Determination of IL-8&inflammatory indicators (ESR,CRP) in Iraqi Multiple Sclerosis Patients

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Abstract

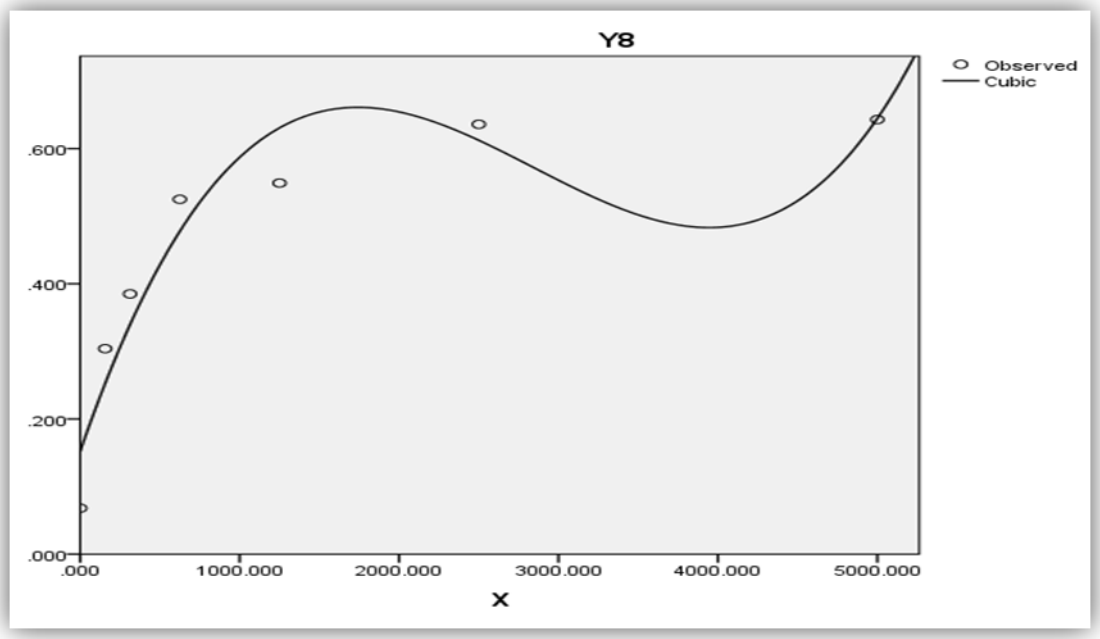
Multiple sclerosis (MS) is an inflammatory condition that affect the central nervous system (CSN) causing neurological dysfunction, the present study aimed to evaluate the role of Interleukin 8 (IL-8) and some inflammatory indicators: Erythrocytes Sedimentation Rate (ESR) and C Reactive Protein (CRP). Forty-five Iraqi MS patient and 15 apparently healthy control individuals were enroll in this study, the patients were attended the multiple sclerosis unit at consulting clinic of Baghdad teaching hospital at medicine city. During the period august – December 2014. The patient were clinically diagnosed by the consultant medical staff at the clinic, which was based on a clinical examination, magnetic resonance image (MRI), immunological tests, and under the supervision of this staff. The patients were divide into: (Untreated patients, Patients treated with methylprednisolone, Patients treated with interferon beta).

- It has been found that IL-8 serum level was decreased significantly ($P \leq 0.026$) in untreated patients (0.003 ± 0.001)), and anon significant decrease in both patients treated with methylprednisolone (0.010 ± 0.003) and patients treated with Interferon beta (0.014 ± 0.004) compared to control (0.016 ± 0.006).
- It has been found that the ESR was) a anon significant increase in both untreated patients (22.77 ± 3.19) and patients treated with methylprednisolone (22.73 ± 2.43) and significant increases ($P \leq 0.034$) in patients treated with Interferon beta (28.73 ± 4.80) compared to control (17.40 ± 3.88).
- It has been found that CRP serum level was increased non significantly in all patients. untreated patients (0.34 ± 0.22), patients treated with

methylprednisolone (0.20 ± 0.14) and patients treated with Interferon beta (0.26 ± 0.08) compared to control (0.08 ± 0.05).

Introduction:

- **(MS)**: is one of the world's most common neurological disorders, it is the leading cause of non-traumatic disability in young adults arouned the wolrd, it is an inflammatory immune mediate demyelinating disorder that attak the centr (CNS) --the brain and spinal cord--. Its caused by tolerance failure to self-antigens in a genetically susceptible individual, it is a chronic disease characterized by inflammation, demyelination, gliosis (scarring), and neuronal loss. It involves an immune-mediated process in which an abnormal response of the body's immune system is directed against (CNS). (Hauser and Josephson , 2013)
- **IL-8 (CXCL 8) :** is a proinflammatory chemokine (Zeilhofer and Schorr, 2000). It is secreted from arrange of cell types including leukocytes, fibroblasts, endothelial cells and malignant cancer cells, (Nazzal Saakhi, 2014). But it is originally identified as aneutrophil chemoattractant, which makes an important contribution to the induction of innate immunity, because it effects on neutrophil chemotaxis and activation (Zeilhofer and Schorr, 2000)
- **(ESR):** It is the rate of a red blood cells column sediment (fall) in plasma in a period of one hour.(internet-1, 2015) .its raised with increased plasma viscosity, in the plasma the concentration of two proteins (fibrinogen and globulins) is the major determinant of viscosity, ESR and plasma viscosity used to indicates acute phase response since its raised in inflammation .ESR concedared anon specific
- **(CRP) :** is a protein ,synthesized by the liver and found in blood plasma. (Pepys, 2003; Thompson, 1999) .the liver responed to certain cytokines secreated by macrophages in case of infection or tissue injury by producing arrange of serum acute-phase proteins ,CRP is the most classical and frequently measured by clinical laboratories.(Helbert, 2006) which has been used as aclinical marker of inflammation for mor than 70years. (male,2013) its level increases rapidly with in 4-6 houres and fallrapidly as well, it increases as much as 100-1000 fold during infection. (Mahon, 2006), as aside effect of the increased concentration is the aqute-phase proteins is the plasma viscosity increases and this is ofen measured in clinical labotatories as ESR. (Helbert, 2006). as meancent befor.



Materials and methods:

Materials:

- CRP kit AGAPI-Swiss
- Interleukin-8 ELISA Development kit PeproTech -USA

Laboratory methods

- ESR measured by the classical western green method
- CRP qualitative and quantative test by method suggested in kit leaflet and concentration assessed by using an equation mention in it.
- Serum Level of IL-8 was assessed by means method of sandwich ELISA ,

A standard curve is prepared from standard dilutions and human cytokine sample level is determined from a curve fitting equation. (Fig- 1)

Figure (1) standard curve of IL-8

IL-8 Formula: Y8 = 0.09+1.266X-1.026X2+0.243X3

R square = .989

Statistical Analyses

The result was analyzed using the computer program SPSS (Statistical Package for Social Sciences) version 20 Their data were given as mean ± standard error (S.E.), and differences between means were assessed by ANOVA (Analysis of Variance), followed by LSD (Least Significant Difference).

Result :

- **IL-8 estimation for MS patients and Control**

1. Control versus untreated patients

The serum level of IL-8 (0.016 ± 0.006 vs 0.003 ± 0.001) showed a decreasing in untreated patients compared to control, and the difference was significant (P ≤0.026), (Table -1),(Fig. -2)

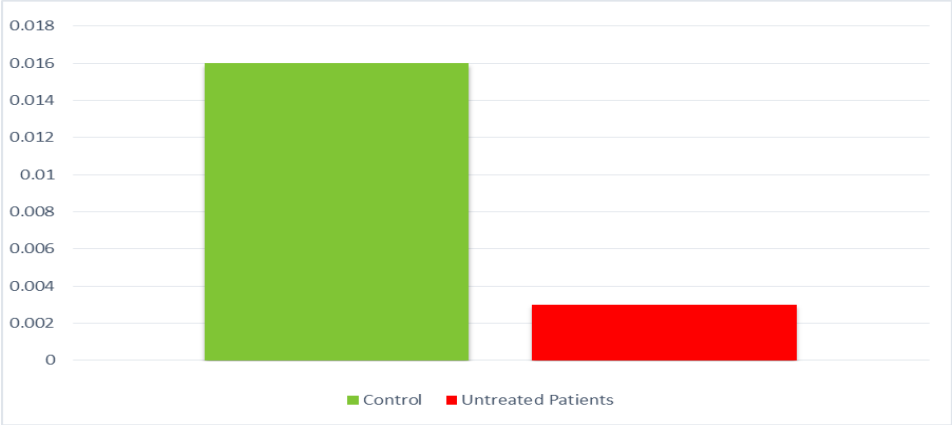


Figure (2) IL-8 in Control and untreated patients

Table (1) IL-8 serum levels in Control versus untreated patients

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Control	Untreated patients		Lower Bound	Upper Bound
IL-8	0.016 ± 0.006	0.003 ± 0.001	0.026	0.00157	0.02357

2. Control versus patients treated with methylprednisolone :

The serum level of IL-8 (0.016 ± 0.006 vs 0.010 ± 0.003), showed a decreasing in patients treated with methylprednisolone compared to control, and the differences were not significant. (Table -2), (Fig.-3)

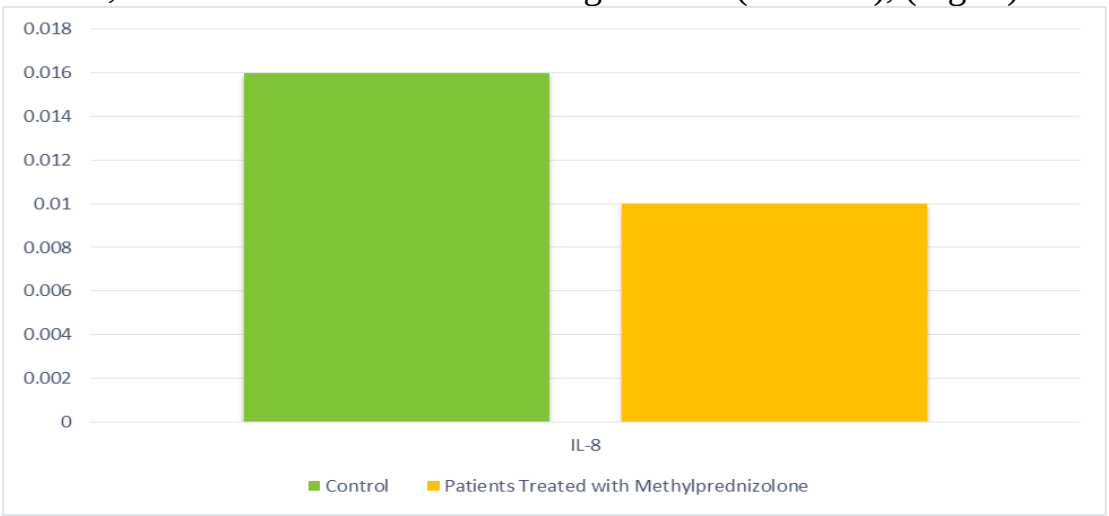


Figure (3) IL-8 serum level in Control and patients treated with methylprednisolone

Table (2) IL-8 serum level in Control and patients treated with methylprednisolone

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Control	M.S (late onset) Meth		Lower	Upper
IL-8	0.016 ± 0.006	0.010 ± 0.003	N.S.	-0.00495	0.1704

3. Control versus patients treated with Interferon beta

The serum level of IL-8 (0.016 ± 0.006 vs 0.014 ± 0.004) showed a decreasing in patients treated with Interferon beta compared to control, and the differences were not significant.(Table -3), (Fig.-4)

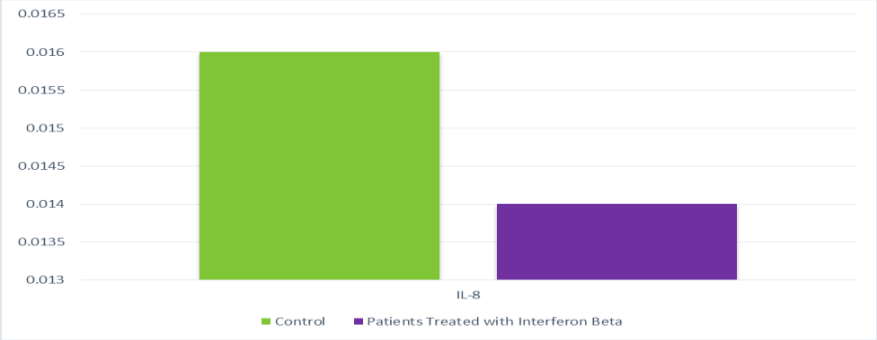


Figure (4) IL-8 serum level in Control and patients treated with interferon beta

Table (3) IL-8 serum level in Control and patients treated with interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Control	Patients Treated with Interferon Beta		Lower	Upper
IL-8	0.016 ± 0.006	0.014 ± 0.004	N.S.	-0.0933	0.01267

4. Untreated patients versus patients treated with methylprednisolone

The serum level of IL-8 (0.003 ± 0.001 vs 0.010 ± 0.003 showed a non-significant. increasing in patients treated with methylprednisolone compared to untreated patients. (Table -4), (Fig.- 5)

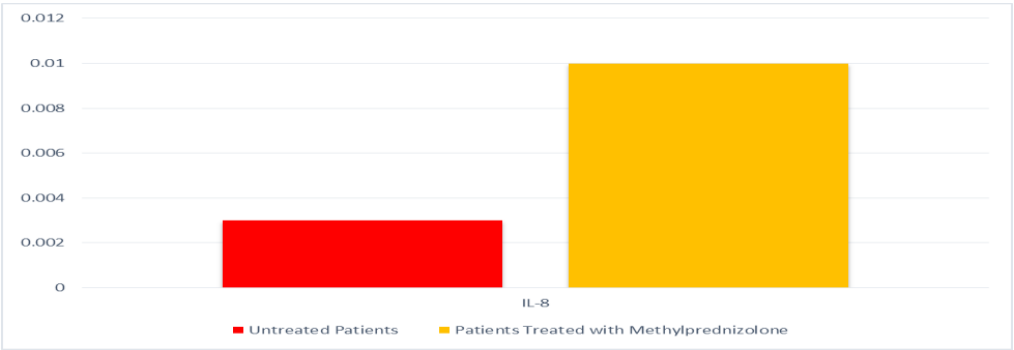


Figure (5) IL-8 serum level for Untreated patients and patients treated with methylprednisolone

Table(4) IL-8 serum level for Untreated patients and patients treated with methylprednisolone

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Untreated Patients	Patients Treated with Methylprednizolone		Lower	Upper
IL-8	0.003 ± 0.001	0.010 ± 0.003	N.S.	-0.01753	0.00447

5. Untreated patients versus patients treated with Interferon beta

The serum level of IL-8 (0.003 ± 0.001 vs 0.014 ± 0.004) showed an increasing in patients treated with Interferon beta compared to untreated patients, and the differences were not significant.(Table -5) (Fig.-6).

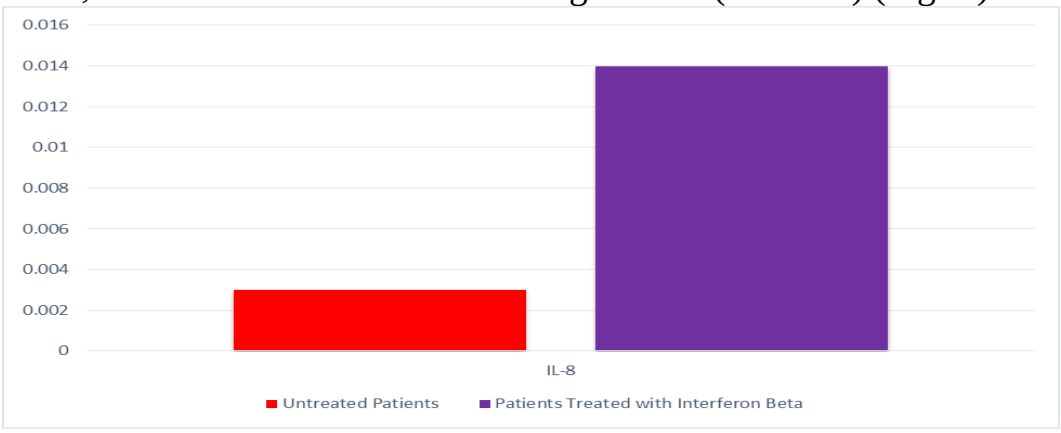


Figure (6) IL-8 serum level of Untreated patients versus patients treated with Interferon beta

Table (5) IL-8 serum level of Untreated patients versus patients treated with Interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Untreated Patients	Patients Treated with Interferon Beta		Lower	Upper
IL-8	0.003 ± 0.001	0.014 ± 0.004	N.S.	-0.02191	0.00009

6. patients treated with methylprednisolone versus patients treated with Interferon beta

The serum level of IL-8 (0.010 ± 0.003 vs 0.014 ± 0.004) showed an increasing in patients treated with Interferon beta compared to patients treated with methylprednisolone , and the differences were not significant. (Table-6), (Fig.-7)

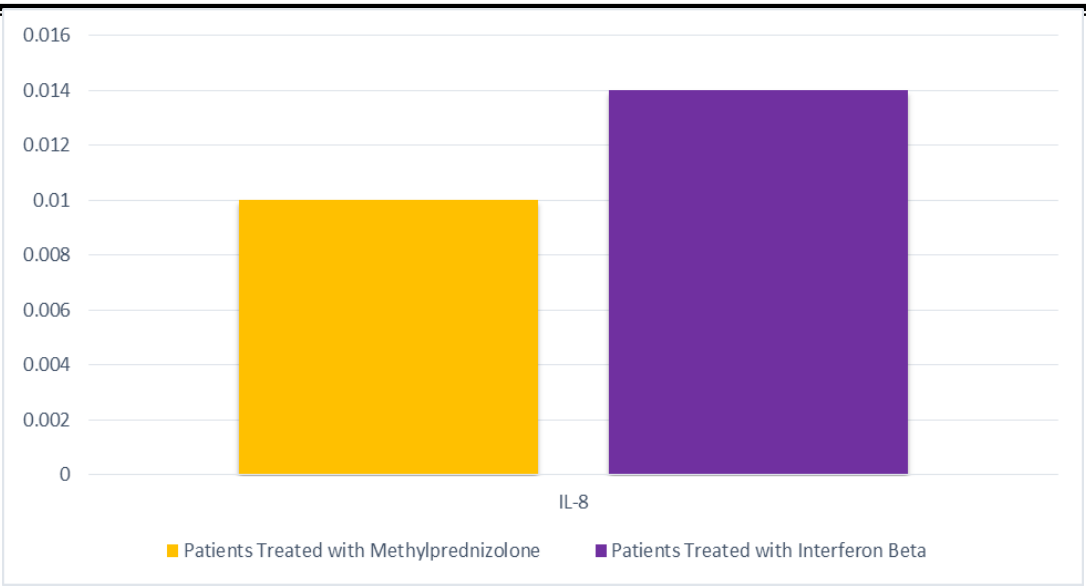


Figure (7) serum level of IL-8 in patients treated with methylprednisolone and patients treated with Interferon beta

Table (6) serum level of IL-8 in patients treated with methylprednisolone and patients treated with Interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Patients Treated with Methylprednizolone	Patients Treated with Interferon Beta		Lower	Upper
IL-8	0.010 ± 0.003	0.014 ± 0.004	N.S.	-0.01538	0.00662

4.3 ESR Estimation for MS Patients and Control

4.3.1 Control versus Untreated Patients

The ESR level (17.40 ± 3.88 vs 22.77 ± 3.19) showed an increasing in patients Untreated Patients compared to control, and the differences were not significant.

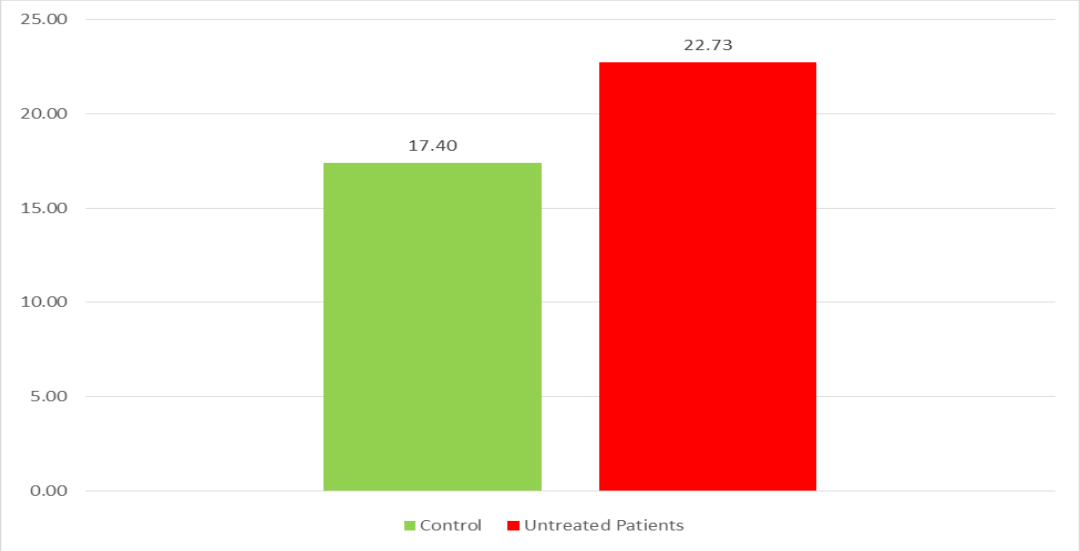


Figure (8) ESR in Control and untreated patients

Table (1) ESR in Control versus untreated patients

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Control	Untreated Patients		Lower Bound	Upper Bound
ESR	17.40 ± 3.88	22.77 ± 3.19	N.S.	-15.7896	5.0509

4.3.2 Control versus Patients Treated with Methylprednizolone

The ESR level (17.40 ± 3.88 vs 22.73 ± 2.43) showed an increasing in patients Patients Treated with Methylprednizolone compared to control, and the differences were not significant.

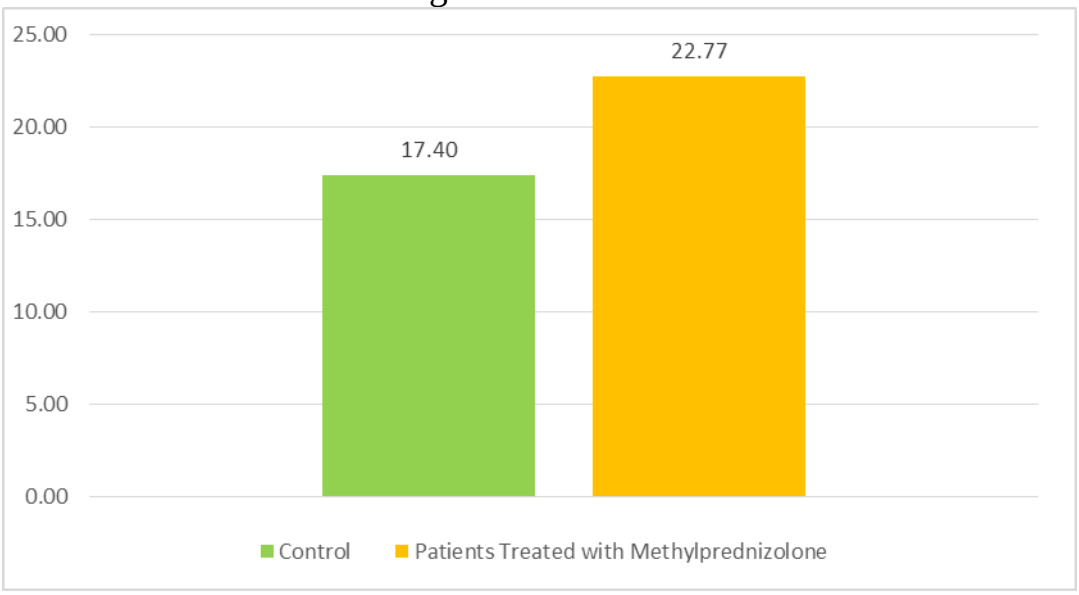


Figure (9) ESR in Control and patients treated with methylprednisolone

Table (8) IL-8 serum level in Control and patients treated with methylprednisolone

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Control	Patients Treated with Methylprednizolone		Lower	Upper
ESR	17.40 ± 3.88	22.73 ± 2.43	N.S.	-15.7469	5.0936

4.3.3 Control versus Patients Treated with Interferon Beta

The ESR level (17.40 ± 3.88 vs 28.73 ± 4.80) showed an increasing in patients Patients Treated with Interferon Beta compared to control, and the difference was significant (P ≤ 0.034).

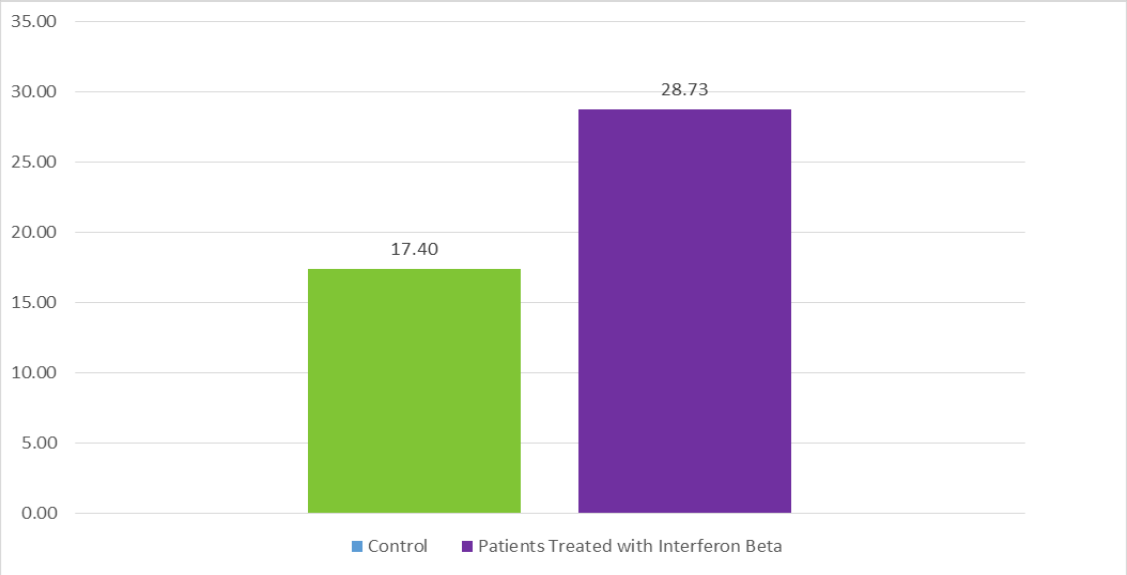


Figure (10) ESR in Control and patients treated with interferon Beta

Table (9) ESR in Control and patients treated with interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Control	Patients Treated with Interferon Beta		Lower	Upper
ESR	17.40 ± 3.88	28.73 ± 4.80	0.034	-21.7536	-0.9131

4.3.4 Untreated Patients versus Patients Treated with Methylprednisolone

The ESR level (22.77 ± 3.19 vs 22.73 ± 2.43) showed an increasing in patients Untreated Patients compared to Patients Treated with Methylprednisolone, and the difference was not significant

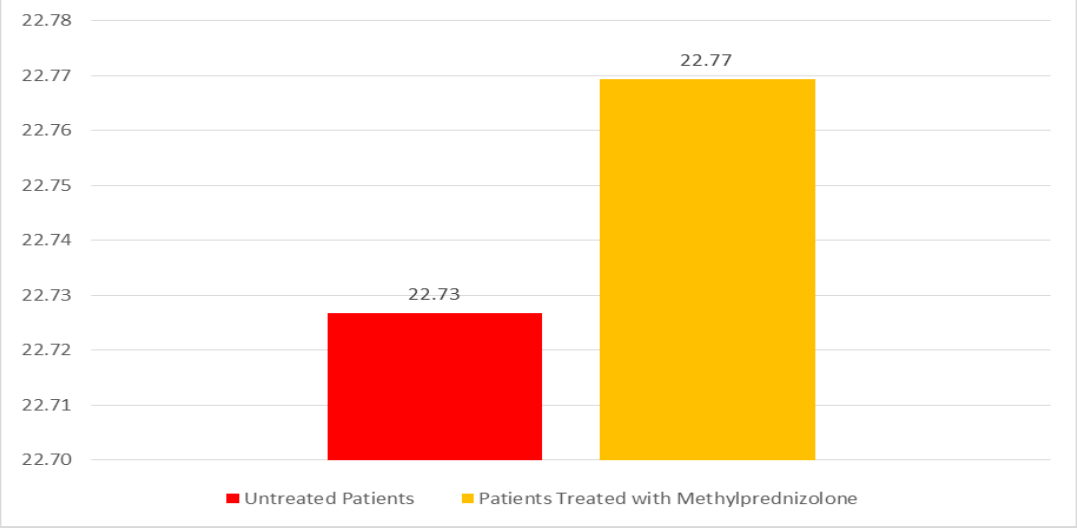


Figure (11) ESR in Untreated patients and patients treated with methylprednisolone

Table(4) ESR in Untreated patients and patients treated with methylprednisolone

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	Upper
	Untreated Patients	Patients Treated with Methylprednizolone		Lower	
ESR	22.77 ± 3.19	22.73 ± 2.43	N.S.	-10.3776	10.4629

4.3.5 Untreated Patients versus Patients Treated with Interferon Beta

The ESR level in un (22.77 ± 3.19 vs 28.73 ± 4.80) showed an increasing in patients Patients Treated with Interferon Beta compared to Untreated Patients, and the difference was not significant.

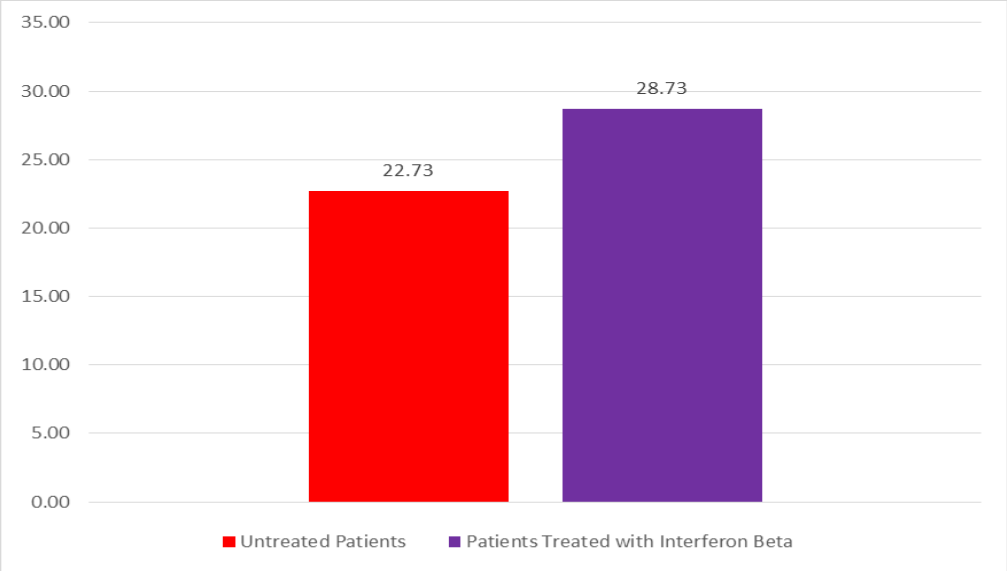


Figure (12) ESR in Untreated patients versus patients treated with Interferon beta

Table (11) ESR in Untreated patients versus patients treated with Interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	Upper
	Untreated Patients	Patients Treated with Interferon Beta		Lower	
ESR	22.77 ± 3.19	28.73 ± 4.80	N.S.	-16.3842	4.4562

4.3.6 Patients Treated with Methylprednizolone versus Patients Treated with Interferon Beta

The ESR level (22.73 ± 2.43 vs 28.73 ± 4.80) showed an increasing in patients Patients Treated with Interferon Beta compared to Patients Treated with Methylprednizolone, and the difference was not significant.

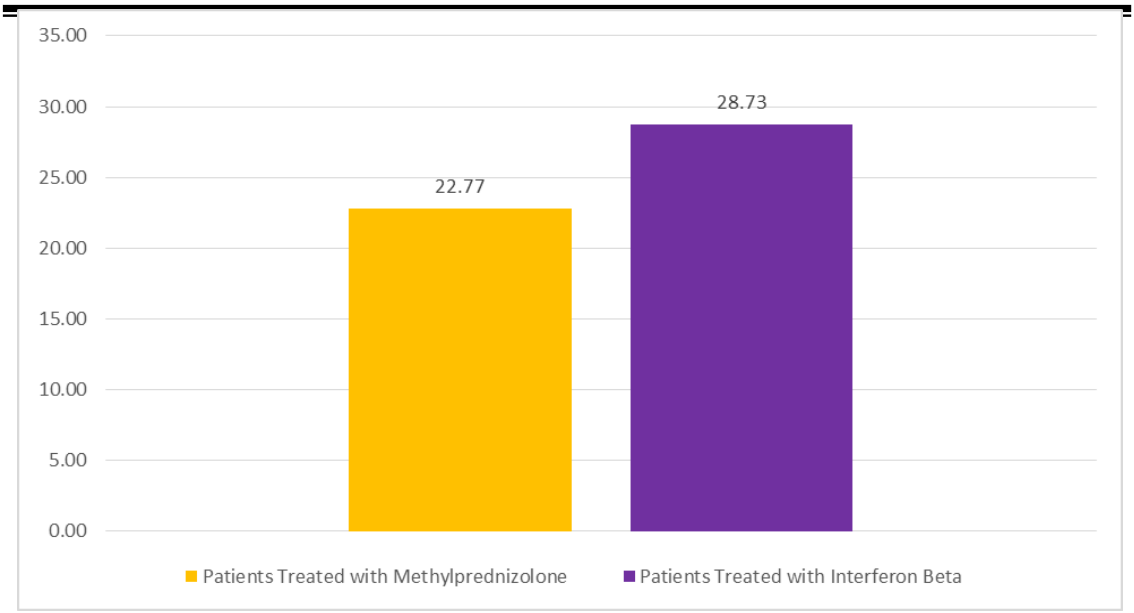


Figure (13) ERS in patients treated with methylprednisolone and patients treated with Interferon beta

Table (12) ESR in patients treated with methylprednisolone and patients treated with Interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Patients Treated with Methylprednizolone	Patients Treated with Interferon Beta		Lower	Upper
ESR	22.73 ± 2.43	28.73 ± 4.80	N.S.	-16.4269	4.4136

4.4 C.R.P Estimation for MS patients and control

4.4.1 Control versus Untreated Patients

The CRP level (0.08 ± 0.05 vs 0.34 ± 0.22) showed an increasing in patients Untreated Patients compared to control, and the differences were not significant

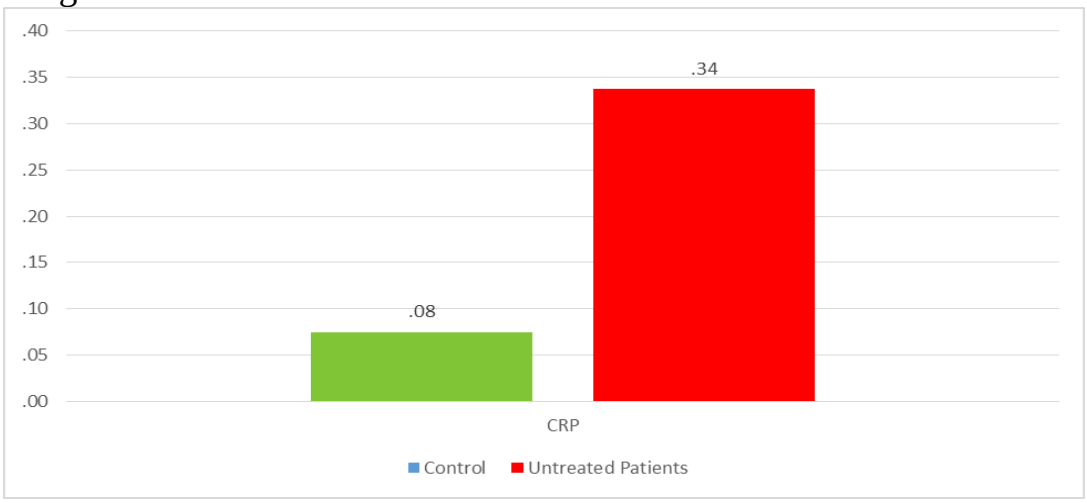


Figure (14) CRP in Control and untreated patients

Table (13) CRP in Control versus untreated patients

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Control	Untreated Patients		Lower Bound	Upper Bound
CRP	0.08 ± 0.05	0.34 ± 0.22	N.S.	-0.6471	0.1222

4.4.2 Control versus Patients Treated with Methylprednizolone

The CRP level (0.08 ± 0.05 vs 0.20 ± 0.14) showed an increasing in patients Patients Treated with Methylprednizolone compared to control, and the differences were not significant.

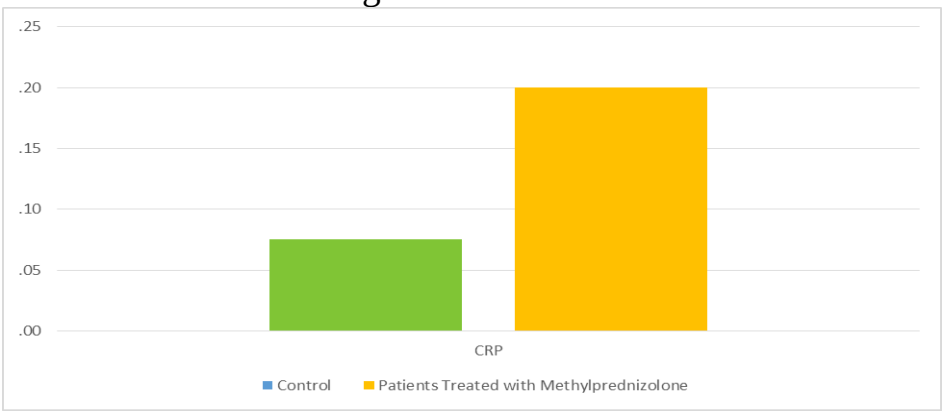


Figure (15) CRP in Control and patients treated with methylprednisolone

Table (14) CRP in Control and patients treated with methylprednisolone

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Control	Patients Treated with Methylprednizolone		Lower	Upper
CRP	0.08 ± 0.05	0.20 ± 0.14	N.S.	-0.5097	0.2597

4.4.3 Control versus Patients Treated with Interferon Beta

The CRP level (0.08 ± 0.05 vs 0.26 ± 0.08) showed an increasing in patients Patients Treated with Interferon Beta compared to control, and the differences were not significant.

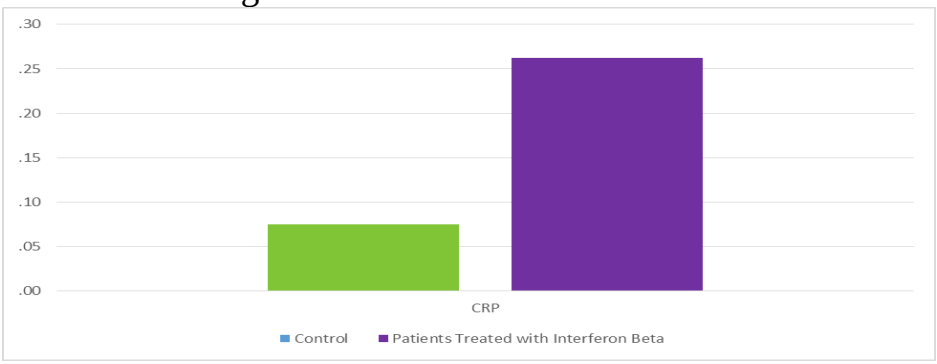


Figure (16) CRP in Control and patients treated with interferon beta

Table (15) CRP in Control and patients treated with interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	`Confidant interval	
	Control	Patients Treated with Interferon Beta		Lower	Upper
CRP	0.08 ± 0.05	0.26 ± 0.08	N.S.	-0.5721	0.1972

4.4.4 Untreated Patients versus Patients Treated with Methylprednizolone

The CRP level (0.34 ± 0.22 vs 0.20 ± 0.14) showed an increasing in patients Untreated Patients compared to Patients Treated with Methylprednizolone, and the differences were not significant.

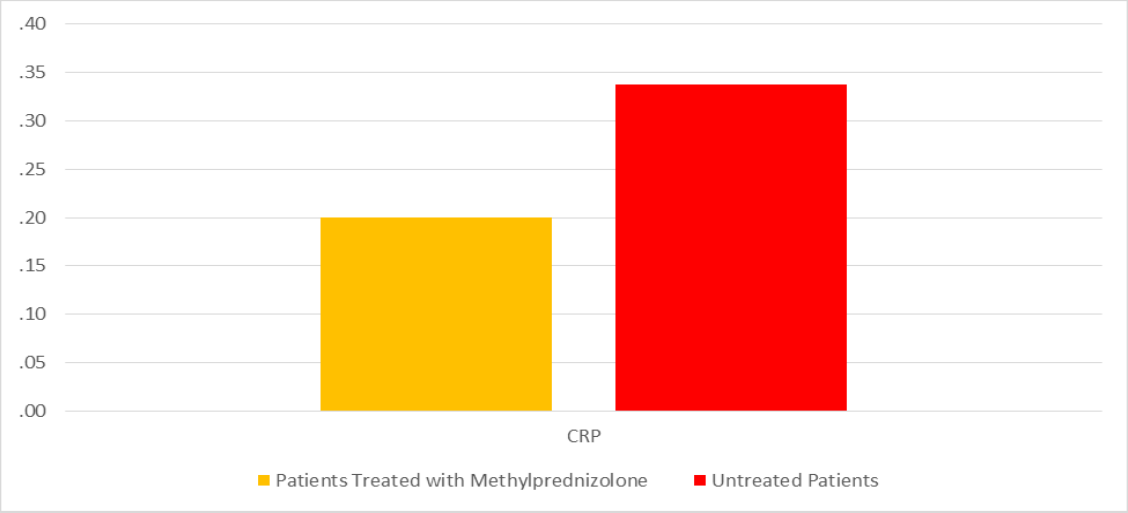


Figure (17) CRP in Untreated patients and patients treated with methylprednisolone

Table(16) CRP in Untreated patients and patients treated with methylprednisolone

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Untreated Patients	Patients Treated with Methylprednizolone		Lower	Upper
CRP	0.34 ± 0.22	0.20 ± 0.14	N.S.	-0.2472	0.5221

4.4.5 Untreated Patients versus Patients Treated with Interferon Beta

The CRP level (0.34 ± 0.22 vs 0.26 ± 0.08) showed an increasing in patients Untreated Patients compared to Patients Treated with Interferon Beta, and the differences were not significant.

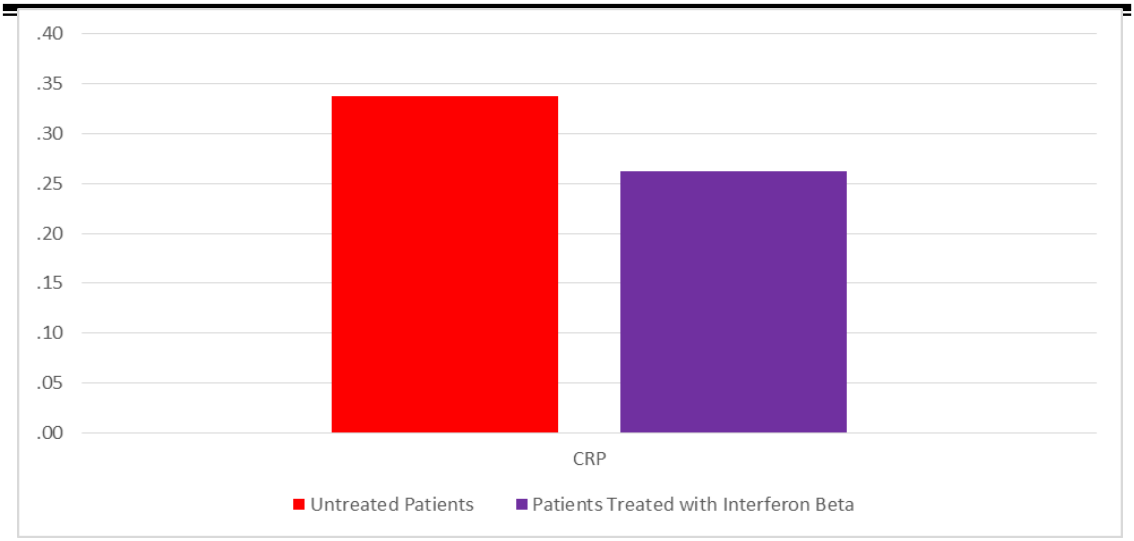


Figure (18) CRP in Untreated patients versus patients treated with Interferon beta

Table (17) CRP in Untreated patients versus patients treated with Interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Untreated Patients	Patients Treated with Interferon Beta		Lower	Upper
CRP	0.34 ± 0.22	0.26 ± 0.08	N.S.	-0.3097	0.4597

4.4.6 Patients Treated with Methylprednizolone versus Patients Treated with Interferon Beta

The CRP level (0.20 ± 0.14 vs 0.26 ± 0.08) showed an increasing in patients Patients Treated with Interferon Beta compared to Patients Treated with Methylprednizolone, and the differences were not significant.

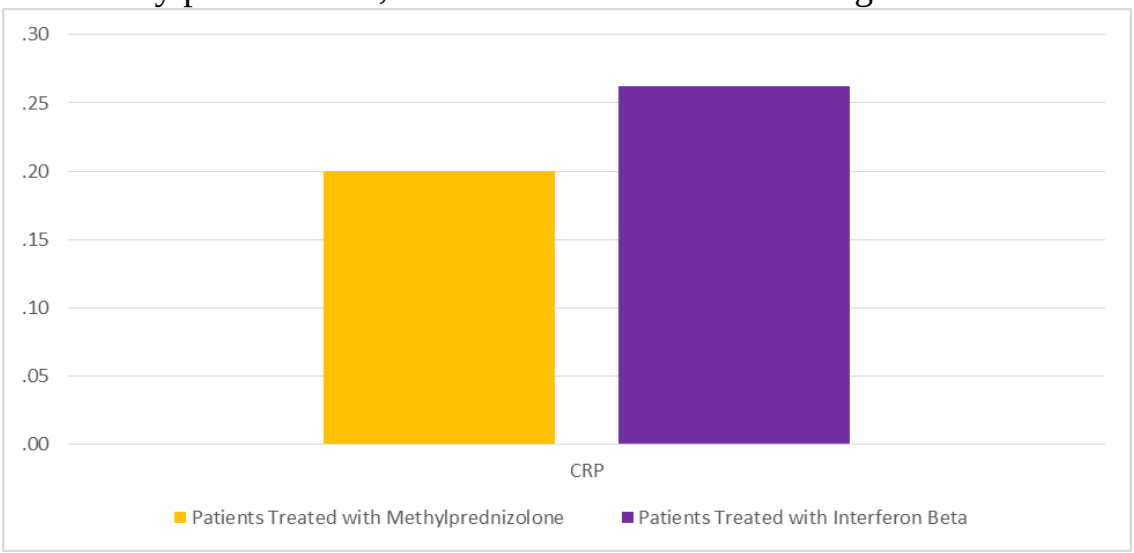


Figure (19) CRP in patients treated with methylprednisolone and patients treated with Interferon beta

Table (18) CRP in patients treated with methylprednisolone and patients treated with Interferon beta

Parameter	Mean ± S.E.		P≤ 0.05	Confidant interval	
	Patients Treated with Methylprednizolone	Patients Treated with Interferon Beta		Lower	Upper
CRP	0.20 ± 0.14	0.26 ± 0.08	N.S.	-0.4471	0.3222

Discussion :

The presented result clearly demonstrate that the:

- level of IL-8 decreases significantly in untreated patient compare to control and that disagree with (lund .2004).also serum levels of IL-8 decrease in patients treated by methyl prednisolone compared to control and that agree with (Mirowska-Guzel .2006) and that due to drug (methyl -prednisolone) effect ,which decreasing synthesis or release of pro-inflamatory cytokines such as IL-8 as a part of its action mechanism (Rudick,1999). and decrease monocytes producing IL-8 (Mirowska-Guzel et al.2006). Also serum level of IL-8 decreased in patients treated with Interferon beta ,this decreasing due to beta feron effect.).in untreated patients IL-8 should be increasing cause they are experience aqute-phase response and CRP level result support that.so why it decreased significantly? Since antigens are recognized by macrophages various receptors, leading to phagocytosis. which macrophages phagocytosis apoptotic polymorphonuclear neutrophils which lead to increases transforming growth factor-β and MCP-1 secretion and decreases IL-8 production, leading to a shift in chemokine favouring monocyte recruitment. As neutrophils are depleted from the inflammatory site, blood monocytes,in contrast, accumulate and differentiate into inflammatory macrophages, which complete phagocytosis and destruction of the injurious agents (Ryan, 1977; Melnicoff, 1989; Doherty, 1988).

  One of the factors, roduced in an inflammatory response is IL-8 which is a chemokine.Chemokines are a group of cytokines with a hemotactic and other function. Some of IL-8 produced is held in the extracultular matrix on the endothelial surface and can bind to IL-5 receptor, on the neutrophil surface. The binding of IL-8 to neutrophil achvata the neutrophil and LFA-1 change conformation and bind friendly to ICAM-1on the endothelium (wood,,2006). So this may explain the decreasing level of IL-8 in explanation. (then ware outhuz explention, where they are produced so in this disease IL-8 may wasn't produced by emdothlic alls so its's levels

Most cytokines act close to where they are produced so in this disease may IL-8 increased in CSF (local of information)(Abbas 2005).

This with regard to untraeated with methylprednisolone the decreasing may be due to drug effect (mirowska-Guzel, 2006).

Which decreasing synthesis or release of pro-inflamatory cytokines such as IL-8 as part of its action mechanisim (Rudick, 1999), and decrease monocytes producing IL-8 (Mirowska-Guzul,2006), also serum level of IL-8 decleard in patients treated with betaferon, this decreasing may due to drug effect also .

- ESR level of patients treated with betaferon is significantly increas compared to control. The other patients groups are increases compared to control, and that agree with (Palm, 1982; Abdulamir,2009) .
- CRP level in all groups of patients increases compare to control and that agree with (Sellner, 2008). The highst level go's for untreated patients because CRP is an acute-phase protein(Thompson,1999) and the patients have acut inflammation because the recently diagnoses when they hit by an attack.

Conclusion

1. It has been found that IL-8 inversely proportion with disease Action so it decrease significantly in untreated new patient the experience a cut attack, while it just decreased in treated patients which they sort of over com attacks.
2. Interferon beta do not decrease inflammation effectively in patients, by which ESR increased significantly in patient treated with interferon beta.
3. CRP showed non-significant increasing differences. in all patients compared to control.

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دراسة للوسيط الخلوي 8 ومؤشرات التهابية (CRP & ESR) لدى مرضى التصلب المتعدد العراقيين

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الخلاصة

التصلب المتعدد (MS) هو حالة التهابية تؤثر على الجهاز العصبي المركزي (CNS) مما تسبب خلل في الوظائف العصبية، وتهدف هذه الدراسة إلى تقييم دور الانترلوكين 8 (IL-8) وبعض المؤشرات الالتهابية مثل: معدل ترسيب كريات الدم الحمر (ESR)، ومعدل البروتينات الفعالة (CRP)، شملت هذه الدراسة 45 مريضاً عراقياً مصاب بتصلب المتعدد فضلاً عن 15 شخصاً سليماً (سيطرة)، جمعت عينات التصلب المتعدد من المرضى المراجعين إلى العيادات الاستشارية لمستشفى بغداد التعليمي في مدينة الطب. خلال المدة من أغسطس إلى ديسمبر 2014. تم تشخيص هذه الحالات سريريا من قبل الكادر الطبي في العيادات الاستشارية لهذه المراكز، والتي تقوم على الفحوصات السريرية، والصور بالرنين المغناطيسي (MRI)، والاختبارات المناعية، وتحت إشراف مستشارين طبيين. وتم توزيع المرضى إلى: (المرضى غير المعالجة، المرضى الذين المعالجين بالمثل برتيزيلون، والمرضى المعالجين بالانترفيرون بيتا).

اظهر مستوى الانترلوكين IL-8 في المصل انخفاض معنوي واضح عند مستوى احتمالية ($P\leq0.026$) في المرضى غير المعالجة (0.001 ± 0.003)، وانخفاض غير معنوي في كلاً من المرضى المعالجين بالمثل برتيزيلون (± 0.010)

- (0.003 والمرضى المعالجين بالانترفيرون بيتا (0.004 ± 0.014) عند المقارنة مع مجموعة السيطرة (0.006 ± 0.016).
- اظهر معدل ترسب كريات الدم الحمر (ESR) زيادة غير معنوية في كلا المرضى غير المعالجين (3.19 ± 22.77) والمرضى المعالجين بالمثل برتيزيلون (2.43 ± 22.73)

وزيادة معنوية عند مستوى احتمالية ($P \leq 0.034$) في المرضى المعالجين بالانترفيرون بيتا (28.73 ± 4.80) عند المقارنة مع مجموعة السيطرة (17.40 ± 3.88).

- اظهر مستوى البروتينات الفعالة (CRP) في المصل زيادة غير معنوية في جميع المرضى. المرضى غير المعالجين (0.22 ± 0.34)، والمرضى المعالجين بالمثل برتيزيلون (0.14 ± 0.20)، في المرضى المعالجين بالانترفيرون بيتا (0.08 ± 0.26) عند المقارنة مع مجموعة السيطرة (0.05 ± 0.08).