53.3%	35	46.7%	75	100.0%	-

#pvalue calculated by Pearson Chi square.
*pvalue calculated by Fisher exact test

What is more the age of patients with MC has no significant relationship with the level of IgG and IgM, while it has a significant relationship with the level of IL 18R1 (p value= 0.038) as 71.9% of patients with high levels of IL18R1 are less than 30 years (Table -7).

Table -7: Values of IL18R1, IgM and IgG Regarding Age of Patients Group

		<= 16 years		17-30 years		31-45 years		> 45 years		Total		P value
		No	%	No	%	No	%	No	%	No	%	
IL-18R1	Normal	8	18.6%	13	30.2%	16	37.2%	6	14.0%	43	100 %	0.038
	High	12	37.5%	11	34.4%	3	9.4%	6	18.8%	32	100 %	
IgG	Low	20	26.7%	24	32.0%	19	25.3%	12	16.0%	75	100 %	-
IgM	High	20	26.7%	24	32.0%	19	25.3%	12	16.0%	75	100 %	-

The residence has no significant relationship with the level ofIL18R1, IgM and IgG as p values were > 0.05 (Table -8).

Table -8 :Values of IL18R1, IgM and IgG Regarding Patients Residence

		Rural		Urban		Total		P value
		No	%	No	%	No	%	
IL 18R1	Normal	24	55.8%	19	44.2%	43	100.0%	0.155
	High	23	71.9%	9	28.1%	32	100.0%	
IgM	High	47	62.7%	28	37.3%	75	100.0%	-
IgG	Low	47	62.7%	28	37.3%	75	100.0%	-

Discussion

The present study reveals that most of patients are found in ages ranging between (17-30) years. This result might agree with the study done by [10], who reports that most of the patients of the ages ranging between (11- 30) years, whereas it disagree with the study done by [11] where the range between (31-40) years. This difference in results may be due to the difference in social living levels. The present study shows that male to female ratio is 1:1.14. as it resembled to most

studies in the world, where in study done by [12] the male to female ratio was 1:6.1. The same result of 1:1.6 is obtained from the study done by [13] .The other study reported by [14] the male to female ratio is 1:4.2. IL-18R1 increases in the serum of 41.3% of infected patients which could be assigned to the critical role of IL-18R1 in defense against virus infections and provides a mechanism for evasion of the immune system by MCV [15]. These results are in concurrence with the study done by [16] who has noted an increased level of IL-18R1 in the serum of patients with molluscum contagiosum which is between (20-40%) higher level. Another study also demonstrates an increased levels of IL-18R1 in patients with molluscum contagiosum [17]. When IL-18 is first described, it is as an IFN-gamma—inducing factor during endotoxemia in mice preconditioned with a prior injection of heat-killed P. acnes, a known stimulator of the reticuloendothelial system, particularly of Kupffer cells in the liver. Because of its property to induce IFN-gamma, interleukin -18 is by default a member of the Th1-inducing family of cytokines (IFN-γ, IL-2, IL-10 , IL-12, and IL-15). However, there is also are indication that IL-18 plays a role in hypersensitivity reactions, a characteristic of T-helper 2 responses. Indeed, the biologic activities of IL-18 are clearly related to host response to virus infection (MCV) [18] .Like all interleukin responses to infections, there are 2 sides to the coin, IL-18 protects the host by its capacity to induce IFN-gamma and other immune-stimulatory cytokines in a non-specific fashion (commonly called "innate" immunity), which assists the immune system in a specific T and B cell—mediated response (commonly called "adaptive" immunity).Moreover , one can conclude that some of the pathologic significances of infection are, in part, mediated by IL-18 in somewhat the same fashion as mediated by IL-1 and TNF-alpha. These are clearly harmful to the host and are the targets of therapeutic intervention [19]. Through the development of the innate immune response, cytokines and cytokine receptors evolved early. Several mechanisms are intrinsic to the capacity of a cytokine to cause inflammation. These are identical to those that assist the host in fighting the infection. In the case of interleukin -18, the IFN-gamma—inducing property and the induction of endothelial adhesion molecules facilitate the containment and killing of the invading microbe. For example, IL-18 increases the expression of some adhesion molecules such as ICAM-1 [19, 20] and VCAM-1 [20,21], which facilitate the migration of neutrophils and lymphocytes in containing a nidus of infection. Migration of neutrophils from the vascular compartment into the tissue spaces is also a primary process in inflammatory diseases. The capacity of interferon-gamma to increase the synthesis of inducible nitric oxzide (NO) is essential for killing organisms (intracellular). In this regard, IL-18 as an IFN-γ-inducing factor serves a key role in controlling infections due to molluscum contagiosum ,Salmonella, Cryptococcus, Toxoplasma, Candida, and Mycobacterium organisms, since the production of nitric oxzide is important for intracellular killing. Earlier studies show that MCV encodes a family of proteins with homology to mammalian interleukin -18 binding proteins. Interleukin -18 is a pro-inflammatory cytokine that induces synthesis of interferon-gamma, activates natural killer cells, and is essential for a T-lymphocyte helper type 1 response. The reliance of MCV on IL-18 inhibitors supports another

evidence that IL-18 is important for IFN-gamma induction by microbial organisms and viruses. Moreover, inhibition of IFN-gamma alone would not block the induction of the other cytokines by IL-18. Therefore, targeting IL-18, MCV can prevent the cascade of downstream effects that follow the activation of the IL-18 receptor. [15,22]. IL-18 performance a major role in the production of interferon-gamma from T-cells and natural killer cells [23] .The level of IgG and IgM, among MC patients , It has been shown that all patients with MC have low levels of IgG and high levels of IgM which indicates acute infections [24] and there results are consistent with many reports [25, 26, 27] ,in addition to the finding of this study in contrast to the study done by [28] where IgG is high and IgM is low. The immunoglobulins (humoral immunity) plays an important role in the body's defense against molluscum infection. Most adults are resistant to MCV infection because they have developed IgG antibodies against the viral antigen. However, patients with weakened cellular immunity, such as in HIV infection or post-transplant immunosuppression, are more likely to develop widespread infections that are solid to treat. It has been reported that up to 24 % of patients with molluscum have a concomitant diagnosis of atopic dermatitis, and these children also experience more difficulty in clearance. While it is accurate that immunosuppressed patients are more likely to develop more severe *Molluscum Contagious* virus infections, recent data have shown that contrary to common belief, the prevalence of immunosupression among children with molluscum contagiosum is low [29]. In conclusion :The IL-18R1 is increased in the serum of 41.3% of infected patients ; all patients with MC have low levels of IgG and high levels of IgM, finally most of the patients are found in ages ranging between 17-30 years.

References:

[1]Bateman, F. 1953 .*Molluscum Contagiosum* In : Shelley WB and Crissey JT, Editors. Classics In Dermatology. Charles C Thomas:. useful summary of the poxviruses that can zoonotically infect man, which indicates which of these infections are clinically important. springfield (il): p. 20.

[2]Billstein, SA. and Mattaliano, VJ. 1990 .The "Nuisance" Sexually Transmitted Diseases: *Molluscum Contagiosum*, Scabies, And Crab Lice. *Med Clin North Am*. 74: 1487-1505.

[3]William, D. J, Timothy, G. B. and Dirk, M.E. 2012. Andrews' Diseases of The skin clinical Dermatology - 11 th ed:1-910.

[4]Dohil, M.A.; Lin, P.; Lee, J.; Lucky, A.W.; Paller, A.S. and Eichenfield, L.F. 2006. The epidemiology of molluscum contagiosum in children. *J AmAcad Dermatol*. 54:47-54.

[5]Nakamura, K., Okamura, H., Wada, M., Nagata, K.and Tamura, T. 1989. Endotoxin-induced serum factor that stimulates gamma interferon production. *Infect. Immun*.57:590–595.

[6]Mallat, Z, Corbas, A, and Scoazec, A.2001.Expression of interleukin-18 in human atherosclerotic plaque and relation to plaque instability .Circulation 104:1598-1603.

[7]Mancini, G.; Carbonara, A.O. and Heremans, J. F. 1965. Immunochemical quantitation of antigens by single radial immune diffusion. *Immunochemis.* 2 (3):235–254

[8]Barth, W.F.; McLaughlin, C.L. and Fahey, J. L. 1965. The immunoglobulins of mice. VI.

Response to immunization. *J Immunol.* 95 (5):781–790

[9]Nisi, A.D. 2004. Satistiscal analysis in medical research 2ndedition, 22: 21–30.

[10] Kuchabal D.S. Kuchabal, B. Siddaramappa, P.S.M. Katti and P.V. Patil. 2011. *Molluscum Contagiosum* a clinical and epidemiological study. *Internet J. Of Dermato.* 8 : 2

[11] Chandrshekar, L.; Devinder, M. and Telanseri, J. 2002. Clinical profile *of molluscum contagiosum* in children versus adults .*Dermato. online J.* 9 (5): 1

[12] Skuchabal. D.; Kuchabal, B.; Siddaramappa ,p. and Katti, P. 2010 . *Molluscum contagiosum* a clinical and epidemiological study .*The internet J.of Dermat.* 8(2) :3

[13] Laxmisha,C, Thappa,D. M. and Jaisankar, T.J. 2003. Clinical profile of *molluscum contagiosum i*n children versus adults. *Dermatol Online J.* 9(5):1

[14] Fayemiwo, O.A.; Adesina, J.O.; Akinyemi,, G.N.; Odaibo, T.O.; Omikunle, B. and Adewole, I.F. 2014 .*Molluscum contagiosum* virus infection amongst plwha in ibadan, Nigeri .J.home.15(1):8-13

[15] Brian, K.; Xiangzhi, M.; Yongchao, L.i; Yan, X., and Junpeng, D. 2008. Structural basis for antagonism of human interleukin 18 by poxvirus interleukin 18-binding protein. *J.PNAS.* 105: 20711-20715.

[16] Yan, X. and Bernard, M, 2003. *Molluscum Contagiosum* Virus Interleukin-18 (IL-18) Binding Protein Is Secreted as a Full-Length Form That Binds Cell Surface Glycosaminoglycans through the C-Terminal Tail and a Furin-Cleaved Form with Only the IL-18 Binding Domain.*J.Viral*.4(77):2623-2630.

[17] Yan, X and Bernard, M. 1999 . IL-18 binding and inhibition of interferon γ induction by human poxvirus-encoded proteins. *J. PNAS*. 20(96):11537-11542.

[18] Nakanishi, K.; Yoshimoto, T.; Tsutsui, H. and Okamura, H. 2001. Interleukin-18 is a unique cytokine that stimulates both Th1 and Th2 responses depending on its cytokine milieu. Cytokine Growth. *Annu. Rev. Immunol.*19**:**423-474.

[19] Kohka, H.; Yoshino, T.; Iwagaki, H.; Sakuma, I.; Tanimoto, T.; Matsuo, Y.; Kurimoto, M.; Orita, K.; Akagi, T. and Tanaka, N. 1998. Interleukin-18/interferongamma-inducing factor, a novel cytokine, up-regulates ICAM-1 (CD54) expression in KG-1 cells. *J Leukoc Biol*. 64:519-27

[20] Morel, J.C.; Park, C.C.; Woods, J.M. and Koch, A.E. 2001. A novel role for interleukin-18 in adhesion molecule induction through NFκB and phosphatidylinositol (PI) 3-κinase—dependent signal transduction pathways. *J. Biol. Chem* . 276: 69-75.

[21] Vidal-Vanaclocha, F.; Fantuzzi, G,.; Mendoza, L.; Fuentes,k.; Anasagasti,J. Martín, C. Walsh, L. R ;Soo-Hyun, K. ;Charles, A. and Dinarello,S. 2000. IL-18 regulates IL-1β—dependent hepatic melanoma metastasis via vascular cell adhesion molecule-1. *Proc Natl Acad Sci.* 97:734-9.

[22] Yan, X. and Bernard, M. 2001. Correspondence of the Functional Epitopes of Poxvirus and Human Interleukin-18-binding Proteins. *J. of virol*. 75:9947–9954.

[23] Dinarello, C.A. 1999. IL-18: a Th1-inducing, proinflammatory cytokine and new member of the IL-1 family. *J. Allergy Clin. Immunol.* 103:11–24

[24] Sheila, C. D.; Stephanie, A. S. and Michael J. C. 2011. National

Prevalence Estimates for Cytomegalovirus IgM and IgG Avidity and Association between High IgM Antibody Titer and Low IgG Avidity. *Clin Vaccine Immunol*. 18: 1895–1899.

[25] Mayumi, M.; Yamaoka, K.;Tsutsui, T.; Mizue, H.; Doi, A.; Matsuyama, M.; Ito, S,.; Shinomiya, K. and Mikawa, H. 1986 . Selective immunoglobulin M deficiency associated with disseminated molluscumcontagiosum. *Eur J Pediatr*. 145(1-2):99-103.

[26] Sara, S. and Fatih, K. 2006. Interferon- Treatment of *Molluscum Contagiosum* in a Patient With hyperimmunoglobulin E Syndrome. *Pediatrics* 117:e1253

[27] Vozmediano, J. M.; Manrique, A.; Petraglia, S.; Romero, M.A. and Nieto, I. 1996. Giant molluscum-contagiosum in *AIDS*. *Int J Dermatol*. 35(1):45-7.

[28] Iffat, H.; Anwar, R.; Safiya, B. and Atiya, y. 2014. Aninteresting Cas of GLANT Molluscum with FlORID vul GARIS in an immune competent patient .*Current Issue* 5(3):282-284.

[29] Watanabe, T. Nakamura, K.; Wakugawa, M.; Kato, A.; Nagai, Y. Shioda, T. Iwamoto, A. and Tamaki, K. 2000.Antibodies to *molluscum contagiosum* virus in the general population and susceptible patients. *Arch Dermatol.* 136(12):1518-1522.

تقييم المناعة الخلطية والانترلوكين -18 المستقبل الاول لدى بعض المرضى المصابين بفيروس المليساء المعدية

محمد عبد الدايم صالح

قسم علوم الحياة ، كلية العلوم ، جامعة ديالى ، العراق

الخلاصة:

المليساء المعدية مرض يسببه فيروس (MCV) الذي ينتمي لعائلة poxvirus .المليساء المعدية (MC) مرض غير قاتل وشائع في جميع أنحاء العالم ،قد يشترك مع أورام المليساء التهابات قليله . هدفت الدراسة إلى تقييم الحالة المناعية للمرضى المصابين بفيروس المليساء المعدي من خلال استخدام العوامل او المؤشرات المناعية. والتي تتضمن قياس المناعة الخلطية وبالتحديد قياس مستوى الغلوبيلينات المناعية IgG و IgM من خلال استخدام فحص الانتشار المناعي المفرد (RIA) وقياس مستوى مستقبل الانترليوكين 18 بواسطة فحص الاليزا . اجريت الدراسة للفترة الممتدة من تشرين الثاني 2013 الى نيسان 2014 .شخصت الإصابة في (75) مريض بفيروس المليساء المعدي في مناطق مختلفة من الجسم ،تراوحت أعمار المرضى بين(2-50سنة) ،تضمنت40 (53.3%) مريض من الذكور و35 (46.7%) من الإناث وقد أخذت (15)عينة من الأصحاء وبمعدل اعمار 2-50سنة . ان مستوى مستقبل الانترليوكين 18 مرتفع معنوي في المرضى المصابين بفيروس المليساء المعدي حيث كانت (677.15±874.22) نانوغرام / ملليلتر مقارنة في الأصحاء حيث كانت (178.46±31.79) نانوغرام / ملليلتر ،حيث ان هناك فرق إحصائي معنوي بين كلا المجموعتين ، حيث وجد ارتفاع مستوى مستقبل الانترليوكين للمرضى مقارنة مع الأصحاء.وبينت النتائج ان مستوى الامينوغلوبيولين (IgM) في المرضى كانت (1946.6±825.6) ملغم / ديسيلتر. بينما في الأصحاء كانت (140.1±68.7) ملغم / ديسيلتر يعني وجود فرق إحصائي معنوي حيث وجد ارتفاع في مستوى الامينوغلوبيولين (IgM) للمرضى بالمقارنة مع الأصحاء وبالعكس بينت النتائج انخفاض في مستوى الامينوغلوبيولين (IgG) حيث وجد إن مستوى (IgG) في المرضى كانت (221.9±96.7) ملغم / ديسيلتر بينما في الأصحاء كانت(1229.9±299) ملغم / ديسيلتر، واخيرا لا يوجد فرق احصائي معنوي بين المرضى سواء الساكنين في الحضر او الريف .

الكلمات المفتاحية: داء المليساء المعدية ،انترليوكين 18،الامينوغلوبيولين.