STUDY OF PLASMA PENTRAXIN 3 LEVELS AS A NOVEL MARKER VERSUS LIVER STIFFNESS FOR NONALCOHOLIC STEATOHEPATITIS IN EGYPTIAN PATIENTS

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ABSTRACT:

Background: *In Nonalcoholic fatty liver disease (NAFLD), histopathological differentiating from simple steatosis to nonalcoholic steatohepatitis (NASH) can only be confirmed by liver biopsy. These is new promising non invasive marker , Plasma pentraxin (PTX3) to discriminate NASH from non-NASH patients, and it is related to degree of liver fibrosis in NASH patients.*

Objectives: *our aim is to investigate the clinical usefulness of plasma Pentraxin3 (PTX3) levels versus fibroscan to predict NASH and the potential relationship of its levels with the degree of liver damage in NAFLD /NASH Egyptian patients*

Methods: *Plasma PTX3 levels & traniset elastogrophy (fibroscan) measurements were estimated in 60 Eqyptain patients with NAFLD (30 with NASH, 30 with non-NASH) and 20 healthy controls.*

Results: *PTX3 levels were found significantly higher in the NAFLD group than in the control group (P < 0.001), and in NASH subgroup than non-NASH subgroup (P=0.001). To discriminate NASH from non-NASH, PTX3 had 96.67% sensitivity and 93.33% specificity at the cutoff value of 3.1ng/ml. Plasma PTX3 levels showed no significant correlation with NAFLD activity score, fibrosis stage and steatosis.*

Conclusion: *This study demonstrated markedly higher PTX3 levels in NAFLD patients compared with controls, and in NASH patients compared with non-NASH ones and no correlation with fibroscan stages. Thus, in this cohort we showed that plasma PTX3 may be a promising biomarker for the presence of NASH.*

Keywords: *Nonalcoholic fatty liver disease, Plasma Pentraxin Levels.*

INTRODUCTION:

The most common causes of chronic liver damage worldwide is non alcoholic fatty liver disease[(1)]**.** Macrovesicular hepatic steatosis is histologically character of it, and the NAFLD diagnosis must be in nonalchololic patients or who consumed amounts not enough to cause liver damage[(2)]**.**

The histological differentiating extending from non progressive simple steatosis, to nonalcoholic steatohepatitis (NASH), liver cirrhosis, hepatic failure, and hepatocellular carcinoma[(3)]**.**

The gold standard mean for both the diagnosis and fibrosis grading in NASH is liver biopsy, but it is not preferable as it is invasive method[(4)]**.** There were a lot of clinical predictors of fibrosis severity in NAFLD patients as elderly patients, type 2 diabetic patients, obese persons, serum transaminase level, thrombocytopenia[(5)]**.**

Many recent researches investigated new clinical markers to differentiate simple steatosis from NASH, but no one fulfill the enough requirements [6]**.** The pentraxins is considered multifunctional proteins that include two subgroups differ on the primary structure and protein length: short and long. The acute-phase proteins such as short pentraxins, amyloid P component, and C-reactive protein (CRP), are released from liver in response to inflammatory cytokines[6]**.**

long pentraxin, pentraxin 3 (PTX3), was known as aphla tumor necrosis factor- or inducible interleukin 1b in endothelial cells and fibroblasts inducible in 1990s[7]**.** Inflammatory conditions such as rheumatologic diseases, bronchial asthma, ischemic artery diseases, vasculitic disorders and sepsis, are associated with elevated serum PTX3 level than normal controls[8]**.**

NAFLD is considered an ongoing inflammatory condition, we hypothesized that there is elevated plasma PTX3 levels in NAFLD patients and aimed to investigate the correlation between serum PTX3 levels and the degree of liver injury of NAFLD patients and to show if there is sufficient role of PTX3 in the diagnosis and staging of NAFLD/NASH patients[9]**.**

AIM OF THE WORK:

Is to investigate the clinical usefulness of plasma Pentraxin3 (PTX3) levels versus fibroscan to predict NASH and the potential relationship of its levels with the degree of liver damage in NAFLD /NASH Egyptian patients.

PATIENTS AND METHODS:

In this study 60 patients with NAFLD (30 NASH patients, 30 non NASH patients), and 20 healthy control subjects were enrolled. The study protocol was approved by our local ethics committee, and all subjects gave written informed consent to participate in the study. Patients with NAFLD were seen consecutively at the outpatient clinics of Ain Shams University Hospital between August 2018 and May 2022.

NASH patients had elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels for at least 6 months together with steatosis at ultrasonography (US). They had no history of any hepatotoxic drugs, hormone replacement therapy, or herbal products; no alcohol consumption; and no viral or autoimmune hepatitis, hemochromatosis, Wilson's disease, alpha-1 antitrypsin deficiency, biliary disease, or malignancies.

Patients with conditions that might increase plasma PTX3 levels, such as heart failure, asthma, vasculitis, autoimmune rheumatic disease or patients with conditions known to reduce plasma PTX3 levels, such as a history of statintherapy were excluded. The control group had no illness to cause any inflammation; no usage of alcohol, drug, or herbal substances; no history of previous liver diseases; and was negative for viral hepatitis serology tests and had completely normal liver US. All patients and controls were of Egyptian descent.

All subjects underwent physical examination, waist measurements, and biochemical screening. The estimate of insulin resistance was calculated using the homeostasis model of insulin resistance (HOMA-IR) index. Assessment of steatosis by liver elastography, APRI score and FIB-4 score. Plasma PTX3 levels were measured by Human Pentraxin ELISA Kit using the quantitative sandwich enzyme immunoassay technique.

The Student t-test was used to evaluate differences between the two study subgroups (NASH vs. non-NASH) in normally distributed continuous variables like age. A value of $P < 0.05$ (2-sided) was considered statistically significant. **Kruskal-Wallis test** was used to compare NASH, non-NASH, and control groups. A value of $P < 0.017$, calculated by Bonferroni

correction, was considered statistically significant in this triple comparison. Correlations among the study variables were tested by the **Pearson and Spearman correlation coefficient** according to suitability of the data. To determine independent predictors of NASH, multiple logistic regression analysis was performed by using age, gender, and the significantly elevated variables in NASH compared with non-NASH by univariate analysis (plasma PTX3, and HOMA-IR). Analysis of covariance **(ANCOVA)** was used to assess differences in plasma PTX3 levels between NASH and non-NASH groups byadjusting for factors like stage of fibrosis, grade of steatosis, age, sex, and HOMA-IR 2.

RESULTS:

Table (1) shows that there was statistically significant increase in the level of total cholesterol, LDL and triglyceride in NAFLD group than control group and also decrease in the level of HDL in NAFLD group than control group. Also, the table shows that ALT, AST, FBS, insulin, HOMA IR and west circumference was found higher in NAFLD group than control group. Also, there was statistically significant increase in the level of all laboratory parameters in NASH group than other groups.

Table (1): Comparison between control group and NAFLD subgroups regarding laboratory parameters of the studied patients

		NASH group	Non NASH group	Control group	Test value•	P-value	Sig.
		No.= 30	No.= 30	No.= 20			
TC	Mean±SD	231.63 ± 16.32	233.33 ± 15.98	174.50 ± 9.58	115.172	0.000	HS
	Range	206 – 270	208 – 271	160 – 190			
LDL	Mean±SD	122.70 ± 8.65	123.77 ± 8.77	91.20 ± 8.80	101.050	0.000	HS
	Range	107 – 140	107 – 141	80 – 105			
HDL	Mean±SD	41.27 ± 3.95	42.07 ± 3.71	54.55 ± 6.99	54.037	0.000	HS
	Range	35 – 51	36 – 52	45 – 65			
TG	Mean±SD	162.80 ± 11.52	163.47 ± 11.70	98.85 ± 15.81	189.890	0.000	HS
	Range	140 – 187	142 – 188	76 – 120			
ALT	Mean±SD	90.73 ± 8.19	30.63 ± 6.57	28.50 ± 5.74	702.305	0.000	HS
	Range	65 – 100	17 – 40	20 – 37			
AST	Mean±SD	64.93 ± 9.26	28.20 ± 6.47	26.60 ± 5.47	236.329	0.000	HS
	Range	52 – 91	15 – 38	18 – 35			
FBS	Mean±SD	110.83 ± 26.59	101.07 ± 21.60	88.70 ± 5.37	6.562	0.002	HS
	Range	90 – 180	84 – 157	80 – 97			
Insulin	Mean±SD	23.77 ± 7.03	16.37 ± 6.88	8.25 ± 2.65	38.235	0.000	HS
	Range	16 – 40	1 – 33	6 – 18			
HOMA-IR	Mean±SD	6.43 ± 4.72	3.37 ± 2.13	1.75 ± 0.20	14.386	0.000	HS
	Range	3 – 18	2 – 9	1.2 – 2.2			
West Circumference (cm)	Mean±SD	111.13 ± 24.66	106.93 ± 21.06	74.60 ± 3.55	22.609	0.000	HS
	Range	77 – 145	77 – 140	70 – 81			
Serum pentraxin 3 (ng/ml)	Mean±SD	4.97 ± 1.36	2.21 ± 0.75	1.16 ± 0.29	109.174	0.000	HS
	Range	2.3 – 7.4	1.1 – 4.3	0.7 – 1.6			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

Table (2): shows that there was statistically significant difference found between NASH group and non NASH group regarding serum pentraxin 3 levels with p-value <0.001

Table (2): Comparison between NASH and non NASH groups regarding Serum pentraxin 3 of the studied patients

		NASH group	Non NASH group	Test value•	P- value	Sig.
		No.= 30	No.= 30			
Serum pentraxin 3 (ng/ml)	Mean ± SD	4.97 ± 1.36	2.21 ± 0.75	9.734	<0.001	HS
	Range	2.3 – 7.4	1.1 – 4.3			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

Table (3) shows that there was statistically significant difference found between NAFLD subgroups and control group regarding serum pentraxin 3 levels with p-value <0.001.

Table (3): Comparison between control group and NAFLD subgroups regarding plasma pentraxin3 levels of the studied patients

		NASH group	Non NASH group	Control group	Test value•	P-value	Sig.
		No.= 30	No.= 30	No.= 20			
Serum pentraxin	Mean±SD	4.97 ± 1.36	2.21 ± 0.75	1.16 ± 0.29	109.174	0.000	HS
	Range	2.3 – 7.4	1.1 – 4.3	0.7 – 1.6			
Post hoc analysis							
		NASH group VS Non NASHgroup		NASH group VS Controlgroup		Non NASH group VS Control group	
Serum pentraxin 3 (ng/ml)		0.000		0.000		0.000	

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

The table (4) shows that there was no statistically significant difference found between pentraxin 3 and fibroscan in NAFLD group

Table (4): Relation between serum pentraxin3 and stages by fibroscan in NAFLD group

Stages byFibroscan	Serum pentraxin 3 (ng/ml)		Test value	P-value	Sig.
	Mean ± SD	Range			
F0	3.66 ± 1.93	1.5 – 7.2	0.219•	0.927	NS
F1	3.47 ± 1.26	1.6 – 5.6			
F2-3	3.68 ± 2.07	1.2 – 7.3			
F3-4	3.23 ± 1.53	1.1 – 5.2			
F4	4.15 ± 2.33	2 – 7.4			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

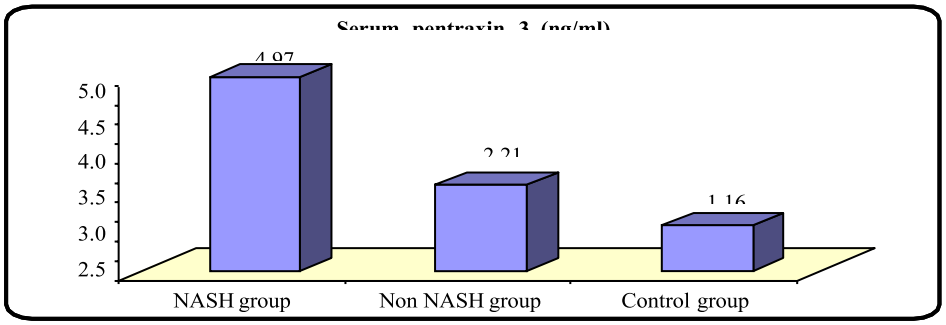
The table (5) shows that there was statistically significant positive correlation found between serum pentraxin 3 level and ALT, AST, FBS, insulin, HOMA-IR, APRI score and FIB-4 score while no statistically significant correlation found between **other studied parameters in NAFLD group.**

Table (5): Correlation for serum pentraxin 3 level with the other studied parameters in NAFLD group (no. = 60)

	Serum pentraxin 3 (ng/ml)	
	R	P- value
Age in years	0.133	0.311
TC	-0.086	0.513
LDL	0.102	0.436
HDL	-0.048	0.713
TG	-0.109	0.409
ALT	0.752**	0.000
AST	0.780**	0.000
FBS	0.348**	0.006
Insulin	0.495**	0.000
HOMA-IR	0.489**	0.000
Waist Circumference (cm)	0.131	0.319
HB (gm/dl)	0.099	0.451
WBCS	-0.007	0.957
PLT	-0.167	0.201
APRI score	0.735**	0.000
FIB-4	0.474**	0.000

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

The following Diagram (1): show that serum pentraxin level was higher in NAFLD group than control group , also it level more in NASH group than non NASH group.



(Diagram 1): Comparison between control group and NAFLD subgroups regarding serum pentraxin3level of the studied patient

The following Diagram (2) show that no relationship between the level of pentraxin and fibroscan grading.

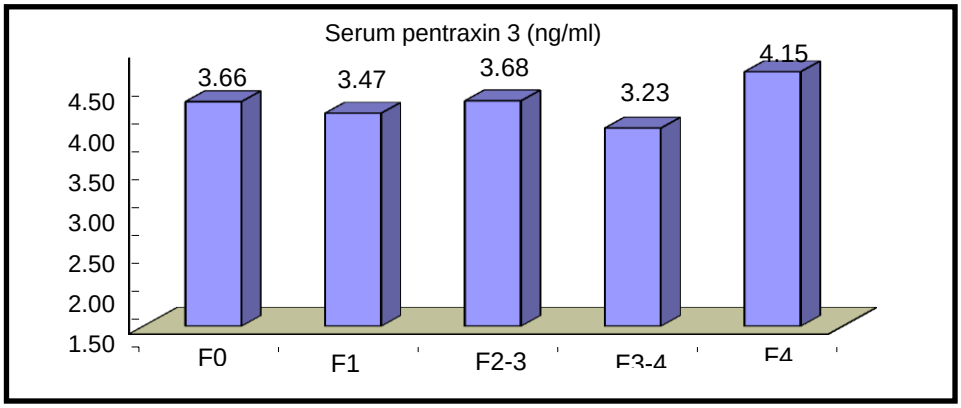
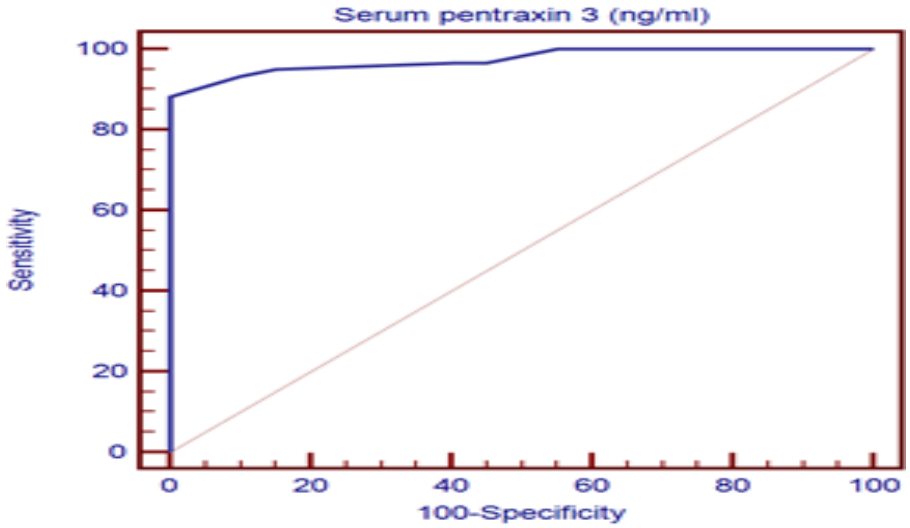


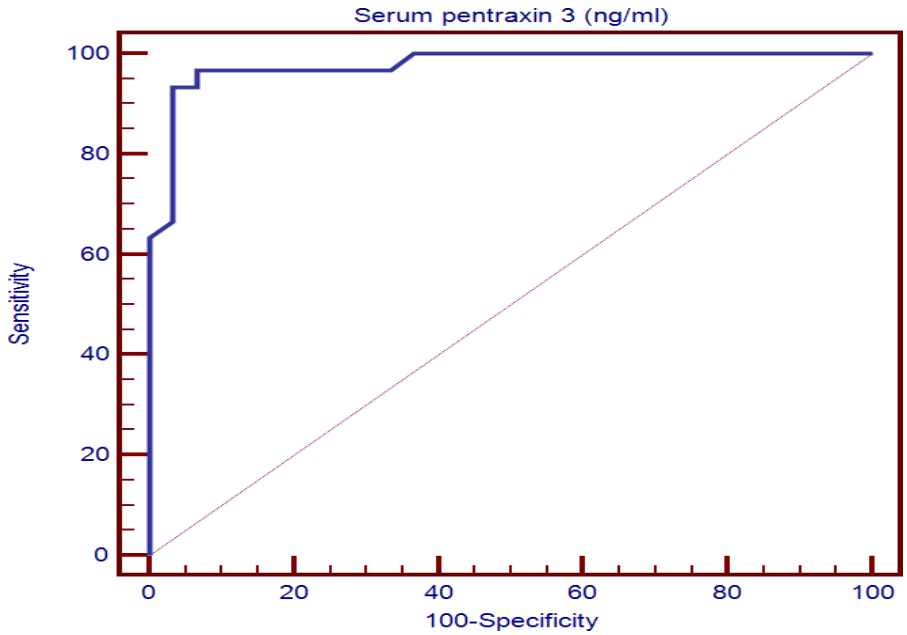
Diagram 2; Relation between serum pentraxin3 and stages by fibroscan in NAFLD group (no.= 60)

The next Diagram (3): show that the best cut off point for serum pentraxin3 to differentiate between control group and NAFLD group was found >1.6 with sensitivity of 88.33%, specificity of 100.0% and area under curve (AUC) of 0.974.



(Diagram 3)

The following diagram 4 show that the best cut off point for serum pentraxin3 (ng/ml) to differentiate between NASH group and Non-Nash group was found >3.1 with sensitivity of 96.67%, specificity of 93.33% and area under curve (AUC) of 0.977.



(Diagram 4)

DISCUSSION:

The NAFLD is the commonest hepatic disorders nowdays [10]**.** It represents more lipid deposition leading to inflammation**[11]** and hepatocytes damage, that can evently end into fibrosis[10]**.** Cirrhosis and hepato-cellular carcinoma are considered with untreated fibrosis[10]**.**

The pentraxins is considered multifunctional proteins that are included two subgroups differ on the primary structure and protein length: short and long. The acute-phase proteins such as short pentraxins, amyloid P component, and C-reactive protein (CRP), are released from liver in response to inflammatory cytokines[6]**.** long pentraxin, pentraxin3 (PTX3), was known as aphla tumor necrosis factor- or inducible interleukin 1b in endothelial cells and fibroblasts inducible in 1990s[7]**.** Inflammatory conditions such as rheumatologic diseases, bronchial asthma, ischemic artery diseases, vasculitic disorders and sepsis, are associated with elevated serum PTX3 level than normal controls[8]**.**

The aim of the work is to investigate the clinical usefulness of plasma Pentraxin3 (PTX3) levels versus fibroscan to predict NASH and the potential relationship of its levels with the degree of liver damage in NAFLD /NASH patients. This study was conducted to evaluate the role of serum pentraxin3 as a diagnostic biomarker for NAFLD. We included a total of 80 subjects who were divided into two groups; patient group 1 included (60 patients) with NAFLD divided into (2) subgroups; NASH group (30 patients) and non NASH group (30 patients), and Group (2) included (20 healthy controls).

Our findings showed highly significant elevation of fasting blood sugar, insulin &HOMA -IR in cases of NAFLD compared to controls ($p < 0.001$) same as[12]**.** On the other hand, another study negated any significant difference between cases and controls regarding the prevalence of impaired fasting glucose levels ($p= 0.131$)[13]**.**

Our findings showed a highly significant elevation of serum total cholesterol, LDL & triglycrides levels in cases against controls ($p = 0.001$), **Yilmaz et al.**[14] **and Rhobny et al.**[15] were agreed with our findings. Our findings showed a highly significant decrease of HDL in the cases group compared to controls which was agree with **Dai et al** study[16]**.**

In this study: we found that plasma PTX3 are higher in patients with NASH (4.97 ng/ml) than in those with a more benign form of NAFLD, namely non-NASH (2.21ng/ml). Also, higher plasma PTX3 levels were associated with fibrosis, although the correlation between plasma PTX3 levels and fibrosis grade was weak. In partial agreement with our study, **Yoneda et al.[17]** conducted a study with liver biopsied 70 NAFLD patients in 2008 and showed that plasma PTX3 was useful for differentiating NASH patients from non-NASH ones and PTX3 levels increased with the increasing fibrosis stages.

Different from the study of **Yoneda et al.**[17]**, Boga et al.**[9]**,** investigated plasma PTX3, and also they evaluated the histology of liver biopsies. Both showed a significant correlation between plasma PTX3 levels and the histologic grade of steatosis. But the same of our study, **Rhobny B.O et al.**[15] reported that serum pentraxin3 level positively related to degree of fibrosis detected by fibroscan.

Also in agreement with our study, **SAMIR et al**.[18] concluded that serum pentraxins 3 was higher level in NAFLD group than control group with more higher in NASH group than non NASH group. and it is level is statistically insignificant in relation to different grading of disease severity in NAFLD patients. In our study,

there was insignificant correlation between serum pentraxins level and fibroscan staging that was agree with **Donnelly and Simpson**[19] reported that no significant association between pentraxins level and fibrosis staging .On another hand **Arora and Sharma(20)** showed more increasing level of pentraxin with more degree of fibrosis

In our study there was statistically significant relationship between the level of pentraxins3 level and the liver enzymes, APRI and FIB4. These results went with agreement with **SAMIR et al**[18] that showed positive correlation between them. Also **Rhobny B.O et al.**[15] concluded positive relationship between serum pentraxin3 level and liver enzymes, APRI.

Our results showed that there was no statistically significance relationship between the serum pentraxins3 level and serum cholesterol , triglycerides, LDL, HDL .These finding went against **SAMIR et al.**[18] study that concluded positive relationship with serum pentraxin3 and lipid profile .

Several limitations are inherent in our study. First, the relatively small sample size limits the generalizability of our conclusions. Second, our patient group consists of subjects with Egyptian nationality, so that results may not be extrapolated to populations with different ethnic backgrounds. Third, we were unable to assess PTX3 with immunohistochemical analysis in liver biopsy materials. Such data would generally provide more information about the source of elevated circulating PTX3

Conclusion: the present study demonstrated markedly higher plasma PTX3 levels in NAFLD patients than controls and in NASH patients than the non-NASH ones and showed that plasma PTX3 may be a promising biomarker for the presence of NASH. Also, we found no correlation between serum pentraxin 3 and stages by fibroscan &its stages in NAFLD group.

Conflict of interest:

The authors report no conflicts of interest.

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دراسة مستوى البنتراكسين3 فى البلازما كمقياس جديد مقابل تصلب الكبد فى ألتهابات الكبد الدهنى الغير كحولى فى المرضى المصريين

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قسم الكبد, الجهاز الهضمى والباطنة العامة . كلية الطب جامعة عين شمس.

الخلفية العلمية : فى مرض الكبد الدهنى غير الكحولى (NAFLD) لا يتم التفرقة بين نسيج التكيس الدهنى المرضى البسيط وبين ألتهاب الكبد الدهنى غير الكحولى (NASH) إلا عن طريق أخذ عينة من الكبد. **مستوى البنتراكسين3 فى البلازما :** يعتبر مقياس فعال ليس له آثار جانبية سلبية للتميز بين مرضى (NASH) وغير (NASH) ، مع وجود علاقة جيدة مع تليف الكبد.

الهدف من الدراسة :التحقق من فائدة دلالة قياس مستوى البنتراكسين3 فى البلازما مقابل الفيبروسكان وذلك لمحاولة التنبؤ بـ(NASH)، مع تحديد العلاقة المحتملة لمستوياتة مع درجة تليف الكبد مع مرضى (NASH).

المرضى / الطرق : تم قياس مستوى البنتراكسين3 فى البلازما وعمل الفيبروسكان ل (80) مريض مصرى : (30)مريض (NASH) – (30) مريض(Non NASH) – (20) شخص صحيح].

نتائج الدراسة : مستوى البنتراكسين3 فى البلازما تكون أعلى بشكل ملحوظ فى مرضى (NAFLD) مقابل مستواه فى الأشخاص الأصحاء وأعلى فى مرضى (NASH).عن المرضي الغير**NASH)]**حساسية الأختبار(96،67)% ، تخصص (93،33)%]. **وايضا اضهرت الدراسة ان** لا توجد علاقة بين مستوى البنتراكسين3 فى البلازما ودرجة تليف الكبد.

الأستنتاج :أظهرت الدراسة أرتفاع ملحوظ فى مستوى البنتراكسين3 فى البلازما فى مرضى (NAFLD) فى مرضى (NAFLD) مقابل مستواه فى الأشخاص الأصحاء وأعلى فى مرضى (NASH).عن المرضي الغير NASH) وايضا اضهرت الدراسة ان لا توجد علاقة بين مستوى البنتراكسين3 فى البلازما ودرجة تليف الكبد. قد يكون مستوى البنتراكسين3 فى البلازما له علاقة بيلوجية واعدة لإحتمالية تشخيص مرضى (NASH).