Journal of Experimental and Clinical Medicine
https://dergipark.org.tr/omujecm

Research Article

J Exp Clin Med
2025; 42(4): 388-393
doi: 10.52142/omujecm.42.4.5

Pentraxin-3 in central nervous system infections: A promising but limited biomarker?

Berfin ÇİRKİN DORUK[1,*], Yusuf ÖNLEN [2], Serdar DOĞAN [3], Tayibe BAL[4]

[1]Department of Infectious Disease and Clinical Microbiology, Mersin City Hospital, Mersin, Türkiye
[2]Department of Infectious Disease and Clinical Microbiology, Faculty of Medicine, Mustafa Kemal University, Hatay, Türkiye
[3]Department of Biochemistry, Faculty of Medicine, Mustafa Kemal University, Hatay, Türkiye
[4]Department of Infectious Disease and Clinical Microbiology, Faculty of Medicine, Abant İzzet Baysal University, Bolu, Türkiye

Received: 15.05.2025 • **Accepted/Published Online:** 16.12.2025 • **Final Version:** 31.12.2025

Abstract

Central nervous system (CNS) infections lead to high morbidity and mortalitty rates, so there is a need for rapid and reliable biomarkers in diagnosis. The aim of this study was to determine the diagnostic value of Pentraxin-3 (PTX3) levels in CNS infections and to compare these with classic parameters. The study included 42 adult patients diagnosed with CNS infection between July 2020 and August 2021. The cerebral spinal fluid (CSF) and serum PTX3 levels were measured with the ELISA method. The findings were compared with clinical and laboratory parameters. The diagnostic performance was evaluated with ROC curve analysis. Bacterial meningitis was diagnosed in 39.0% of the patients, viral encephalitis in 51.2%, tuberculous meningitis in 7.3%, and fungal meningitis in 2.4%. The mortality rate was determined to be 43.9%. The CSF PTX3 levels were found to be higher in the bacterial cases, but with no significant difference between the serum and CSF PTX3 levels (p=0.327). A negative correlation was determined between CSF PTX3 and the Glasgow Coma Score, and a positive correlation between CSF PTX3 and the CSF protein level. In the ROC analyses, the strongest parameters for differentiation of bacterial and aseptic meningitis were seen to be CSF cell count (AUC: 0.888) and PMN percentage (AUC:0.882, while CSF PTX3 (AUC:0.586) and serum PTX3 (AUC:0.561) had the lowest differentiating power. PTX3 can be measured in CNS infections and higher levels are seen in bacterial meningitis cases. However, according to the ROC analysis results the diagnostic power is limited and these parameters lag behind classic CSF parameters. Therefore, PTX3 alone is not sufficient, but when combined with other biomarkers it can contribute to the clinical diagnostic process. The specific contribution of this study is that by evaluating both CSF and serum PTX3 levels simultaneously a relationship was determined between local and systemic inflammation.

Keywords: central nervous system infections, Pentraxin-3, ROC analysis, cerebral spinal fluid, biomarker

1. Introduction

Central nervous system (CNS) infections are a significant cause of morbidity and mortality throughout the world, especially in low and middle-income countries. According to 2016 data, encephalitis led to 103,000 deaths and 670.4 million Disability Adjusted Living Years (DALY), and meningitis led to 318,000 deaths and 21.9 million DALY (1). Early diagnosis and treatment are of critical importance in respect of increasing survival and preventing the development of sequelae.

The diagnosis of CNS infections is usually made from clinical findings and cerebral spinal fluid (CSF) analysis. Although culture, polymerase chain reaction (PCR), and biochemical analyses are valuable in diagnosis, they are not always sufficient (2). Therefore, various biomarkers have come to prominence in recent years with the aim of increasing sensitivity and specificity. From acute phase reactants, the markers most frequently used for this purpose include C-reactive protein (CRP) and procalcitonin (PCT) (3).

Pentraxin-3 (PTX3), belonging to the long pentraxin family, is an important effector molecule of the innate immune system. Although not found in the CNS under normal conditions, it is synthesised locally following stimulation of proinflammatory cytokines and microbials. The primary production sources are monocytes, macrophages, dendritic cells, endothelial cells, and glial cells in neural tissue. These properties make PTX3 a rapidly elevating biomarker in the early stages of inflammatory response (4, 5). In recent years, CSF PTX3 levels have beeen shown to be associated with various neurological diseases. It has been reported that PTX3 levels are increased in subarachnoid hemorrhage, ischaemic stroke, traumatic brain injury, epilepsy, multiple sclerosis, and Parkinson's disease (6). However, there are few studies that have investigated an increase in PTX3 in CNS infections. The available data have shown that CSF PTX3 levels are significantly higher in bacterial meningitis cases than in aseptic

*Correspondence: berfin.cirkin@hotmail.com

meningitis cases (7). Some research has also shown that CSF PTX3 levels could be correlated with disease severity, symptom duration, length of stay in hospital and classic inflammatory markers such as CRP (8).

In this context, PTX3 has emerged as a potential biomarker for both the differentiation of bacterial/aseptic meningitis and for understanding the pathophysiology of CNS infections (9).

A specific aspect of this study is the simultaneous evaluation of both CSF and serum PTX3 levels in patients with CNS infection. The data available in literature related to PTX3 have mostly focused on plasma levels or a limited number of CSF measurements and there are very few studies that have compared the two parameters. As this study was conducted during the COVID-19 pandemic, it also presents a different perspective in terms of difficulties in patient management and mortality rates. Therefore, the results obtained in this study can be considered to make a specific contribution to the literature by not only determining the diagnostic value of PTX3 but also by revealing the potential relationship between disease severity and mortality.

2. Material and Method

2.1. Patients and Samples

Study Group

This study included 41 patients, aged 18-65 years, who presented at Hatay Mustafa Kemal University, Health Practice and Research Hospital between July 2020 and August 2021, and had a confirmed diagnosis of CNS infection. At the beginning of the study it was aimed to have a sample size of 50 patients and 50 healthy control subjects, but because of the COVID-19 pandemic, these numbers could not be reached and so a control group was not included in the study.

Study Exclusion Criteria

Cases with the following characteristics were excluded from the study:

- Age <18 years
- The presence of neurological diseases that can mimic CNS infection (eg. autoimmune encephalitis, multiple sclerosis, vasculitis)
- The presence of systemic inflammatory or autoimmune diseases (eg. systemic lupus erythematosus, rheumatoid arthritis) which could affect PTX3 levels independently of CNS infection
- A confirmed infection with a focus other than the CNS determined during presentation
- Incomplete clinical or laboratory data, which would be required for the analyses
- Those with incomplete medical records or whose data were not available for ethical reasons

2.2. Definitions

- ***Acute bacterial meningitis****:* The isolation of bacteria from CSF with culture and/or molecular methods or the determination of features consistent with bacterial infection in CSF analysis (CSF cell count ≥250 cells/mm³, ≥60% polymorphonuclear cell predominance and low CSF/serum glucose ratio).
- ***Aseptic meningoencephalitis****:* Cases with no production in culture and no agent seen in gram staining despite an increase determined in cell count in CSF. In this case, viruses, microbacteria, fungi, and parasites may play a role as the agent. Diagnosis is confirmed with molecular amplification and/or serological tests.

2.3. Collection and Storage of CSF and Blood Samples

- ***Blood Samples***: An 8mL venous blood sample was collected from each patient into yellow-capped biochemistry tubes with gel. After leaving the samples for 20 mins for clotting, they were centrifuged at 4000 rpm for 10 mins. Supernatants were separated into Eppendorf tubes and stored at -80°C until the day of assay.
- ***CSF Samples****:* The CSF samples obtained with lumbar punction were placed in biochemistry tubes. In the same way as for the blood samples supernatants were separated by centrifuging at 4000 rpm for 10 mins and the samples were then stored at -80°C.

2.4. Measurement of CSF and Serum Pentraxin-3

The PTX3 levels were measured using a commercial kit and the Sandwich ELISA method (FineTest PTX-3 ELISA Kit, Cat. No: EH0263). Optic density was determined with spectrophotometry at 450nm wavelength. Concentrations were determined with a 4P-logistic calibration curve. The analysis range was 0.313-20 ng/mL, sensitivity: 0.188 ng/mL, precision: intra-assay CV<8%, inter-assay CV:<10%. The CSF samples were examined at 1/10 dilution and the results were stated as ng/mL units.

2.5. Statistical Analysis

Data obtained in the study were analyzed statistically using SPSS vn. 22.0 software (IBM Corp., Armonk, NY, USA). Conformity of cotinuous variables to normal distribution was examined with the Kolmogorov-Smirnov test. When data were not normally distributed, non-parametric methods were used. Descriptive statistcs were stated as mean ± standard deviation or median (minimumm-maximum) values for continuous variables and as number (n) and percentage (%) for categorical variables. In comparisons between the groups, the Mann-Whitney U -test was used, and in multiple group comparisons, the Kruskal-Wallis test. Categorical variables were evaluated with the Chi-square or Fisher's Exact test. Relationships between the variables were examined with Spearman correlation analysis and correlation coefficients were interpreted as weak:0.00-0.24, moderate:0.25-0.49, strong:0.50-0.74, and very strong: 0.75-1.00.

Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate diagnostic performance and the Area Under Curve (AUC) values were compared. A value of $p<0.05$ was accepted as statistically significant in all the analyses.

3. Results

Evaluation was made of a total of 41 patients diagnosed with CNS infection, comprising 27 (65.9%) females and 14 (34.1%) males with a mean age of 49.3±19.1 years (range, 17-84 years). Acute bacterial meningitis was diagnosed in 16 patients (39.0%), viral encephalitis in 21 (51.2%), tuberculous meningitis in 3 (7.3%), and fungal meningitis in 1 (2.4%). The overall mortality rate was determined to be 43.9% (n=18) and sequelae were observed in 22.0% of the surviving patients. Antibiotic use was recorded in 19.5% of the patients and findings of fever were determined in 29.3%.

Production was determined in 12 patients with bacterial infection [*Acinetobacter spp* (5)., *S. Epidermidis* (3), *Pseudomonas spp*. (1), *S. Maltophilia* (1), *S. Pneumoniae* (1), *ESBL+ E. Coli* (1)], and viral infection was diagnosed with molecular or serological tests in the patients with meningoencephalitis. In the single patient with fungal infection, *Cryptococcus neoformans* production was determined. In the single patient with tuberculous infection, *mycobacterium avium complex* production was obtained with the BACTEC 460TB fluid culture system.The clinical characteristics of the patients included in the study are presented in Table 1.

Table 1. Clinical characteristics of the patients

Variables	Patients (n = 41)
Antibiotic use prior to admission (n, %)	
Yes	8 (19.5)
No	33 (80.5)
Glasgow Coma Scale score, median (min–max)	14 (6–15)
Body temperature, °C, median (min–max)	36.5(36.0-38.5)
Fever (>37 °C), n (%)	12 (29.7)
Final diagnosis, n (%)	
Viral encephalitis	21 (51.2)
Acute bacterial meningitis	16 (39.0)
Fungal meningitis	1 (2.4)
Tuberculous meningitis	3 (7.3)
Etiological classification, n (%)	
Aseptic meningoencephalitis	25 (60.9)
Bacterial meningitis	16 (39.1)
In-hospital mortality, n (%)	
Survival	23 (56.1)
Exitus	18 (43.9)
Clinical outcome, n (%)	
Sequela	9 (22.0)
No sequela	14 (34.1)
Exitus	18 (43.9)

n:Number, Mean:Average, SD:Standard Deviation. Notes: Exitus refers to death occurring during the index hospitalization.

In the evaluation of the CSF parameters, the CSF cell count and polymorphonuclear (PMN) percentage were determined to be significantly high in the bacterial meningitis group ($p<0.001$). The CSF protein levels were significantly high in the bacterial group ($p=0.041$). The CSF glucose levels were observed to be lower in the bacterial group but not at a statistically significant level ($p=0.124$). No significant difference was seen between the groups in respect of the CSF PTX3 levels ($p=0.371$). The comparisons of the CSF parameters of the patients in the study are presented in Table 2.

When the blood parameters were examined, the WBC, CRP, and PCT levels showed a tendency to be higher in the bacterial meningitis group, with a significant difference determined only in the CRP levels ($p=0.036$). No significant difference in the serum PTX levels was observed between the groups ($p=0.041$). The blood parameters of the patients are shown in Table 3.

In the comparisons of the serum and CSF PTX3 levels, the CSF PTX3 values were found to be higher [CSF PTX3 median (min–max): 10.11(0.34–171.0) ng/mL; serum PTX3: 0.41 (0.27–6.48) ng/mL]. This difference was not statistically significant ($p=0.327$). The detailed comparison is shown in Fig. 1.

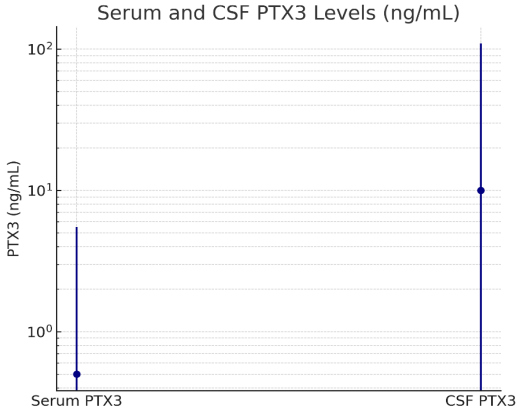


Fig. 1. Comparisons of the Serum and CSF PTX3 levels

The serum and CSF PTX3 levels of the patients according to clinical characteristics are shown in Table 4. No statistically significant difference was determined between the serum PTX3 and CSF PTX3 levels in respect of mortality and the development of sequelae ($p>0.05$).

The data related to relationships between the CSF PTX3 levels and the clinical characteristics, CSF, and blood parameters are presented in Table 5. A moderate-level negative correlation was determined between CSF PTX3 levels and the Glasgow Coma Score ($r=-0.354$, $p=0.023$). A moderate-level positive correlation was determined between CSF PTX3 levels and CSF protein levels ($r=0.322$, $p=0.040$). No significant relationships were found between the other parameters.

Table 2. CSF and blood parameters (Median, Min-Max)

Variables	Bacterial	Fungal	Tuberculous	Viral	*p* (Kruskal–Wallis)
CSF cell count (cells/mm³)	400 (80–80,000)	224 (224–224)	15 (0–175)	0 (0–500)	<0.001
PMN (%)	83.5 (20–90)	10 (10–10)	8 (0–12)	0 (0–65)	<0.001
Mononuclear cells (%)	16.5 (10–80)	90 (90–90)	88 (0–92)	0 (0–85)	0.153
CSF glucose (mg/dL)	50 (2–120)	6 (6–6)	58 (28–91)	67 (28–170)	0.124
CSF protein (mg/dL)	147.5 (19–419)	53 (53–53)	128 (30–142)	49 (0.5–198)	0.041
CSF PTX3 (ng/mL)	10.91 (0.40–171.00)	89.90 (89.90–89.90)	3.89 (0.40–12.91)	9.06 (0.34–133.00)	0.371

Table 3. Comparisons of the blood parameters of the meningitis patients

Variables	Bacterial	Fungal	Tuberculous	Viral	*P* (Kruskal–Wallis)
WBC (cells/mm³)	12,440 (4,200–48,000)	2,140 (2,140–2,140)	9,330 (6,400–9,870)	11,900 (4,590–23,000)	0.121
CRP (mg/L)	94.0 (4.0–261.0)	3.0 (3.0–3.0)	13.0 (3.0–121.0)	39.0 (3.0–160.0)	0.036
Procalcitonin (ng/mL)	0.14 (0.00–21.74)	0.24 (0.24–0.24)	0.00 (0.00–0.01)	0.06 (0.00–1.88)	0.122
Serum PTX3 (ng/mL)	0.53 (0.32–6.48)	0.40 (0.40–0.40)	0.32 (0.31–0.32)	0.40 (0.27–4.78)	0.041

WBC, white blood cell count; CRP, C-reactive protein; PTX3, pentraxin-3

Table 4. PTX3 levels according to clinical characteristics of patients

Variables		Serum PTX3 (Median, Min-Max)	p*	CSF PTX3 (Median, Min-Max)	p*
Mortality	Survival (n=23)	0.41 (0.31-3.63)	0.321	6.34 (0.34-119.47)	0.085
	Exitus (n=18)	0.46 (0.27-6.48)		11.25 (0.34-171.00)	
Sequela	Yes (n=9)	0.41 (0.31-1.26)	0.744	6.34 (0.40-45.78)	0.896
	No (n=13)	0.44 (0.32-3.63)		1.51 (0.34-119.47)	

n: Number, Min: Minimum, Max: Maximum, *Mann-Whitney U Test

In the ROC analysis performed to determine diagnostic performance, the CSF cell count (AUC: 0.888) and the PMN percentage (AUC: 0.882) were found to be the strongest parameters in the differentiation of bacterial and aseptic meningitis. CSF protein (AUC:0.746) and CSF glucose (AUC: 0.693) were found to have differentiating power at a moderate level. Of the serum parameters, CRP (AUC:0.638) showed limited performance, and the serum and CSF PTX3 levels were found to be low in terms of differentiating power (AUC: 0.561 and 0.586, respectively). The differentiating power of WBC was not significant (AUC: 0.490) (Fig. 2).

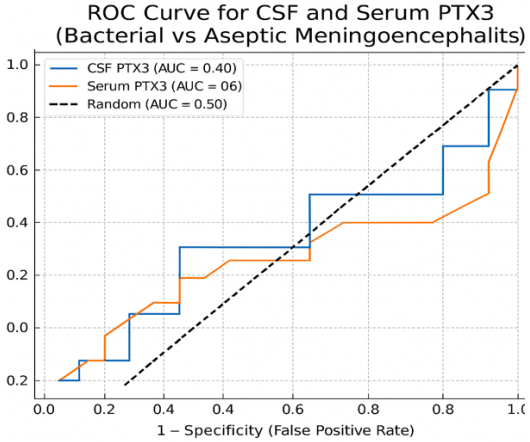


Fig. 2. ROC curve analysis of the CSF and serum PTX3 levels

Table 5. Relationship between CSF pentraxin-3 levels and clinical, CSF and blood parameters

Variables	CSF PTX-3	
	r	*p*
Age	0.054	0.737
GCS score	**-0.354**	**0.023***
Fever	0.092	0.567
Cells/mm³	0.270	0.087
Glucose (mg/dL)	-0.260	0.100
Protein (mg/dL)	**0.322**	**0.040**
PMN (%)	0.269	0.089
Mononuclear Cells (%)	0.181	0.256
WBC	-0.038	0.814
CRP	0.028	0.862
Procalcitonin	0.110	0.492
Serum Pentraxin-3	0.020	0.903

GCS: Glasgow Coma Scale score, PMN: Polymorphonuclear Leukocyte, WBC: White Blood Cell, CRP: C-Reactive Protein, Test: Spearman correlation analysis, r: Spearman correlation coefficient, *p<0.05

4. Discussion

Central nervous system (CNS) infections continue to be a serious health problem because of diagnostic difficulties and high morbidity and mortality rates. In this study, the CSF and serum PTX3 levels were evaluated together and were

compared with classic biomarkers with the aim of determining diagnostic value.

The demographic data of the patients in this study showed a general similarity to that of patient groups with CNS infection reported in the literature. However, the mortality rate (43.9%) was higher than expected. The most important reasons for this can be said to be that the study was conducted during the COVID-19 pandemic, the patient group had a severe clinical course, there was widespread use of immunnomodulator treatments, and there was restricted intensive care unit capacity. All of these factors could have had a negative effect on the mortality rate.

Experimental and clinical data have shown that PTX3 expression is not seen in the brain under normal conditions, but it is seen to have been produced by glial and endothelial cells after traumatic, vascular, or infectious stimuli (9). Previous studies have reported that CSF PTX3 levels are significantly higher in bacterial meningitis cases compared to cases of viral and aseptic meningitis (7, 8, 10). The current study results are consistent with those findings as CSF PTX3 levels were seen to be higher in bacterial cases.

The CSF PTX3 levels in the current study were determined to be negatively correlated with the Glasgow Coma Score and positively correlated with the CSF protein level. This suggests that PTX3 is not only diagnostic but may also be a marker reflecting disease severity. However, in the ROC analyses CSF PTX3 (AUC=0.586) and serum PTX3 (AUC=0.561) were found to have limited differentiating power, and lagged far behind the classic parameters of CSF cell count and PMN percentage. This demonstrates that PTX3 alone is not a reliable biomarker.

PTX3 has come to prominence as an assistive marker that could contribute to the diagnostic process when combined with biomarkers such as CRP and procalcitonin, when CSF findings are indeterminate or in conditions where classic parameters are not sufficient. In addition, that serum PTX3 levels were higher in bacterial cases shows that it could be included in the diagnostic process of the systemic inflammatory response. It has also been reported that PTX3 levels in sepsis patients are correlated with mortality (11).

The findings of the current study demonstrate that PTX3 can be measured in CNS infections and the fact that higher levels were observed in bacterial cases could be associated with disease severity. However, according to the ROC analysis results, the diagnostic value of PTX3 alone is low. Therefore, it can be concluded that PTX3 is an assistive parameter that can contribute to diagnostic algorithms when evaluated together with established biomarkers.

There were some limitations to this study, primarily that the planned number of patients could not be reached because of the COVID-19 pandemic, and therefore the study was limited to only 41 patients. The very low number of cases in the subgroups such as tuberculous and fungal meningitis weakened the statistical power of outcomes related to these groups.

The most important limitation of the study was the lack of a control group. This meant that there could be no comparisons of the CSF and serum PTX3 levels with the values of the normal healthy population and severely restricted the interpretation of the results obtained. As there was no control group, reference ranges for PTX3 could not be determined and therefore the diagnostic power of the biomarkers could not be fully established.

In addition, the fact that the study was conducted in a single centre limits the generalisability of the results. Only biomarker levels obtained in the acute period were evaluated and the relationship between PTX3 and the long-term prognosis could not be investigated. There is a need for further multicentre, prospective studies with larger samples and including a control group to be able to more clearly reveal the potential for clinical use of PTX3.

This research is one of the very few studies to have evaluated both CSF and serum PTX3 levels together in patients with CNS infection. The data obtained showed higher PTX3 levels in bacterial meningitis and these were correlated with parameters reflecting disease severity.

However, in the results of the ROC analysis, CSF PTX3 (AUC=0.586) and serum PTX3 (AUC=0.561) were found to have limited diagnostic power, and CSF cell count (AUC= 0.888) and PMN percentage (AUC=0.882) emerged as much stronger markers. This finding shows that PTX3 alone is not a reliable diagnostic test but could have clinical value within a combined biomarker approach.

A specific contribution of this study was the comparison of local and systemic inflammation by simultaneously measuring the CSF and serum levels of PTX3. This approach shows that PTX3 should not be used alone, but in combination with classic biomarkers such as CRP and procalcitonin it can be integrated into the diagnostic algorithms for CNS infections.

Conflict of interest

The authors have no conflict of interests to declare.

Funding

This study was supported by the Scientific Research Project Committee of Hatay Mustafa Kemal University (Project number: 20.U.016).

Acknowledgments

None to declare.

Authors' contributions

Concept: B.Ç.D., Y.Ö., S.D., T.B., Design: B.Ç.D., Y.Ö., S.D., T.B., Data Collection or Processing: B.Ç.D., Y.Ö., S.D., T.B., Analysis or Interpretation: B.Ç.D., Y.Ö., S.D., T.B., Literature Search: B.Ç.D., Y.Ö., S.D., T.B., Writing: B.Ç.D., Y.Ö., S.D., T.B.

Ethical Statement

The study was conducted with the permission of the Clinical Research Ethics Committee of Hatay Mustafa Kemal University Medical Faculty (deecision no: 2020/93).

References

1. Brindle HE, Choisy M, Christley R, et al. The global burden of central nervous system infections: a modelling study. Front Public Health. 2024;12:1371502. doi:10.3389/fpubh.2024.1371502
2. van Zeggeren IE, ter Horst L, Heijst H, Teunissen CE, van de Beek D, Brouwer MC. Neurofilament light chain in central nervous system infections: a prospective study of diagnostic accuracy. Sci Rep. 2022;12(1):14140. doi:10.1038/s41598-022-18450-9
3. Giuliano S, Flammini S, Giacobbe DR, et al. Role of procalcitonin and C-reactive protein in patients with bacterial meningitis. Eur J Clin Microbiol Infect Dis. 2022;41(3):387-94. doi:10.1007/s10096-021-04358-8
4. Bottazzi B, Inforzato A, Messa M, et al. The pentraxins PTX3 and SAP in innate immunity, regulation of inflammation and tissue remodelling. J Hepatol. 2016;64(6):1416-27. doi:10.1016/j.jhep.2016.02.029
5. Garlanda C, Bottazzi B, Bastone A, Mantovani A. Pentraxins at the crossroads between innate immunity, inflammation, matrix deposition, and female fertility. Annu Rev Immunol. 2005;23:337-66. doi:10.1146/annurev.immunol.23.021704.115756
6. Bonacina S, Barbieri SS, Cutuli L, et al. Pentraxin 3 in cardiovascular and neuroinflammatory disorders. Front Immunol. 2022;13:841095. doi:10.3389/fimmu.2022.841095
7. Zatta M, Di Pietro A, D'Abramo A, et al. Cerebrospinal fluid pentraxin 3 in patients with meningitis: diagnostic and prognostic significance. Eur J Clin Microbiol Infect Dis. 2020;39(4):665-70. doi:10.1007/s10096-019-03759-7
8. El-Kady H, Mostafa MG, Hassan R, et al. Cerebrospinal fluid pentraxin 3 as a biomarker for bacterial meningitis in children. BMC Pediatr. 2025;25(1):7. doi:10.1186/s12887-024-05065-1
9. Porte R, Davoudian S, Asgari F, et al. The long pentraxin PTX3 as a key component of humoral innate immunity and a candidate diagnostic/prognostic biomarker in infectious and inflammatory diseases. Front Immunol. 2019;10:794. doi:10.3389/fimmu.2019.00794
10. Thomsen MM, Munthe-Fog L, Garred P, et al. Pentraxin 3 levels in cerebrospinal fluid as a diagnostic marker of bacterial meningitis. PLoS One. 2023;18(3):e0282004. doi:10.1371/journal.pone.0282004
11. Hamed GM, Megahed NA, Abdelhakeem G, et al. Plasma pentraxin 3 in patients with sepsis: relationship with severity and outcome. BMC Infect Dis. 2017;17:554. doi:10.1186/s12879-017-2663-0.