Research Square

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Serum Phoenixin-14 Levels in Inflammatory Bowel Disease

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Research Article

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Abstract

Background:

Phoenixin-14 (PNX-14), a hypothalamic peptid, has anti-inflammatory, antioxidant and anxiolytic effect in rodents. Considering brain-gut interaction in the pathogenesis of inflammatory bowel disease (IBD) we aimed to investigate serum PNX-14 levels in patients with the disorder.

Methods:

Consented patients with ulcerative colitis (UC, n = 26), with Crohn's Disease (CD n = 19), sex and age matched healthy volunteers (HC n = 38) were enrolled in a pilot cross-sectional study. All subjects filled out the QoL Short Form and the Hospital Anxiety and Depression Scale (HAD) questionnaires. Serum PNX-14 levels were assayed with ELISA. Data was expressed as median (quartiles) and compared by nonparametric ANOVA or t test. $p < 0.05$ was accepted as significant.

Results:

PNX-14 levels were similar in all study groups. However, patients on azathioprine and/or any biological agent (n = 24) had lower PNX-14 levels as compared with those who were only on mesalamine (n = 18). PNX-14 levels negatively correlated with HAD-A scores. HAD-D score was high and QoL sub-scores were low in IBD patients as compared with HCs. PNX-14 was significantly lower in UC patients with depression as comparedwith the rest of UC group.

Conclusion:

Serum PNX-14 levels do not discriminate IBD patients from HC. However, statistically significant low serum PNX levels in patients on azathioprine and /or any biological agent may reflect the heterogeneity of pathogenesis of IBD and particularly of patients with UC. Moreover, serum PNX-14 levels in IBD patients negatively correlated with HAD-A scores reflecting brain-gut interaction. Studies that will be conducted in treatment naïve patients may clarify role of PNX in the disease.

Introduction

Ulcerative colitis (UC) and Crohn's Disease (CD) are chronic idiopathic inflammatory diseases (IBD) of the gut. IBD prevalence tends to increase especially in the industrialized countries as well as in south Asian countries [1]. Lack of curative treatment implies that IBD is going to be one of the most common and major health resource consuming disease of the GI tract in the next decades.

Pathogenesis of IBD, which is accepted as both an autoimmune and autoinflammatory disease [2] is related to genetic susceptibility [3], dysbiosis, altered immune response to pathogens and exposure to harmful environmental factors [2]. Products and/or components of dysregulated enterocytes and/or neutrophils as well as components of intraluminal pathogens serve as intracellular and extracellular signal molecules [2]. Initiation and perpetuation of inflammation is modulated by neuro-immune interaction [4] as was evidenced by altered brain connectivity [5].

Phoenixin (PNX) is a hypothalamic peptide existing in 14- and 20 -amino acid form generated by proteolysis of SMIM20, which is a mitochondrial membrane protein [6]. It acts by binding to GPR 173, a G protein coupled transmembrane receptor [7]. The first described function of PNX is to increase gonadotrophin releasing hormone (GnRH) receptor expression in the anterior pituitary [8]. Later, its role in metabolism, reproduction, food intake, water intake and in stress responses were described [8–10].

PNX-14 immunoreactivity has been shown in various central nervous system (CNS) structures such as hypothalamus, stria terminalis, area postrema, spinocerebellar tractus, dorsal vagal nucleus and nucleus tractus solitarius (NTS) and peripheral sites including tubular gastrointestinal tractus of rats [6, 11, 12]. NTS is the major integration centre of interoceptive information that conveys visceral information to the brain, but also is the site that gathers descending modulatory inputs from cortical sites controlling immune response and emotions [13], respectively insular cortex [14] and medial prefrontal cortex [13]. Insular and medial prefrontal cortex are the sites where altered brain connectivity [5] and inhibitory/excitatory neurotransmitter balance [15] had been shown in IBD patients.

There is no human data that reveals the exact localization of either PNX or GPR 173. According to human protein atlas, PNX precursor SMIM mRNA and protein were found mostly in ependymal cells and plasma cells, GPR173 mRNA was found in brain, hypothalamus, pituitary, astrocytes, oligo-dendritic cells, gonadotroph cells, pro-adipocytic cells, glial cells of colon and fibroblasts of stomach. [16]

PNX-14 controls visceral pain emerging from abdominal viscera [12], and skin [17], it relieves anxiety [18], improves memory [19] in animals. Somatic [20], inflammatory [21] and psychological stress [22, 23] activates PNX immune-stained neurons in various brain regions in rats. PNX controls excitability of NTS neurons in response to environmental and endogen stressors including corticosterone [24] are probably in concert with the other neuropeptides and cannabinoids [25]. This crucial central location makes PNX a good candidate for being one of the players in diseases related to brain-gut interaction

Another culprit that makes PNX-14 a possible player in the pathogenesis of IBD is its proven anti-inflammatory and antioxidant effects on colonocytes [26], astrocytes [27]and spinal glial cells [28] in experimental models.

Thus, we aimed to investigate serum PNX-14 levels in a group of IBD patient in a pilot cross-sectional study.

Methods

Marmara University Ethical Committee approved the study on July 19th 2024, registration number was #09.2024.932.

Participants

This pilot cross-sectional study was conducted between August 1st - October 30th 2024 in the IBD outpatient' and family medicine outpatients' facilities of Marmara University Hospital

Patients who had diagnosed with IBD according to European Crohn's and Colitis Organisation (ECCO) guidelines [29] and had been followed up in the IBD specific gastroenterology outpatients' clinics of Marmara University Hospital, were included in the study if they gave written consent after they were informed about the aim and the protocol of the study.

Exclusion criteria were presence of any organ failure, bleeding diathesis, diabetes, hypo or hyperthyroidism, polycystic ovary syndrome, anorexia nervosa, major depression, and psychosis. Patients who had the last biological therapy within the previous 30 days and patients on corticosteroid therapy equal or more than 20 mg/day of methyl prednisolone were also excluded.

Age, sex and body mass index (BMI) matched healthy people (HC) who had been seen in family medicine outpatients' clinic to verify health status at the beginning of a job entry were informed about the study and were asked to participate. Consented people were enrolled to the control group.

Overall, 26 patients with UC (F/M: 8/18), 19 patients with CD (F/M: 7/12) and 38 healthy controls (HC) (F/M: 14/24) were included in the study

Study design

All subjects 'medical history was taken and physical examination was performed. Involved bowel segments were recorded according to Montreal classification [29]. CD activity was assessed by Harvey-Bradshaw index (HBI). General well-being, abdominal pain, stool frequency, abdominal mass was scored between 0–4 and one point was added for every complication of CD if there were any. Cases were defined as mild (5–7 points), moderate (8–16 points) and severe (more than 16 points) [30]. Severity of UC was evaluated by partial Mayo Score (pMS), which includes non-invasive parts of Mayo score; stool frequency, blood in faeces, and physicians' general assessment. UC patients who had 3 or more points in pMS were accepted as having active disease [31].

All participations had filled out Hospital anxiety and depression scale (HADS), a self-reported test with multiple-choice questions that measure both the risk and the severity of anxiety and depression in patients with physical diseases. HADS has 14 questions and a Likert scale. The cut-off points were identified as above 10 for anxiety and above 7 for depression in the Turkish population [32].

All filled in a short form Quality of Life (sQoL) questionnaire (SF-36), which includes multiple-choice questions and a Likert scale. It was developed to evaluate QoL of patients with physical diseases. SF-36 evaluates eight categories of health with 36 items such as physical functioning, physical and emotional role limitations, social functioning, mental health, vitality, pain, and general health perception [32].

After quitting alcohol, food and cigarette intake for at least 24, 8, and 2 hr. and a resting period of 5 minutes, peripheral blood was collected, centrifuged at -4°C immediately and stored at -80°C until assayed. PNX-14 was assayed by using Human Phonexin-14 ELISA kit 96, (BT-Lab catalog no: E7481H) Blood chemistry and hemogram were recorded using recent patient files.

Statistics:

Data was expressed as median and quartiles. Quantitative data were compared by parametric/nonparametric ANOVA or t test. Categorical data were compared by chi square test. Correlations between continuous parameters were investigated by Spearman's test. P values < 0.05 were accepted as significant.

Results

In the UC groups two patients had ulcerative proctitis, 9 patients had left-sided colitis and 13 patients had extensive colitis or pancolitis. Two of the UC patients did not have full colonoscopy at the time of enrolment. All had mild or moderate disease activity, median pMS was 2 (IQR1-3). Fifteen patients were on local or systemic mesalamine (5ASA) treatment, 5 patients were on azathioprine (AZA) alone, 1 patient was on infliximab IFX alone, 3 patients had AZA in combination with either IFX (n = 2) or vedolizumab (n = 1), 1 patient was on corticosteroid (CS) therapy. Out of 8 women only one had menopause.

CD was ileal in 8 patients, colonic in 3 patients and ileocolonic in 7 patients. One patient had not yet completed intestinal work-up at the time of enrolment. Eight patients had fistular disease. All had mild or moderate disease activity, median HBI was 3 (IQR 2–5). Nine of the CD patients were on biological therapy, either alone (n = 7, all IFX) or in combination with AZA (1 with IFX, 1 with ustekinumab), 5 were on AZA only, and 3 on 5ASA only. Out of 7 women 5 had menopause. In the HC group only one subject was in menopause.

HC, UC and CD groups were similar in terms of age, gender, BMI, blood glucose, renal function and hematologic indices (Hgb concentration of CD patients was lower as compared to those of other groups but the magnitude of the difference was not significant (Table.1). There was no significant difference in serum PNX-1 levels between groups (Fig. 1).

Neither localization of the disease nor the presence of fistula altered serum PNX levels significantly. Severity of the disease had also no impact on serum PNX-14 level.

Because almost all of the patients had mild or moderate disease, we presumed that severity of the disease might be reflected by the treatment modality. We grouped patients into two treatment modalities, patients on only 5ASA and patients on either AZA and/or any of the biologicals and corticosteroids (AZA&B).

IBD patients in 5 ASA group had higher PNX-14 levels as compared with patients in AZA&B group (Table 2, Fig. 2). Gender distribution and ages were similar in those treatment groups. (Table.2, chi square test). Considering the possible effect of menopause on serum PNX-14 levels, we analysed only males, the difference between serum PNX-14 levels in two treatment groups was persistent. Serum PNX-14 levels of males in 5ASAgroup were also higher than those of males in AZA&B group (1377 (446–2201) vs 485,5 (404–656) p = 0.017).

Mood and Quality of Life

Depression scores of the IBD patients were higher as compared to HC, but there was no difference between groups in terms of anxiety (Table 3). All sub-scores of SF-36 were lower in IBD patients as compared to HC except for social functioning and pain perception (Table.4).

Number of the depressed patients was significantly higher in CD (8/19) and UC (11/26) groups as compared to the HC (6/38) group ($p < 0.05$, Fisher's test). Anxiety was also more common in disease groups (UC: 6/26, CD: 6/19, HC: 3/38), but magnitude of difference was significant only in CD patients ($p < 0.05$, Fisher's test, Supplementary Table 2).

Gender had no effect on HAD-A, HAD-D and sQoL scores in any group (t test, data not shown). Number of anxious and depressed men and women was similar in all groups (Supplementary Table 3).

Serum PNX-14 levels were lower in depressed UC patients as compared to UC patients who had HAD -D scores lower than 8 (Table 4. Figure 3). Anxiety did not alter serum PNX-14 levels in UC patients. Treatment modalities were similar in UC patients with depression and without depression (Supplementary Table 4.)

Serum PNX-14 level was negatively correlated with HAD-A score significantly in all IBD patients ($r=-0.33$, $p = 0.025$). Correlation between HAD-D score and serum PNX-14 level was weaker and did not reach statistical significance ($r= -0.268$, $p = 0.07$).

When groups were separated, PNX-14 levels were correlated negatively with HAD-A score ($r = 0.5$, $p = 0.09$, Fig. 4A) in UC group. Pain ($r = 0.53$, $p = 0.05$, Fig. 4B), mental health ($r = 0.48$, $p = 0.013$ Fig. 4C), social functioning ($r = 0.41$, $p = 0.036$,), general well-being ($r = 0.42$, $p = 0.034$) and emotional role limitation ($r = 0,41$, $p = 0.037$) scores positively correlated with serum PNX-14 levels only in UC group. There was no correlation between PNX-14 serum levels and any of the parameters related to mood or sQoL in CD and in HC groups (Table 5).

Discussion

As to the best of our knowledge serum PNX-14 levels have not been studied in IBD patients. The serum PNX-14 levels of IBD patients were similar as compared with that of HCs. We assumed that similarity of serum PNX-14 in IBD and HC groups may be due to enrolment of patients who had mild or moderate disease activity. Thus, we categorised patients according the actual treatment modality which may reflect severity of the disease [29]. We found that IBD patients on azathioprine and/or any of the biological agents had statistically significant lower serum PNX-14 levels. Because almost all of our CD patients were treated with biological agents, and also immune pathologies and genetic background of the UC and CD are different [2, 3], we repeated the analyses only in UC patients. The statistical difference in PNX-14 levels between the two treatment modalities of UC persisted. Observed difference between serum PNX-14 levels of patients who were treated with immunomodulatory/biological agent and those of healthy controls may reflect the presence of a novel risk factor for refractory ulcerative colitis.

Proven antioxidant effect of 5ASA [33] or anti TNF therapy might have altered any of the steps of the PNX production. We do not know the exact mechanisms of PNX-14 production. An in vitro experiment conducted in mouse hypothalamic cell lines showed that fatty acids promoted PNX-14 mRNA expression but nitric oxide, cAMP and neuroinflammation did not have any effect [34]. An in vivo experiment depicted increase in serum PNX-14 immediately after commencing of the meal in rats [35]. Anti-inflammatory and antioxidant effects of PNX-14 [8, 9, 26–28] involve modification of transcription factors that are sensitive to redox balance [36] but there is no data about the regulation of secretion of PNX-14. Another possibility is that patients who had remission in response to 5ASA treatment may represent a subgroup of patients that have similar genotypical/immunological /environmental traits. Alternatively, PNX-14 may be innocent bystander of this particular phenotype but our sample size do not allow us to define such a group.

We also speculate that the severity of inflammation overwhelmed anti-inflammatory and antioxidant capacity of the host which includes PNX secretion within yet unidentified organ/s in humans. We propose presence of neuroimmune exhaustion similar to the immunological one which was described in severe CD cases [37]. Exhaustion of central stress response that was regulated by PNX was described in rodents [23, 24].PNX-14 attenuates effects of stress by counteracting the effect of nesfatin which is another peptide co-expressed with PNX in hypothalamic nuclei that activates hypothalamo-pituitary-adrenal (HPA) axis directly [38].

PNX-14 levels are lower as compared with healthy controls in hypertensive [39] and diabetic [40] patients. PNX-14 levels negatively correlate with anxiety score in obese male adolescents but do not correlate with perceived stress [41]. Young adults who had bipolar disease [42], adolescent females who had anorexia nervosa [43] adult females who had endometriosis [44] have also low serum PNX-14 levels.

Depression, as was shown before [5, 15], was more prevalent in our patients who had IBD. Serum PNX-14 level was lower in patients with depression as compared to those without depression. The magnitude of the difference reached statistical significance only in UC group. Our study design does not allow us to

infer any causality relationship. Low serum PNX-14 levels may be the result or the cause of depression, or may reflect the presence of another confounding factor.

Clinical depression and anxiety are more prevalent among IBD patients as compared with healthy people [45], especially during flares [46]. Anxiety increases risk of escalation of therapy, hospitalization and emergency department admissions, whereas depression increases risk of flare, need for surgery in addition to previously mentioned risks for anxiety [46, 47]. We previously had shown the positive effect of psychiatric treatment on the course of IBD [32]. It significantly increased adherence to therapy, prevented flares, need for surgery and biological therapy.

In the current study, in all IBD patients, serum PNX-14 levels correlated negatively with HAD-A score. In IBD patients. all sub-headings of QoL were low as compared with HCs. Significant correlation between serum PNX-14 levels and QoL parameters was observed only in the UC group. The most significant positively correlation was observed between pain sub-score of the QoL and serum PNX-14 levels in UC patients. This finding corroborates the observed analgesic properties of PNX in women who had endometriosis [44].

Major limitations of the study are small number of patients, lack of data about the physiological variations of PNX-14 serum levels and lack of treatment naïve patients. Our sample size was too small for power analysis, our serum PNX-14 results may have type two error. We only tried to match patients in terms of age, gender, and BMI according to actual human data at the time of subject enrolment.

In conclusion, IBD patients have comparable serum levels of PNX as compared with healthy controls. However, serum PNX-14 levels were lower in IBD patients who are on AZA and / or any biological agent probably reflecting the diversity of the role of PNX in the pathogenesis of inflammation in IBD or particularly in patients with UC. Additionally, our finding of negative correlation between serum PNX-14 levels and HAD-A scores may point out to the role of PNX in the brain-gut interplay of the disorder

Declarations

Ethics approval and consent to participate

Ethical approval was granted by the Marmara University Faculty of Medicine Clinical Research Ethics Committee (Approval Date: July 19th 2024, Approval Number: 09.2024.932).

Written informed consent was obtained from all participants.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Conceptualisation: LSS, NI, YOA; Clinical evaluation and enrollment of patients: TYY, THK, OA, YA, BUK, LSS; Collecting data: ENA, FB, IG, NK; Biochemical analysis: ENA, FB, IG, NK, EU, GH; Statistical analysis: IA, PA, SH, NI, LSS; Interpretation of results :GH, PA, NI, LSS

Clinical trial number

Not applicable

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Artificial Intelligence Statement

Artificial intelligence (AI) and AI-assisted technologies were not used in this article.

Data Availability

The study data is not public due to patient privacy. The data may be provided upon reasonable request and ethical committee approval.

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Tables

Table 1. Demographic and Biochemical Characteristics of Participants

Median (p25- p75)	UC n=26	CD n=19	IBD n=45	HC n= 38
Age, years	41 (46 – 26)	33 (29 -54)	38 (26-50)	37 (31 – 47)
BMI (weight/height$^{2)}$	25,0 (22,5 - 26,5)	26,3 (21,4 - 29,1)	25,24 (22,49-27,18)	26,1 (23,4 – 27,8)
Glucose (mg/dL)	87,1 (81,9 – 92,7)	89, 3 (84,6 - 96,2)	87,9 (83,9 -95,4)	86,2 (81,1 – 93,2)
Hgb (g/dL)	14,6 (12,5 – 15,4)	13,2 (12,1 – 14,7)	13,8 (12,5 – 15,1)	14,8 (12,8 – 15,5)
WBC /mm^3	7,34 (5,6 – 8,72)	7,08 (5,88 – 8,95)	7,08 (5,8 – 8,72)	6,85 (6,04 – 7,67)
PLT / mm^3	235 (199 – 329)	256 (236 – 305)	242 (204 – 317)	236 (191 – 287)
CRP IU/L	1,3 (0,55 – 2,70)	3,15 (0,8 – 5,8)	1,55 (0,7 – 4,6)	1,65 (0,6 – 4,6)
BUN mg/dL	13 (10 – 15)	14 (11 – 16)	14 (10 – 15)	12 (9-14)
Creatinin mg/dL	0,8 (0,7 – 0,9)	0,8 (0,7 – 0,9)	0,8 (0,7 – 0,9)	0,8 (0,7 – 0,9)
Phoenixin-14 (ng/L)	466 (416 – 860)	495 (335 – 1125)	476 (380 - 1117	541 (430 – 1050)

Table.2. Serum PNX-14 levels by treatment modality

	F/M	Age years	PNX-14 ng/L
IBD			
5 ASA n=18	7/11	32 (25 – 47)	702.5 (428.3 – 1808)*
AZA and /or Biological n=24	8/16	40,5 (29,2 – 51,2)	422.5 (335.3 – 821.3)
UC			
5 ASA n=15	5/10	38 (25-50)	691 (432 – 1933)*
AZA and /or Biological n=10	3/7	40 (24,7 – 45,2)	410.5 (314.3 – 558)
CD			
5 ASA n=3	2/1	30 (25-34)	1690 (378 – 1766)
AZA and /or Biological n=14	5/9	40,5 (29,7-55)	458,8 (335,8 – 1119)
HC n=38	14/24	37 (31 – 47)	541 (430 – 1050)

*p<0.05, MWU test

Table.3. Depression, Anxiety and Quality of Life Scores

Median (p25- p75)	UC n=26	CD n=19	HC n= 38	p[a]
HAD- A	5 (3-11)	8 (5 - 12)	5 (4 -7)	0.15
HAD- D	7 (2 -10)	6 (6 - 9)	4 (2– 7)	0.043
Physical Funct.	92.5 (60 – 95)	95 (75 - 100)	95 (85 – 100)	0.062
Physical Role Limit.	87,5 (25 – 100)	50 (0.0 – 100)	100 (75 – 100)	0.016
Emotional Role Lim.	66,7 (33,3 – 100)	33,3 (0,0 – 66,7)	100 (66,7 – 100)	0.005
Vitality	55 (35-65)	50 (40 – 70)	65 (50 – 80)	0.036
Mental Health	60 (48 – 68)	60 (52 – 68)	72 (64 – 80)	0.004
Social Funct.	81,2 (62,5 – 100)	75 (50 – 87,5)	87,5 (75 - 100)	0.148
Pain	83,75 (67,5 - 100)	87,5 (67,5 - 100)	90 (67,5 -100)	0,668
General Well Being	50 (35 - 70)	50 (45 – 65)	70 (60 – 90)	<0.001

[a] Kruskal Wallis test

Table 4. Serum PNX-14 levels by Mood Status

	HAD-A> 10		HAD-A ≤10		HAD-D>7		HAD-D≤ 7	
	n	PNX-14 ng/L	n	PNX-14 ng/L	N	PNX14 ng/L	n	PNX-14 ng/L
UC	6	397.5 (250 – 1340)	20	485.5 (418.5 – 823.5)	11*	417 (324 – 691)+	15	509 (432 – 1933)
CD	6*	502.5 (367 – 1672)	13	495 (333 – 1121)	8	851 (335 – 1531)	11	422 (378 – 1117)
IBD	12	450 (345 – 1452)	33	495 (407 – 1105)	19	456 (335– 1092)	26	495 (397- 1485)
HC	3	478 (348 – 1106)	35	547 (430 – 1050)	6	482.5 (413 – 1059)	32	558.5 (427 – 1092)

*p<0.05 chi square test +p<0.05 mann whitney U test

Table 5. Correlation Between Serum PNX-14 and Mood Score

	UC n=26	CD n=19	IBD n=45	HC n=38
Mood Items				
HAD-A	- 0.504**	- 0.012	- 0.335*	0.042
HAD-D	- 0.36+	- 0.024	-0.268+	- 0.145
sQoL Items				
Physical Functioning	0.33	- 0.09	0.161	0.137
Physical Role Limitation	0.32	- 0.428+	0.006	0.094
Emotional Role Limitation	0.412*	- 0.098	0.219	0.035
Vitality	0.312	- 0.163	0.196	0.08
Mental Health	0.483*	- 0.414	0.136	0.144
Social Functioning	0.414*	- 0.173	0.197	- 0.002
Pain	0.529**	-0.221	0.242	- 0.025
General Well Being	0.416*	- 0.121	0.234	- 0.054

+p=0.068, *p<0.05, **p<0.01, ***p<0.001

Figures

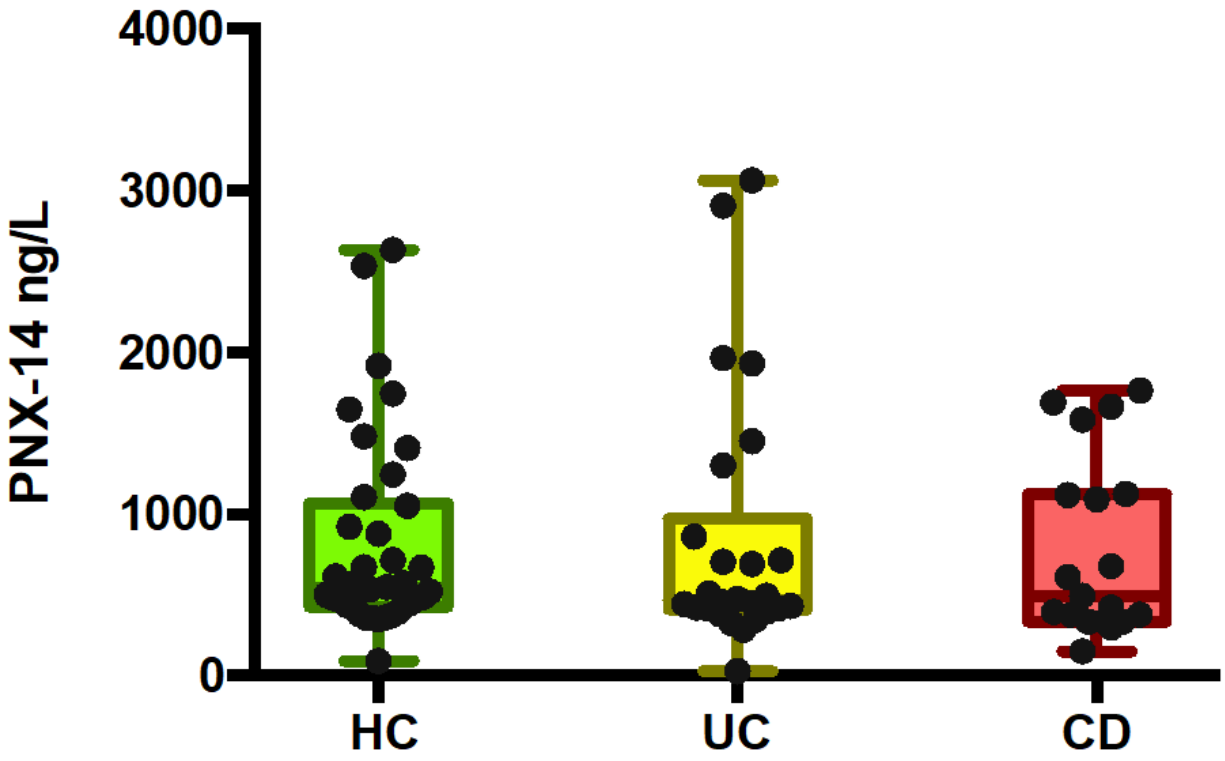


Figure 1

Serum Phoenixin-14 levels in the study groups. Serum PNZ-14 levels were similar. PNX: Phoenixin, HC: Healthy Control, UC: Ulcerative Colitis, CD: Crohns' Disease.

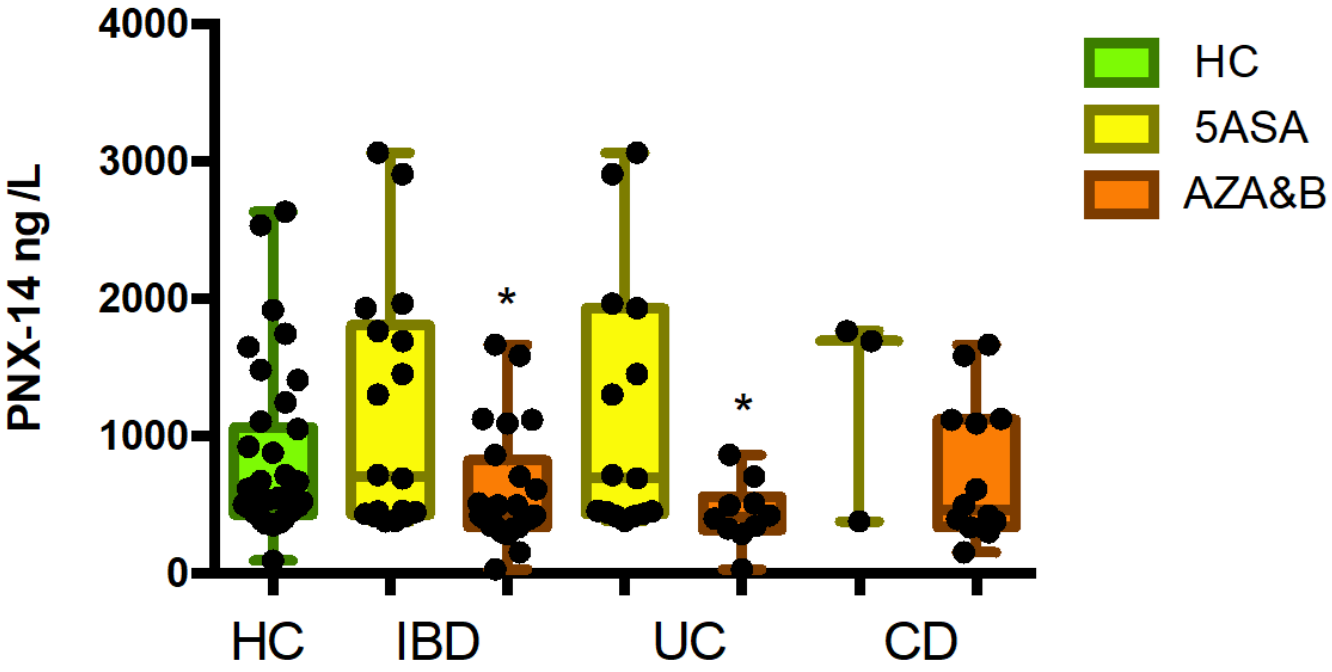


Figure 2

Serum PNX-14 levels in the study groups according to treatment modality. Serum PNX-14 levels were lower in patients who on azathioprine and or biological therapy (AZA&B) as compared to patients who on only 5ASA therapy. * $p<0.05$ as compared to respective treatment group, Mann Whitney U test PNX: Phoenixin, HC: Healthy Control, UC: Ulcerative Colitis, CD: Crohns' Disease.

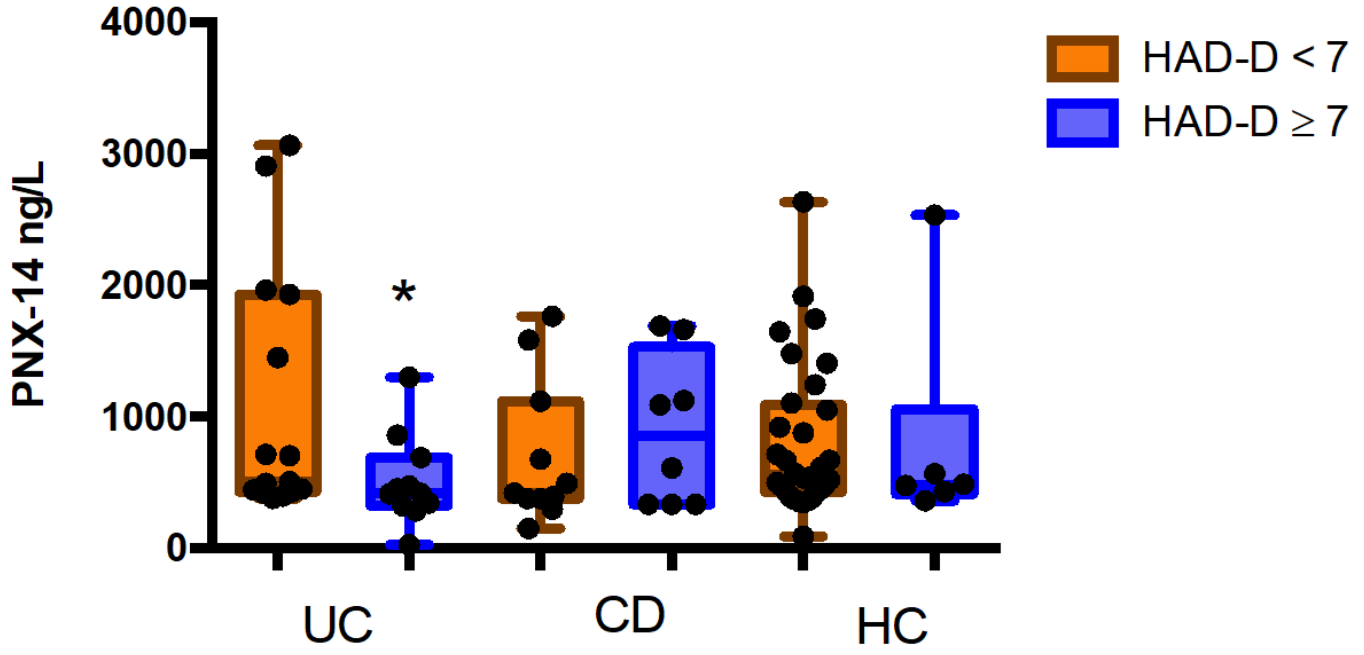


Figure 3

Serum PNX-14 levels in the study groups according to depression scores. Serum PNX-14 levels were lower in UC patients who had HAD-D score less than 7 as compared to those of UC patients who had HAD-D score equal or higher than 7. * $p<0.05$, Mann Whitney U test. PNX: Phoenixin , HC: Healthy Control, UC: Ulcerative Colitis, CD: Crohns' Disease, HAD-D Hospital Anxiety and Depression Questionnaire Depression sub-score.

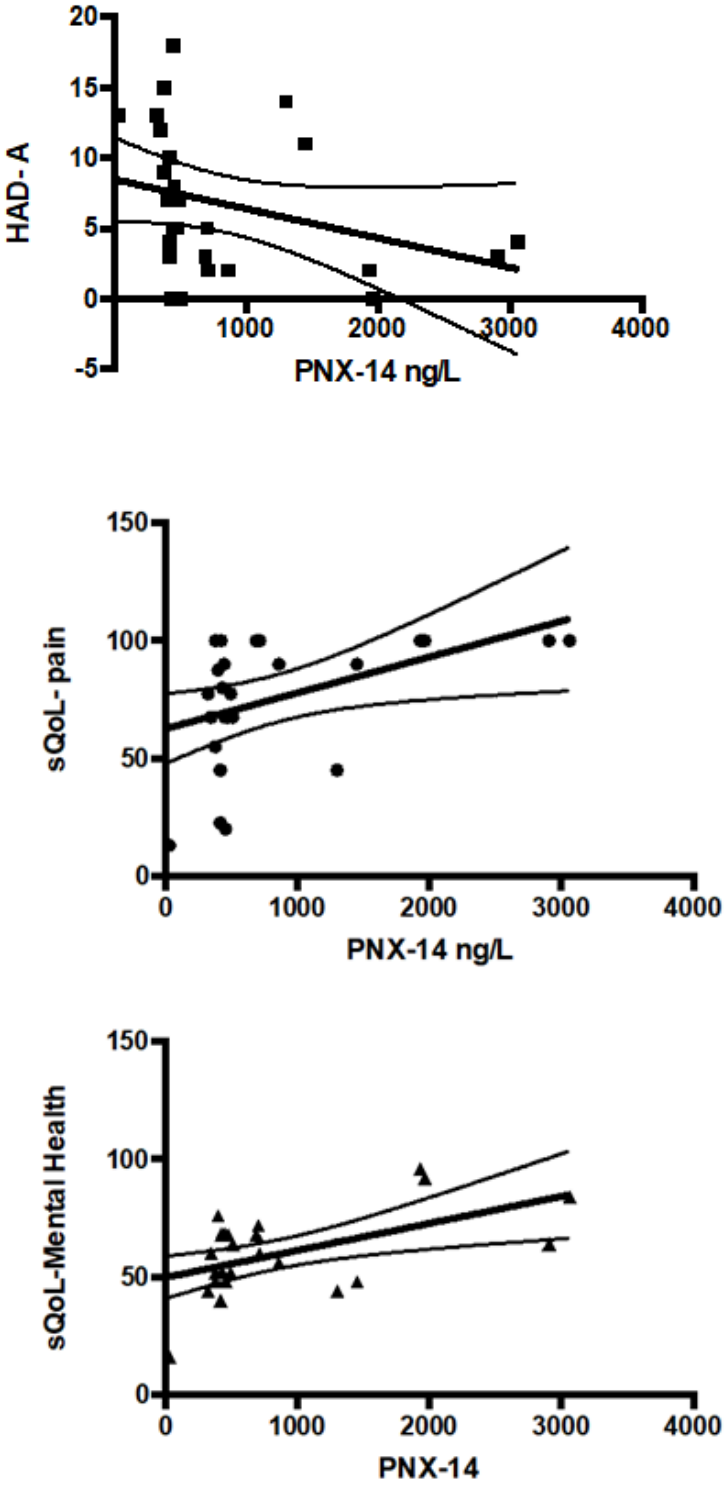


Figure 4

Correlation between serum PNX-14 levels and (A) HAD-A anxiety scores, (B) sQoL-pain subscores (C) sQoL – mental health sub-scores in patients who had UC. Serum PNX-14 levels correlated with (A) HAD anxiety score (r=0.05, p=0.09), (B) sQoL pain subscore (r=0.53, p=0.05) and (C) sQoL mental health sub-score (r=0.48, p=0.013) in patients who had UC. HAD-A :Hospital Anxiety and Depression Questionnaire Anxiety sub-score, sQOL: short form of Quality of Life questionnaire, PNX: Phoenixin

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryTable1.docx