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STUDY OF PENTRAXIN-3 LEVELS IN EGYPTIAN CIRRHOTIC PATIENTS

By

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ABSTRACT

Background: Liver cirrhosis is the most advanced stage of chronic liver disease. Its prevalence is in increasing and it is associated with multiple etiologies. A significantly higher percentage of acute decompensated cirrhotic patients develop in-hospital mortality. Identifying patients with worse prognosis would facilitate early management of potentially severe cases. Inflammation and tissue injury increased PTX3 in the injured liver and, accordingly, circulating PTX3 was induced in patients with chronic liver diseases, and has a positive predictive value for adverse clinical outcomes.

Objective: To study Pentraxins3 levels in Egyptian cirrhotic patients.

Patients and Methods: The study was performed on 40 adult Egyptian patients collected from the Internal Medicine Department at Sayed-Galal Al-Azhar University Hospital. Additionally, 20 healthy subjects were also included as control group. The study was carried out during the period from May 2018 to October 2020.Sixty Egyptian patients were divided into three equal groups: Group I patients with acute decompensation of liver cirrhosis, Group II stable cirrhotic patients, and Group III healthy subjects as controls (age and sex matched).

Results: There was male predominance in the studied subjects (533%). The mean age of the studied subjects was 44.25 years with 76.6% above 40 years. There was a significant increase of AST, ALT, ALP, T. bilirubin, D. bilirubin and CRP among the stable cirrhotic and the acute liver decompensation cirrhotic compared with the controls.Pentraxin-3 was found significantly higher in acute liver decompensation cirrhotic group than control and stable cirrhotic group. It was 2.7 ± 0.7 in acute liver decompensation cirrhotic, 2.4±0.5 in stable cirrhotic and 1, 2 ± 0.2in control group.

Conclusion: Patients with acute liver decompensation cirrhosis showed increased level of Pentrxin-3than in control and stable cirrhotic.

Keywords: Liver cirrhosis, Pentrixin-3, C-reactive protein, Biomarkers.

INTRODUCTION

Liver cirrhosis is the most advanced stage of chronic liver disease. It is characterized histologically by the presence of regenerative nodules. Its prevalence is estimated at 0.27% in the USA, and it is associated with multiple etiologies, most commonly chronic viral hepatitis B and C, ethanol consumptionand diabetes mellitus *(Scaglione et al., 2015)*.

Identifying patients with worse prognosis would facilitate early management of potentially severe cases. Several prognostic markers have been studied to identify mortality associated

with decompensated cirrhosis, including MELD score *(Jéssica et al., 2017)*.

Pentraxin-3 (PTX-3) is an acute-phase protein is a member of the long pentraxin protein family *(Kadir et al., 2016)*. It has been reported that PTX-3 is significantly associated with obesity, metabolic syndrome and cardiovascular diseases *(Gurel et al., 2016)*.

Pentraxins are proteins formed by 5 monomers that form a ring in radial symmetry. They are a class of pattern recognition receptors. Among pentraxins, the main ones are pentraxin-3, CRP and serum amyloid P component. PTX3 is a long-chain pentraxin considered an acute phase marker produced mainly by endothelial and vascular smooth muscle cells at the site of inflammation. It is also produced by macrophages, fibroblasts, neutrophils, epithelial cells, dendritic cells and other cell types both near and far from the inflammation site *(Cieslik and Hrycek, 2012* and *Zhang et al., 2012)*. Pentraxin production is influenced by certain inflammatory stimuli such as IL-1β and TNF-α *(Luchetti et al., 2010)*. It differs considerably from CRP in terms of expression patterns by affected organs. In particular, this is a short pentraxin mainly produced in the liver in response to IL-6 *(Manfredi et al., 2013)*.

PTX3 has been recognized as an independent marker of inflammation associated with various disorders *(Manfredi et al., 2013* and *Ortega et al., 2014)* such as atherosclerosis, cancer, respiratory diseases and CNS diseases in which increased levels are related to the risk of the disease or its progression *(Rajkovic et al., 2016)*.

The present study aimed to study Pentraxins3 levels in Egyptian cirrhotic patients.

PATIENTS AND METHODS

The study was performed on 40 adult Egyptian patients collected from the Internal Medicine Department at SayedGalal Al-Azhar University Hospital. Additionally, 20 healthy subjects were also included as control group. The study was carried out during the period from May 2018 to October 2020.

Ethical approval was gained according to the recommendations of Ethics Unit, Faculty of Medicine, Al-Azhar University. The clinical steps and possible adverse events were plainly demonstrated for all candidates and gave consents to share in this work.

Sixty Egyptian patients divided into three equal groups:

Group I: Patients with acute decompensation of liver cirrhosis.

Group II: Stable cirrhotic patients.

Group III: Healthy subjects as controls (age and sex matched) were included in the current study.

Exclusion criteria:

1. Patients were excluded because of insufficient clinical and or laboratory data.
2. Patients with hepatocellular carcinoma (HCC).
3. Any other chronic illnesses, e.g. autoimmune diseases, chronic renal disease, heart failure, etc.
4. Patients who tooke statins as they have lowering effect on plasma PTX-3.

5. Patients with sepsis.

All subjects were subjected to the following:

- Detailed history taking with special emphasis on: Age, sex, etiology of cirrhosis, drug use, and presence of ascites.
- Full clinical examination including measurements of body mass index, vital signs, abdominal examination and other systems examination.
- Laboratory investigations including CBC, AST, ALT, GGT, AFP, CRP, serum albumin, total bilirubin, FPG, PPPG, PT, INR, sodium, potassium, calcium, creatinine, urea, U/S abdomen and measurements of serum PTX-3 level assessment in serum by ELISA.
- Abdominal ultrasound was done for liver cirrhosis and others with examination of liver size, echogenicity, hepatic focal lesion, splenic size, portal vein diameter, and presence of ascites.
- The severity of liver disease was estimated by Child-Pugh and MELD scores calculated based on laboratory tests performed on admission.

Statistical analysis:

Continuous-normally distributed variables were reported in the form of mean, and standard deviation (SD) and compared by one-way ANOVA test or by Kruskal-Wallis test whereby continuous non-normally distributed data were notified using median and range. Besides that, categorical variables were expressed using number, and percentage and were compared by Chi2 test Correlation analysis was conducted using Spearman's rank correlation coefficient for categorical data. The significance was established when $P< 0.05$. Statistical analysis was performed using SPSS software version 23 for Windows (SPSS Inc., Chicago, IL, USA).

RESULTS

The mean age in control was 44.72, in stable cirrhotic group was 45.280 years, and in acute liver decompensation cirrhotic group was 45.345 years. There was no statistical significant difference between the two studied groups as regard age and gender **(Table 1)**.

Table (1): Demographic data in between the studied groups

Groups / Parameters	Control group (n=20)		Stable cirrhotic group (n=20)		Acute liver decompensation Cirrhotic (n=20)		P value
Age (years):							
Mean ± SD Range	44.720 ± 7.840 (22 - 62)		45.280 ± 7.003 (28 - 68)		45.345 ±4.043 (30 - 68)		0.996
	No.	**%**	**No.**	**%**	**No.**	**%**	**P value**
Gender:							
Female	8	40.0	12	60.0	10	50.0	0.449
Male	12	60.0	8	40.0	10	50.0	

F is for one way ANOVA, X2 for chi square test

There was a significant difference in between the studied groups (control, stable cirrhotic and acute liver decompensation cirrhotic group) as regard to AST, ALT, ALP, T. bilirubin, D. bilirubin and CRP. There was a statistical difference in between the studied groups regarding to blood electrolytes (Na, K, Ca) with P<0.001 **(Table 2)**.

Table (2): Laboratory findings among the studied groups

Parameters \ Groups		Control group (N = 20)	Stable cirrhotic group (N=20)	Acute liver decompensation of cirrhotic (N=20)	^P
AST (U/L)	**M ± SD**	25.0 ± 5.7	66.2 ± 9.0	69.2 ± 5.8	**< 0.001**
	Range	11.0 -33.0	26.0 - 108.0	36.0 - 118.0	
ALT (U/L)	**M ± SD**	27.0 ± 5.7	62.5 ± 9.2	64.1 ± 92.3	**0.001***
	Range	8.0 -35.0	76.0 - 404.0	84.0 - 412.0	
ALP (U/L)	**M ± SD**	57.0 ± 3.6	173.6 ± 48.0	179.4 ± 41.8	**0.000***
	Range	23.0 -100.0	108.0 - 313.0	122.0 - 301.0	
T. bilirubin (mg/dL)	**M ± SD**	0.93 ± 0.2	1.1 ± 0.8	1.3 ± 0.4	**0.000***
	Range	0.6 -1.2	0.6 - 3.6	0.6 - 2.5	
D. bilirubin (mg/dL)	**M ± SD**	0.1 ± 0.1	0.4 ± 0.5	0.5 ± 0.3	**0.036**
	Range	0.1 - 0.3	0.1- 2.1	0.1 - 1.8	
CRP (mg/dL)	**M ± SD**	5.8 ± 0.5	44.8 ± 20.1	45.2 ± 20.4	**0.000***
	Range	0.5 -10	10.0 - 67.0	11.0 -78.0	
Na^+	**Range**	130.0 – 140.0	127.0 – 140.0	115.0 – 142.0	**<0.001**
	M ± SD	134.1^{b}± 3.41	133.4^{ab}± 4.58	137.7 ± 2.58	
K^+	**Range**	4.0 – 5.50	3.60 – 5.70	3.60 – 4.40	**0.001**
	M ± SD	4.56 ± 0.61	4.79^{abd}± 0.75	3.94 ± 0.26	
Ca^{++}	**Range**	8.40 – 9.80	7.90 – 9.40	9.30 – 10.20	**<0.001**
	M ± SD	8.99^{a}± 0.47	8.50^{abcd}± 0.44	9.67 ± 0.28	

PTX-3 level was significantly in acute liver decompensation cirrhotic than in control and stable cirrhotic group with P<0.001 **(Table 3)**.

Table (3): Comparison between the different studied groups according to PTX-3

Groups / PTX-3 (ng/ml)	Control group (n = 20	Stable cirrhotic group (N=20)	Acute liver decompensation cirrhotic group (N=20)	p
Range	**0.7 - 2.2**	**1.7 - 3.2**	**1.7 - 3.9**	**<0.001**
M ± SD	**1,2 ± 0.2**	**2.4±0.5**	**2.7 ± 0.7**	

PTX3 had excellent diagnostic performance with an AUC of 0.940 (95% CI = 0.840 to 1.040, p-value =0.001). A best cut-off criterion of PTX3 ≤ 2.4 ng/ml could discriminate between patients with cirrhotic from control with a sensitivity of 90.0% and specificity of 80% **(Table 4** and **Figure 1)**.

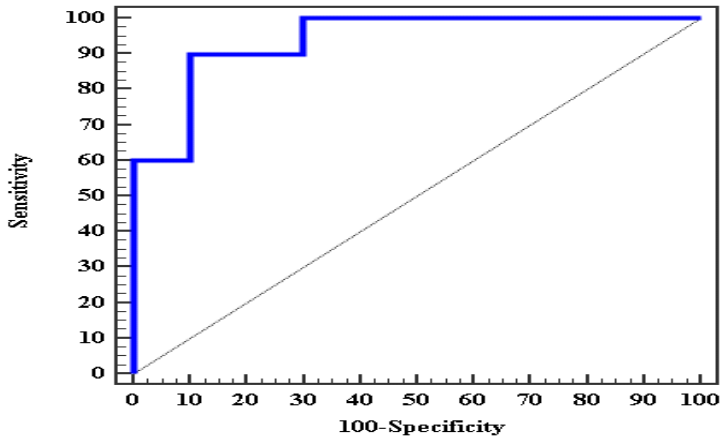

Figure (1):ROC curve for PTX3 in cirrhotic patients from control

Table (4): Diagnostic performance of serum PTX3 in cirrhotic patients from control

	AUC	P	95% CI	Cut-off	Sens.	Spic.	PPV	NPV
PTX3 (ng/ml)	**0.940**	**0.001***	**0.840-1.040**	**≤ 2.4#**	**90.0**	**80.0**	**81.8**	**88.9**

AUC: Area Under a CurveNPV: Negative predictive valueNPV: Negative predictive value
p value: Probability valueCI: Confidence IntervalsPPV: Positive predictive value
#Cut off was choose according to Youden index

There was a significant correlation in between PTX3 level and ALT, AST,serum albumin, T. bilirubin and CRP. There was a significant correlation in between PTX3 level and AFP, PT serum creatinine and urea **(Table 5)**.

Table (5): Correlation between PTX3 and different parameters in acute liver decompensationcirrhotic group

PTX3 (ng/ml) / Parameters	r	p
Age (years)	**-0.172**	**0.105**
Gender	**0.258**	**0.684**
Cr (mg/dl)	**0.812**	**0.041**
Urea (mg/dl)	**0.752**	**0.029**
ALT(U/L)	**0.602**	**<0.001***
AST (U/L)	**0.668**	**<0.001***
Serum albumin (g/dL)	**0.892**	**<0.001***
Hb (g/dL)	**-0.242**	**0.062**
WBC count	**0.582**	**0.058**
Platelet count	**0.355**	**0.052**
AFP	**0.790**	**0.002**
T. bilirubin	**0.762**	**<0.001***
CRP	**0.784**	**<0.001***
PT	**-0.912**	**0.025**
Child class	**0.265**	**0.598**

DISCUSSION

In this study, there was PTX-3 level significantly higher in acute liver decompensation cirrhotic than in control and stable cirrhotic group which in line with the study done by *Fan et al. (2017)* who stated that in comparison with unrelated healthy controls, serum PTX3 levels already significantly increased in well compensated cirrhotic patients and also significantly higher in acute decompensated cirrhotic patients than control individuals and well-compensated cirrhotic patients. *Pereira et al. (2017)* also stated that when comparing PTX3 levels between groups, it was observed that the cirrhotic outpatients had higher means compared to healthy controls. Hospitalized cirrhotic patients had higher means compared both to healthy controls and to cirrhotic outpatients.

This was also consistent with the results of studies that showed elevated PTX3 levels in diseases with an inflammatory component that affect other organs such as acute myocardial infarction *(Latini et al., 2010),* severe infectious diseases affecting patients in intensive care *(Muller et al., 2010),* chronic kidney disease *(Tong et al., 2011),* and acute respiratory distress syndrome (ARDS) *(Mauri et al., 2012).* Serum levels are positively correlated with disease severity.

This finding was corroborated by the positive correlation between serum PTX3 levels and the scores associated with severity of liver cirrhosis (Child-Pugh). *Muller et al. (2010)* stated that in patients in intensive care with systemic inflammatory response syndrome (SIRS), sepsis, severe sepsis and septic shock. There was also a positive correlation between serum PTX3 levels and the clinical severity scores APACHE II (Acute Physiology and Chronic Health Evaluation) and SAPS II (Simplified Acute Physiology Score).

PTX3 was higher in cirrhotic patients than controls, whereas prothrombin conversion as well as thrombin inactivation was impaired in these patients. *Kremers et al. (2017)* suggests that raised PTX3 in liver cirrhosis may in part compensate for deficiencies in pro- as well as anticoagulatory pathways.

In the current study, there was a positive significant correlation in between PTX3 level and ALT, AST serum albumin in both groups (cirrhotic and acute liver decompensation cirrhotic group) which disagreed with the results by *Pereira et al. (2017)* and *Feder et al. (2020)* who found there were no correlations of PTX3 levels with albumin, aspartate aminotransferase, and alanine aminotransferase.

In this study, there was a significant correlation of PTX3 levels with serum urea, creatinine, in both groups which coincided with the results in the study done by *Pereira et al. (2017)* who found that there was a positive correlation between serum levels of PTX3 and creatinine. They explained that due to its high molecular weight (40.6 KD) and multimeric structure, PTX3 levels appear to increase as the glomerular filtration rate (GFR) decreases secondary to reduced clearance *(Tong et al., 2011).*

In the current study, there was no significant correlation in between PTX3 level and total WBC, platelet count in both groups (which coincide with the results in the study done by *Pereira et al. (2017)* as there was no correlation

between PTX3 levels and total leukocyte count and platelet count.

As regard to bilirubin, there was a statistical correlation between PTX3 level and total bilirubin in both groups which in line with *Fan et al. (2017)* who commented that bilirubin was positively associated with PTX3, but that disagreed with *Pereira et al. (2017)* who found no significant correlation in between them.

As regard to CRP, there was a positive significant correlation in between PTX3 level and CRP in cirrhotic patients which coincided with the results in the study done by *Fan et al. (2017)* who stated that a significantly higher serum CRP level was only noted in the high PTX3 group than the low PTX3 group. Meanwhile, the PTX3 significantly positively correlated with serum CRP levels.

But that disagrees with the study done by *Pereira et al. (2017)* and *Feder et al. (2020)* who stated thatPTX3 did not correlate with C-reactive protein (CRP) in the cirrhotic patients

In the present study, there was a significant negative correlation in between PTX3 level and prothrombin time in both groups (cirrhotic and acute liver decompensation cirrhotic group) which in agreement with the study done by *Feder et al. (2020)* who had the same results. Negative correlations of PTX3 with prothrombin time were in accordance with a function of PTX3 in the extrinsic pathway of coagulation. Tissue factor initiates extrinsic blood coagulation, and PTX3 enhances the expression of this protein in activated monocytes and endothelial cells. Thus, shorter prothrombin time in cirrhotic patients with high PTX3 may be because of higher tissue factor expression *(Napoleone et al. 2012* and *Napoleone et al., 2014)*.

In the present study, there was no significant correlation in between PTX3 level and Child class in both groups (cirrhotic and acute liver decompensation cirrhotic group) which agreed with *Narciso-Schiavonet al. (2017)*, PTX3 levels were not different in Child–Pugh class which in line with our results. Hence, PTX3 was not associated with the severity of liver disease. High levels were more likely related to severe complications such as acute-on-chronic liver failure or infections. Also, in another studies by *Pereira et al. (2017)* and *Feder et al. (2020)* who stated that no associations of PTX3 with Child–Pugh score.

CONCLUSION

Patients with acute liver decompensation cirrhosis showed increased level of pentrxin-3than in control and stable cirrhotic.

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دراسة مستوى البنتراكسين-3 فى المرضى المصريين بالتشمع الكبدى

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خلفيــة البحــث: تشــمع الكبــد مرحلــة أكثــر تقــدمًا مــن مــرض الكبــد المزمن,وإنتشــاره آخــذ فــي الازديــاد ويــرتبط بمســببات متعــددة. وقــد ازدادت نســبة الوفيــات بالمستشــفيات فــى حــالات مرضــى التليــف الكبــدي الحــاد الــلا تعويضــي الحــاد. وتحديــد المرضــى الــذين يعــانون مــن تشــخيص أســوأ مــن شــأنه أن يســهل الإدارة المبكــرة للحــالات الشــديدة المحتملــة. ويــؤدى الالتهــاب وإصــابة الأنســجة إلــى زيــادة البنتراكســين3 فــي الكبــد المصــاب، وبالتــالي، تــم تحفيــز تــداول البنتراكســين3 فــي المرضــى الــذين يعــانون مــن أمراض الكبد المزمنة وله قيمة تنبؤية إيجابية لنتائج سريرية سلبية.

الهــدف مــن البحــث: دراســة مســتوى البنتراكســين3 فــي المرضــى المصــريين المصابيين بالتليف الكبدي.

المرضــى وطــرق البحــث: تــم إجراءالدراســةعلى 40 مريضًــا مصــريًا بالغًــا تــم جمعهــم مــن قســم الطــب البــاطني بمستشــفى ســيد جــلال -جامعــة الأزهــر، وتمتضــمين 20 مــن الأشــخاص الأصــحاء كمجموعــة تحكــم. وقــد تــم إجراءالدراســة خــلال الفتــرة مــن مــايو 2018 إلــى أكتــوبر 2020، وتــم تقســيم ســتين مريضًــا إلــى ثــلاث مجموعــات متســاوية: المجموعــة (1) مرضــى يعــانون مــن التليــف الكبــدي الحــاد الــلا تعويضــي, والمجموعــة (2) مرضــى التليــف الكبــدي المســتقر,والمجموعة (3) أشــخاص أصــحاء كمقيــاس للتحكيم (وتم التناظر بين السن والجنس).

نتــائج البحــث: بلغــت نســبة الــذكور فــى الأشــخاص الــذين تــم دراســتهم 53.3%. ومتوســط عمــر الأشــخاص الــذين تــم دراســتهم 44.25 عــام، مــنهم 76.6% فــوق 40 عــام. وجــد أن مســتوى البنتراكســين 3 أعلــى بصــورة ملحوظــة فــي مجموعــة المرضــى المصــابين بالإلتهــاب الكبــدي التــدهني أكثــر مــن مجموعــة المرضــى الغيــر مصــابين

بالإلتهاب الكبدي التدهني، وبصورة ملحوظة أيضا وجد أنه أعلى في مجموعة المرضى الغير مصابين بالإلتهاب الكبدي التدهني أكثر من مجموعة الأصحاء(مقياس التحكيم). فنسبته كانت 5.65 (4.1- 7.15) في مرضى الإلتهاب الكبدي التدهني الغير كحولي، 1.7 (0.85- 2.5) في المرضى الغير مصابين بالإلتهاب الكبدي التدهني، 0.85(0.6 – 1.1) في مجموعة وسطاء التحكيم.

الاستنتاج: المرضى المصابونبتليف الكبد الحاد اللا تعويضي يعلو لديهم مستوى البنتراكسين3 بصورة ملحوظة.

الكلمات الدالة: البنتراكسين-3، التشمع الكبدى، بروتين سي التفاعلي، المؤشرات الحيوية.