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Serum Soluble CD163 as a Marker of Activity in MS Patients

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Abstract

Background: Multiple sclerosis (MS) is a chronic autoimmune disease that affects the central nervous system. Microglia and macrophages have a substantial role in myelin and axonal degeneration by causing neuro-inflammatory damage. Soluble CD 163 one of the myeloid linage biomarkers, showed a better correlation with monocyte count in the CSF of MS patients.

Aim of Study: In the current case control observational study, we aimed to assess serum level of sCD163 as an immunological non-invasive marker for MS activity.

Patients and Methods: Sixty relapsing remitting multiple sclerosis (RRMS) patients were included and divided into 2 groups based on disease activity. Twenty-eight matched healthy controls were included and all subjects' serum levels of sCD163 were measured using ELISA.

Results: This study demonstrated a highly significant-difference between the whole patients compared to controls with a concomitantno statistically significant difference between the patients' groups.

Conclusion: This study emphasized the relevance of serum level of sCD 163 as a non-invasive immunological biomarker in the diagnostic panel of MS reflecting the inflammatory process rather than the activity status of the patients.

Key Words: *Multiple sclerosis – sCD163 – RRMS.*

Introduction

MULTIPLE sclerosis (MS) is a chronic autoimmune neurodegenerative disease affecting the central nervous system [1] and considered the most common cause of neurological disability in young adults [2]. Its diagnosis depends mainly on clinical picture, MRI findings and CSF oligoclonal bands (OCBs) [3]. One of the widely accepted theories of the neurodegenerative process involved in the pathogenesis of MS lesions is the interaction between mononuclear phagocytes [4], such as microglia and macrophages, and the cells of the adaptive immune system [5]. Cluster of Differentiation 163 (CD 163), a transmembrane protein expressed mainly on monocytes and macrophages, belongs to the scavenger receptor cysteine rich family class B domains that promote removal of altered-self or non-self-targets [6,7]. After shedding process of transmembrane CD 163, a soluble form is produced [8]. Many studies indicated that markers of myeloid lineage such as sCD 163 may be a useful biomarker of intrathecal microglial and macrophage activation [9] as a way to monitor disease activity and therapeutic response [10]. In this study, we investigated serum sCD163 level in RRMS patients as a noninvasive diagnostic biomarker reflecting disease activity.

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Patients and Methods

Study population:

We recruited 60 RRMS patients diagnosed according to McDonald's criteria 2017 [11], patients in remission with EDSS <3 and all patient with other neurological diseases or other chronic inflammatory diseases were excluded from the study. All cases were diagnosed in MS unit of Ain Shams University Hospital at the period between May 2020 and May 2021 divided according to the disease activity in to 2 groups (30 RRMS patients in relapse and 30 RRMS patients in remission). Twenty-eight age and sex matched healthy controls were recruited to the study collected from sampling room of Ain Shams University Hospital.

A verbal informed consent was obtained from all individuals before starting in the study. The study was approved by Institutional Ethics Committee of Medicine Ain Shams University with Assurance No. FWA 000017585.

Specimen collection:

Venous blood samples were collected aseptically from all individuals and were stored at <–80 °C till processing with rejection of any sample with hemolysis or lipemia.

Clinical and analytical methods:

All individuals in the study were subjected to full history taking, neurological examination by Expanded Disability Status Scale (EDSS) assessment and detection of sCD 163 level in serum using ELISA techniqueas instructed by the manufacturer using Human sCD 163 ELISA Kit (Catalog No: E-EL-H0036, Elabscience Biotechnology Inc®, United States, 14780 Memorial Drive, Suite 216, Houston, Texas 77079). ELISA plate reader (das®, Viale Tivoli 102-00018 Palombara Sabina, Rome, Italy) was used to measure theoptical density.

Statistical analysis:

Data were collected, revised, coded and entered to the Statistical Package for Social Science (IBM SPSS) version 23. Data was presented and suitable analysis was done according to the type of data obtained for each parameter.

Results

This case-control observational study included 60 RRMS cases (8 males and 52 females) and their ages ranged from 16-47 years old with mean of 28.80±6.99 years. The control group included 28 healthy subjects (8 males and 20 females) and their ages ranged from 14-45 years old with mean of 28.14±8.42 years. As the whole patients group and the control group were age and sex matched, there was no statistical significant difference between them as regard age and sex. Comparative statistics between both patients' groups revealed statistically highly significant differences ($p<0.01$) between both groups as regard EDSS, EDSS degree, MRI findings and number of relapses during the last year. On the other hand, no significant statistical differences were revealed between the two groups ($p>0.05$) as regard all the other parameters (Table 1).

Table (1): Statistical analysis of the cases groups as regard demographic, laboratory and clinical parameters.

Parameter Group	RRMS in relapse n=30	RRMS in remission n=30	*p*- value	Sig.
Sex:				
Female n (%)	27 (90.0)	25 (83.3)	0.448*	NS
Male n (%)	3 (10.0)	5 (16.7)		
Age (years):				
Mean ± SD	29.73±6.92	27.87±7.05	0.305*	NS
Range	16-45	16-47		
Age of disease onset (years):				
Mean ± SD	25.15±6.50	23.86±7.09	0.466**	NS
Range	14-40	14-44		
Serum level of sCD163 (ng/ml):				
Median (IQR $25^{th}-75^{th}$ percentile)	1350 (850-2400)	1000 (830-2400)	0.662≠	NS
Range	150-4000	500-5000		
Family history of MS:				
Positive family history n (%)	3 (10.0)	2 (6.7)	0.529*	NS
Negative family history n (%)	26 (86.7)	28 (93.3)		
NA n (%)	1 (3.3)			
Presenting symptoms:				
Sensory n (%)	4 (13.33)	10 (33.33)	0.067*	NS
Motor n (%)	3 (10.0)	3 (10.0)	1.000*	NS
Visual n (%)	3 (10.0)	1 (3.33)	0.301*	NS
Focal spinal n (%)	1 (3.33)	2 (6.66)	0.554*	NS
Cerebellar n (%)				
Brain stem n (%)	–			
Pyramidal n (%)		2 (6.66)	0.150*	NS
Combined n (%)	19 (63.33)	12 (40.0)	0.071*	NS
EDSS:				
Median (IQR $25^{th}-75^{th}$ percentile)	3 (2-3.5)	2 (1-2)	0.000≠	HS
Range	1.5-7.5	1-2.5		
EDSS degree:				
Mild (EDSS <3) n (%)	11 (36.66)	30 (100)	<0.001*	HS
Moderate (EDSS 3-6.5) n (%)	17 (56.66)			
Sever (EDSS >6.5) n (%)	2 (6.66)	–		

Table (1): Count.

Parameter Group	RRMS in relapse n=30	RRMS in remission n=30	*p*-value	Sig.
CSF - OCB:				
More than two bands no (%)	21 (70.0)	26 (86.7)	0.129*	NS
Not done no (%)	3 (10.0)	3 (10.0)		
NA no (%)	6 (20.0)	1 (3.3)		
MRI findings (evidence of activity):				
No evidence of activity no (%)	10 (33.3)	21 (70.0)	0.006*	HS
Evidence of activity no (%)	5 (16.7)	0 (0.0)		
NA no (%)	15 (50.0)	9 (30.0)		
Duration of the disease (years):				
Median (IQR $25^{th}-75^{th}$ percentile)	3 (2-5)	3.5 (2-5)	0.726≠	NS
Range	0.25-14	0.33-13		
Number of relapses (during last year):				
Median (IQR $25^{th}-75^{th}$ percentile)	1 (1-2)	1 (0-1)	0.002≠	HS
Range	0-2	0-3		
Medications:				
Patients not receiving DMDs n (%)	9 (30.0)	8 (26.6)	0.559*	NS
Patients receiving:				
Interferon β 1a 44 µg subcutaneous (SC) 3 times/week n (%)	9 (30.0)	5 (16.7)	0.222*	NS
Fingolimod 0.5mg orally once-daily n (%)	9 (30.0)	4 (13.3)	0.117*	NS
Interferon β 1b 250 SC every other day n (%)	–	2 (6.7)	0.150*	NS
Interferon β 1a 20mg intramuscular n (%)	2 (6.7)	4 (13.3)	0.389*	NS
Ocrelizumab 600mg intravenous every 6 months n (%)		2 (6.7)	0.150*	NS
Dimethyl fumarate 240mg/24 hours n (%)	1 (3.3)	3 (10.0)	0.301 *	NS
Teriflunomide 7mg orally n (%)		2 (6.7)	0.150*	NS

p-value >0.05: Non-significant (NS). *p*-value <0.05: Significant (S). *p*-value <0.01: Highly significant (HS).
*: Chi-square test. ≠: Mann-Whitney test. **:Independent *t*-test.

Post hoc analysis was done to compare the two patients' groups with the control group as regard serum level of sCD163. It showed no statistically significant difference between the two patients' groups ($p> 0.05$). While, showed highly significant difference between each of the patients groups with the control group ($p<0.01$) (Table 2).

Correlation studies were done between serum level of sCD 163 and different parameters studied among the whole RRMS cases collectively and among each group specifically. There was an overall no significant correlation between serum level of sCD163 and the following parameters: Age, EDSS, duration of the disease and number of relapses during last year ($p>0.05$). The receiver operating characteristic (ROC) curve was done for the serum level of sCD 163 (ng/ml) between patients and controls to assess its diagnostic performance at cut off point >100ng/ml. It revealed a sensitivity of 100%, specificity of 100%, positive predictive value (PPV) of 100%, negative predictive value (NPV) of 100% and area under the curve (AUC) of 1 (Fig. 1).

Table (2): Comparative analysis between the studied groups as regard the serum level of sCD163.

Serum level of CD163 (ng/ml)	Group A (Cases in relapse) n=30	Group B (Cases in remission) n=30	Controls n=28
Median (IQR $25^{th}-75^{th}$ percentile)	1350 (850-2400)	1000 (830-2400)	81.5 (51-99)
Range	150-4000	500-5000	8-100
Post hoc analysis			
Group A Vs Group B	Group A Vs Controls		Group B Vs Control
p-value: 0.662 Sig.: NS	*p*-value: 0.000 Sig.: HS		*p*-value: 0.000 Sig.: HS

p-value >0.05: Non-significant (NS). *p*-value <0.01: Highly significant (HS).
p-value <0.05: Significant (S). ≠: Kruakal-Wallis test.

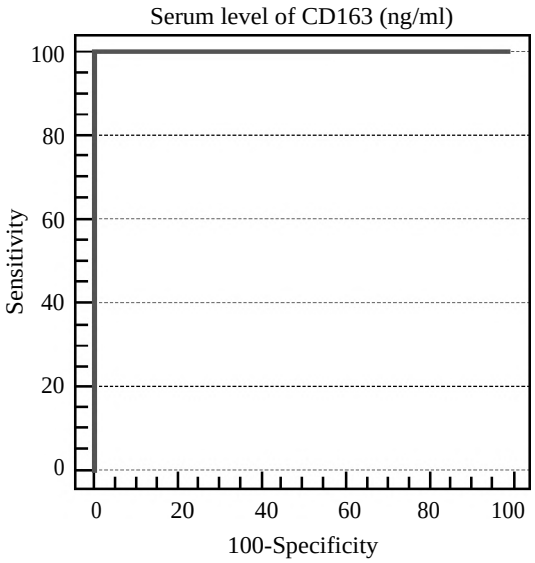

Fig. (1): ROC curve for serum level of sCD163 between patients and controls.

- AUC: 0.9-1 Excellent, 0.8 -0.9 Good, 0.7-0.8 Fair, 0.6-0.7 Poor, 0.5-0.6 Fail **[12]**.

Discussion

Our study aimed to associate between the level of serum sCD163 and the clinical activity of MS and disability measured by EDSS score. A total of 88 subjects (60 RRMS cases and 28 age and sex matched apparently healthy controls from Ain Shams University Hospitals were included in the study).

As true for most autoimmune diseases, MS is more common in females. The female to male ratio in the present study was 6.5:1 and this high ratio may be due to hormonal difference between males and females or can be attributed to more compliance of females than males in follow-up or due to higher relapse rates in females. A slightly lower ratio than those described in other studies as in studies conducted by Farrokhi et al., **[13]** and Hamdy et al., **[14]** who revealed female to male ratio about 2.24:1 and 2.11:1 respectively. However, some studies showed lower female to male ratio, e.g. in Oman (0.8:1) **[15]** and in Italy (0.44:1) **[16]**.

In our studied population, the mean age at disease onset was 24.51 ±6.78 years, slightly lower than the overall reported estimate in a meta-analysis of 52 studies in the Middle Eastern and North African countries in which the age of onset they reported was (27.61-29.48 years) **[15]**. Mirmosayyeb et al., **[17]** reported an earlier age of disease onset in which the mean age of disease onset they observed was 14.8±2.29 years. This early age of onset may be attributed to vit D deficiency occurring during critical developmental stages as in prenatal and perinatal stages of life **[18]**. A study conducted by Lotti et al., **[19]** who reported an older age of onset of MS in which the median age of disease onset was 54 years. This may be attributed to delayed diagnosis because of other co morbidities like vascular diseases of CNS which presented by similar symptoms and occurred in a higher prevalence in this age group **[20]**.

A study conducted in Egypt done by Hamdy et al., **[14]** who demonstrated a population of patients with a positive family history of MS of 2.28% vs 12.6% in a meta-analysis study done byHarirchian et al., **[21]** and 8.33% in our study. However, a larger proportion with this risk factor for MS was found in Kuwait (26.2%) which may be related to a high incidence of consanguineous marriage in this country **[22]**.

In the present study, the most frequent presenting symptoms among all patients were combined symptoms (51.66%), followed by sensory symptoms (23.33%). In each patients group specifically the most frequent presenting symptoms also were combined symptoms followed by sensory symptoms, while motor symptoms in all patients collectively were presented by only 10% and in each group specifically it also presented by 10%. These findings came in co ordinance with Sawaya and Kanso **[23]** who reported that combined symptoms(46.2%), followed by sensory symptoms (42.5%) were the most common initial presentation of MS. While motor symptoms were reported by Hasan et al., **[24]** and Inshasi and Thakre **[25]** as the most common presentationfollowed by sensory symptoms. Another study conducted by Alroughani et al., **[26]**, they reported that sensory symptoms were the most common presenting symptoms followed by motor symptoms.

In the present study, serum level of sCD163 showed a highly significant statistical difference between MS patients group and healthy controls (p<0.01). It ranged from 150 to 5000ng/ml with median of 1200ng/ml in all RRMS patients while it ranged from 1.5 to 100ng/ml with median of 76.5ng/ml in controls. This finding came in consistency with Farrokhi et al., **[13]** who found that the serum level of sCD163 was high in patients with different subtypes of MS than in healthy controls. Their mean of serum levels of sCD163 was 2160ng/ml ±1140 SD in RRMS patients, while in the control group was 1450ng/ml±730 SD. However, De Fino et al., **[16]** and Stilund et al., **[9]** reported no significant statistical differences between the studied groups of MS patients and controls as regard serum levels of sCD163 (p>0.05).

De Fino et al., [16] reported the mean of serum levels of sCD163 in RRMS patients of 470ng/ml ±464.2 SD while Stilund et al., [9] reported a medianserum level of sCD163 in RRMS group of 1490ng/ml with a range of 390 to 4530ng/ml. This discrepancy may be attributed to their use of different method (Luminex) in the former andthe use of different ELISA kits with different reference ranges in the latter or due to the difference in their both control groups (subjects with other neurological diseases).

In the present study, although the difference in the serum level of sCD 163 was significant between MS patients and healthy controls,it was non-significantly differ between the relapsing and the remitting MS patient groups suggesting the usage of sCD163 serum levelamong the markers of disease diagnosis rather than disease activity. Also, Stilund et al., [27] who assessedthe sCD163 CSF/serum ratio in diagnosis of MS, reported that sCD163 CSF/serum ratio contributed positively as a diagnostic marker to a panel of established MS biomarkers and they suggested the combination of this ratio with other biomarkers to increase the diagnostic power rather than the usage of individual biomarker levels in diagnosis.

In the present study, non-significant statistical correlation was observed in MS patients collectively and in each patients group specifically (p>0.05) between the serum level of sCD163 and some clinical and demographic featuresincluding: Age, EDSS, duration of the disease and number of relapses during last year. This insignificant correlation could be explained by the effect of treatment given to the patients. These findings came in coordinance with Gjelstrup et al., [28] who reported non-significant statistical correlation in the studied MS patients between the serum level of sCD163 and some clinical features including: EDSS, the number of attacks, the time since last attack, the IgG index and the total number of MRI white matter lesions. Also, Stilund et al., [9] reported non-significant statistical correlation in the studied MS patients between the serum level of sCD163 and some clinical and demographic features except for the age. This difference may be referred to their choice of newly diagnosed MS patients regardless their age.

Other studies conducted by Stilund et al., [29] and Farrokhi et al., [13] revealed significantly positive correlation in the studied MS patientsbetween the serum level of sCD163 and some clinical features. The former revealed significantly positive correlation in their MS patients between the serum level of sCD163 and MRI lesions (p=0.04, r=0.27) and the latter revealed significantly positive correlation in the studied MS patients between the serum level of sCD163 andEDSS (p=0.00 and r=0.63). This discrepancy may be due to the larger sample size in their studies and the inclusion of many different MS subtypes, also their inclusion criteria differ from ours in that no patients excluded according to the values of EDSS, but in our study, all patients in remission with EDSS more than 3 were excluded as we targeted our patients in the early disease reflecting the inflammatory stage of the disease not the progressive one.

The diagnostic value of sCD163 serum levels for diagnosis of MS was investigated by calculating ROC curves. The value of sCD163 serum level in discrimination between patients and healthy controls was assessed, the best cut off point was >100ng /ml with 100% calculated sensitivity and 100% specificity. The PPV and NPV were 100% and 100% respectively (AUC of 1).

Other study conducted by Stilund et al., [9] who assessed the diagnostic value of sCD163 serum levels for diagnosis of MS and reported an AUC of 0.37 in their ROC curve analysis. This difference may be referred to the difference in sample size and population as their controls were not healthy controls but presented with unspecific neurological symptoms. Furthermore, they assessed the sCD163 CSF/serum ratio and reported AUC of 0.72 which indicated that the sCD163 CSF/serum ratio as only a fair diagnostic marker of MS. In 2015, Stilund et al., [27] assessed the sCD163 CSF/serum ratio as a new marker of inflammation and axonal degeneration with other biomarkers in newly diagnosed MS cases. The combination of all tested biomarkers including the sCD163 CSF/serum ratio in their study revealed an AUC of 0.97 and that provided a high level of diagnostic reliability. In the present study, we preferred the assessment of serum samples over CSF samples to avoid the possible complications of lumber puncture as post lumber puncture headache, local hematomas and infections [30].

The findings of the current study have to be seen in light of some limitation that could be addressed in future researches. First, the study focused on limited subtype of MS and small sample size. One of the caveats for our study is the follow up of the patients (to compare the marker serum level in both remission and relapse for each patient) was difficult in the era of COVID-19 pandemic making the compliance of patients very difficult.

To conclude, serum level of sCD163 was significantly higher in MS patients compared to controls and it could be a valuable tool of diagnosis together with other diagnostic biomarkers of MS. Furthermore, the potential role of sCD163 as a marker of monocytes and macrophages, could provide additional information about the inflammatory status in MS lesions. Moreover, the non-significant difference in the serum level of sCD163 between the relapsing and the remitting MS patients, makes the utility of sCD163 serum level limited in reflecting disease activity. These results suggested that sCD 163 serum level can be used as a non-invasive immunological biomarker in the diagnostic panel of MS reflecting the inflammatory process rather than the activity status of the patients.

Finally, the presented results support that the markers of the innate immune system can be used in diagnosis of MS reflecting the inflammatory status of MS lesions. Also the obtained results could be useful in the future studies to perform a novel noninvasive panel for diagnosis of MS. Further studies are recommended with a larger number of MS cases to individuate new immunological markers reflecting disease activity.

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- *Conflict of interest:* None.

- *Availability of data and materials:* The raw data of this study are available from the corresponding author on reasonable request.

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CD163 المذاب فى مصل الدم كمؤشر للنشاط فى مرضى التصلب المتعدد

مرض التصلب المتعدد هو مرض مناعى مزمن ويعتبر السبب الأكثر شيوعاً فى حدوث إعاقة عضبية فى سن الشباب، وتشخيصه يعتمد على الفحص الإكلينيكى، والأشعة المقطعية، وفحص السائل النخاعى.

يعتبر CD163 التى تلعب دور أساسى المذاب فى الدم مؤشر لنشاط خلايا البلاعم وفى مرض التصلب المتعدد.

هدفت هذه الدراسة إلى قياس CD163 المذاب فى مصل الدم فى مرضى التصلب المتعدد كمؤشر لنشاط المرض باستخدام تقنية الإليزا.

أجريت الدراسة على ٦٠ مريضاً بالتصلب المتعدد (٣٠ مريض تصلب متعدد متكرر انتكاسى فى حالة نشاط للمرض، و ٣٠ مريض تصلب متعدد متكرر انتكاسى فى حالة خمول للمرض) بالإضافة إلى شخص سليم مطابق من ناحية الجنس والعمر كمجموعة ضابطة.

أوضحت النتائج الإحصائية لهذه الدراسة أن CD163 المذاب فى مصل الدم لم يظْهِر منسوب.

فروقات إحصائية بين مرضى التصلب المتعدد المتكرر الانتكاسى فى حالة نشاط المرض وبين المرضى فى حالة خمول المرض ولكن أوضحت النتائج فروقات إحصائية بشكل كبير بين مرضى التصلب المتعدد ككل وبين المجموعة الضابطة.

وفى الختام، تشير نتائج الدراسة الحالية إلى أن CD163 المذاب فى مصل الدم يمكن أن يستخدم فى تشخيص مرضى التصلب المتعدد كمؤشر غير اختراقى يعكس عملية الالتهاب التى تحدث فى بؤر المرض.