International Journal of Emergency Medicine

REVIEW

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Exploring the pentraxin-3 as a prognostic biomarker in paraquat poisoning: a systematic-narrative hybrid review

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Abstract

Purpose Paraquat is a commonly used pesticide that is highly toxic to humans and leads to fatal lung fibrosis upon consumption. Due to its rapid clearance from the bloodstream, there are no reliable biomarkers for the diagnosis and prognosis of paraquat intoxication. Pentraxin 3 (PTX3), an acute-phase inflammatory mediator, has emerged as a potential biomarker, particularly in the context of lung injury and fibrosis. This review aimed to assess the prognostic value of the level of PTX3 in predicting clinical outcomes in patients with paraquat poisoning and to evaluate the correlation between PTX3 levels and the severity of poisoning.

Methods A comprehensive literature search was conducted using PubMed, MEDLINE (Ovid), EMBASE (Ovid), Web of Science, Scopus, CROSSREF, and Google Scholar. The included studies were observational (cohort or case-control), involving human subjects with confirmed paraquat poisoning, and reported PTX3 levels related to clinical outcomes. Data on PTX3 expression, disease severity, and prognostic correlations were extracted.

Results Two primary studies were performed. PTX3 levels were significantly elevated in non-survivors compared to survivors and were positively correlated with serum paraquat levels and disease severity. PTX3 levels peaked between 12 and 24 h post-ingestion and maintained a prognostic value over subsequent days. A PTX3 cut-off of 8.9 ng/mL was associated with higher mortality, with moderate sensitivity and specificity.

Conclusion PTX3 is a promising prognostic biomarker for paraquat poisoning, particularly in cases where traditional markers are unreliable. Due to the limited sample size, a multicenter study is recommended to validate the role of PTX3 in clinical and forensic settings.

Keywords Paraquat, Poisons, Pentraxin 3, PTX3, Prognostic biomarker, Lung fibrosis

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Introduction

Paraquat dichloride (1,1′-dimethyl-4,4′-bipyridinium dichloride methyl viologen) is a bipyridyl (bipyridinium) herbicide compound widely used as a herbicide and is highly toxic to humans [1]. Although paraquat (PQ) has been banned in more than 67 countries, it continues to be used in some other regions, particularly in Asia and Latin America, where it is subject to tight restrictions [2]. Its extensive use in agricultural industries has resulted in a significant increase in the number of PQ poisoning incidents, both accidental and intentional [3]. When ingested through the gastrointestinal tract, it enters the bloodstream and primarily affects the lungs, causing acute lung injury and pulmonary fibrosis, which can lead to respiratory failure and is the primary cause of death [4]. Despite efforts to develop effective treatments for PQ poisoning, the mortality rate remains high at 80%, posing a significant challenge to healthcare professionals in severe cases [4].

PQ is rapidly absorbed through the gastrointestinal tract and is swiftly excreted in urine within 12 to 24 h [5–7]. The prediction of survival in cases of PQ intoxication is closely associated with the quantity ingested and the duration before treatment is administered. The development of effective diagnostic and prognostic tools is hindered by the rapid clearance of PQ from the bloodstream within hours [1, 3, 4, 6]. The difficulty in measuring serum PQ levels in cases with delayed reporting presents a significant challenge, thereby limiting clinicians' capacity to accurately diagnose and evaluate the prognosis [1, 3–7].

In recent years, pentraxin-3 (PTX3), a member of the long pentraxin family, has emerged as a promising biomarker. This acute-phase inflammatory glycoprotein mediator bridges the gap between the innate and adaptive immune systems [8, 9]. Studies have shown that the level of PTX3 is significantly increased in patients with lung fibrosis, the leading cause of mortality following PQ ingestion. It regulates inflammation, tissue healing, and remodelling of the extracellular matrix [10]. PTX3 is synthesised locally in inflammatory areas by diverse cells, including macrophages and endothelial cells, in response to proinflammatory stimuli [10]. It aids in the elimination of infections and apoptotic cells, assists in the transformation of macrophages from a pro-inflammatory (M1) to a reparative (M2) phenotype, and promotes the resolution of inflammation [10].

Animal studies have unequivocally demonstrated the dual role of PTX3 as both a marker of inflammation and fibrosis and as a modulator of lung injury severity. This provides compelling justification for investigating PTX3 as a prognostic biomarker in PQ poisoning, where oxidative stress, inflammation, and pulmonary fibrosis are central to the pathogenesis. Consequently, in recent years, PTX3 has emerged as a promising biomarker [11, 12].

There is strong evidence that PQ poisoning induces oxidative stress, systemic inflammation, and pulmonary fibrosis, all of which are pathways where PTX3 is biologically relevant. While PTX3 is not specific to paraquat, its role may still be important as a prognostic marker of severity, like C-Reactive protein (CRP) and interleukins (IL-6) are used in sepsis despite being non-specific [13–15]. Hence, PTX3 is a potential non-specific acute-phase biomarker for PQ poisoning [8–10].

Initially, the authors intended to conduct a systematic review on PTX3 as a biomarker for PQ poisoning, but they found only two relevant human studies. Due to the limited evidence, a hybrid narrative review was conducted, incorporating findings from related fields such as toxicology, pulmonary fibrosis, and Acute Respiratory Distress Syndrome. This study does not claim that PTX3 is a definitive marker for PQ poisoning, but rather to highlight the paucity of data and to emphasize PTX3 as a promising area for further exploration.

This study aimed to critically evaluate and synthesize the available evidence on the role of Pentraxin-3 (PTX3) as a prognostic biomarker in PQ poisoning, through a systematic review of the literature combined with narrative synthesis to investigate the research question: What is the potential of PTX3 as a prognostic biomarker for PQ poisoning?

Materials and methods

This study was initially planned and registered as a systematic review of 'PTX3 as a biomarker for PQ poisoning' in PROSPERO [16]. However, there are only two human-specific original studies available directly examining this association PTX3 and PQ poisoning. Due to the insufficient number of studies for a systematic analysis, a hybrid narrative review was chosen instead. This hybrid narrative review allows integration of these scarce data points with indirect evidence from toxicology, pulmonary fibrosis, and ARDS literature, where PTX3 has been studied as a prognostic biomarker.

This hybrid review was conducted to evaluate the potential of PTX3 as a prognostic biomarker in patients with confirmed PQ poisoning, with a focus on human studies. The study population included patients of any age or sex with a confirmed diagnosis of PQ poisoning. The intervention or exposure was defined as elevated levels of PTX3, measured in blood or serum samples. Studies with or without a comparator group were considered, including those comparing patients with differing PTX3 levels, other prognostic biomarkers, or healthy controls. The primary outcome of interest was the prognostic

Table 1 PRISMA flowchart:

Identification:
Records identified through database searching ($n=7$ databases, 523)
Records after duplicates removed ($n=412$)
Screening:
Records screened by title/abstract ($n=412$)
Records excluded ($n=385$)
Eligibility:
Full-text articles assessed for eligibility ($n=27$)
Full-text articles excluded, not meeting criteria ($n=23$)
Included:
Studies included in qualitative synthesis ($n=4$)
(Human studies = 2; Animal studies = 2)

value of PTX3 in predicting clinical outcomes such as severity, survival, or mortality in paraquat poisoning.

Studies were included if they were observational in design (cohort, case-control, or cross-sectional), involved human participants of any age or sex with confirmed PQ poisoning, measured PTX3 levels, and reported clinical outcomes such as mortality, survival time, or severity of poisoning. Exclusion criteria comprised animal studies, in vitro experiments, case reports, reviews, editorials, letters to the editor, and studies that either focused on biomarkers other than PTX3 or lacked relevant data on PTX3 levels and clinical outcomes. These inclusion and exclusion criteria were applied systematically to ensure consistency and reduce selection bias.

Results

Literature search results

A PRISMA flowchart has also been added to clearly depict the screening process and the final number of studies included (Table 1**).** A comprehensive literature search was carried out across multiple electronic databases, including PubMed, MEDLINE (Ovid), EMBASE (Ovid), Web of Science Core Collection, Scopus, CrossRef, Google Scholar and ProQuest. The search strategy employed a combination of Medical Subject Headings (MeSH) and relevant keywords such as "Paraquat poisoning," "Pentraxin 3," "PTX3," "Prognostic biomarkers," "Mortality," and "Survival period," using Boolean operators (AND, OR) to enhance the sensitivity and specificity of the results.

Two reviewers independently screened all titles and abstracts retrieved from the manual search to identify potentially eligible studies. Full-text articles that appeared relevant were then reviewed in detail for final inclusion. Any disagreements were resolved through discussion and consensus. Data were extracted using a standardized template and included details on study design, participant characteristics, PTX3 levels, details of PQ exposure, lung involvement, and reported associations between PTX3 and clinical outcomes. This structured yet flexible approach enabled the systematic identification of relevant literature and the narrative synthesis of emerging evidence, making it suitable for an area where research is limited and heterogeneous. Table 2

Characteristics of included studies

Two studies were identified as eligible for inclusion in this review. The first, conducted by Yeo et al. (2023), was an observational study involving 27 patients with paraquat poisoning, titled "The role of pentraxin-3 as a prognostic biomarker in paraquat poisoning." The second, by Zhang et al. (2023), was a prospective cohort study including 58 patients, titled "Prognostic Value of White Blood Cell Count, C-reactive protein, and Pentraxin-3 Levels in Patients with Acute Paraquat Poisoning." The main characteristics of these studies are summarized in Table 3.

Prognostic significance of PTX 3

Yeo et al. reported that maximal PTX3 concentrations were significantly higher in non-survivors (5.9 ± 3.7 ng/mL) compared to survivors (3.2 ± 2.6 ng/mL). In the same study, plasma paraquat levels were also found to be markedly elevated among non-survivors (40.2 ± 35.6 µg/mL) versus survivors (5.0 ± 6.6 µg/mL).

Zhang et al. (2023) further highlighted the prognostic utility of PTX3. The area under the ROC curve (AUC) values for PTX3 in predicting mortality were 0.48 (95% CI: 0.15–0.82) on Day 0, 0.73 (95% CI: 0.61–0.81) on Day 1, and 0.68 (95% CI: 0.56–0.75) on Day 2. Using a PTX3 cutoff value of 8.9 ng/mL, sensitivity was 67.7%,

Table 2 Table: database search results for studies on PTX3 in Paraquat poisoning

Database	Records Retrieved	After Removing Duplicates	Screened (Title/Abstract)	Included for Full-Text Review
PubMed	132	110	110	7
Scopus	94	81	81	5
Web of Science	88	73	73	4
Embase	72	61	61	3
Cochrane Library	41	28	28	2
Google Scholar	71	44	44	5
ProQuest	25	15	15	1
Total	523	412	412	

Table 3 Summary of studies investigating pentraxin 3 (PTX3) as a biomarker in Paraquat poisoning

Study	Study Design	Sample Size	Key Findings
Yeo et al. (2023)	Observational study	27	Maximal PTX3 levels significantly higher in non-survivors (5.9 ± 3.7 ng/mL) compared to survivors (3.2 ± 2.6 ng/mL). Plasma paraquat levels higher in non-survivors (40.2 ± 35.6 µg/mL) vs. survivors (5.0 ± 6.6 µg/mL).
Zhang Y et al. (2023)	Prospective cohort study	58	Day 0: AUC = 0.48 (95% CI: 0.15–0.82); Day 1: AUC = 0.73 (95% CI: 0.61–0.81, Day 2: AUC = 1.68 (95% CI 0.56, 0.75). PTX3 cutoff of 8.9 ng/mL: Sensitivity 67.7%, Specificity 76.1%, Youden Index 0.44. PTX3 levels consistently higher in non-survivors at all time points. Non-survivors: PTX3 peaked on Day 2, slight decline by Day 3. Survivors: Gradual, moderate rise in PTX3 levels.

PTX3 Pentraxin 3

AUC Area Under the Curve

CI Confidence Interval

specificity 76.1%, with a Youden Index of 0.44. PTX3 levels were consistently higher in non-survivors at all measured time points, with non-survivors peaking on Day 2 followed by a slight decline, while survivors demonstrated only a gradual, moderate increase.

Discussion

PQ poisoning results in multi-organ failure, predominantly affecting the lungs, which leads to interstitial inflammation, oedema, epithelial-mesenchymal transition, increased fibroblast proliferation, and extracellular matrix deposition, ultimately resulting in pulmonary fibrosis within days to weeks following exposure [17]. The inflammatory response to PQ toxicity is pivotal in the development of lung injury and systemic organ damage. Upon consumption, PQ triggers oxidative stress, resulting in cellular damage and the activation of inflammatory pathways [18]. PTX3, produced at sites of inflammation, provides a rapid and sensitive marker of tissue injury in PQ poisoning cases.

PTX3 is a plasma protein of the pentraxin superfamily that functions as a chemoattractant for various inflammatory cells [19]. PTX3 is localised intracellularly, particularly within neutrophil granules [20]. In murine models of bleomycin-induced lung fibrosis, PTX3 expression is elevated during the fibrotic process. Transgenic mice that overexpress PTX3 exhibit reduced fibroblast activation, lower collagen deposition, and decreased immune cell infiltration, indicating a protective function of PTX3 in reducing fibrotic tissue formation. In contrast, PTX3-deficient mice demonstrate increased fibrosis and reduced survival, underscoring the significance of endogenous PTX3 in regulating fibrotic responses [21].

In comparison, Chi et al. found that PTX3 facilitates lung fibrosis by stimulating the connective tissue growth factor (CTGF), transforming growth factor-beta (TGF-β), and WNT signalling pathways, resulting in fibroblast activation, collagen deposition, and extracellular matrix accumulation. It is significantly expressed in fibrotic lung tissues and facilitates disease development by augmenting inflammation and fibrocyte differentiation. PTX3 directly interacts with CD44, a surface receptor on fibroblasts, thereby enhancing fibroblast activation and fibrotic signalling. Inhibition of PTX3 in bleomycin-induced lung damage models markedly diminishes fibrosis [22]. The above studies suggest a potential dual role for PTX3 in the development of pulmonary fibrosis. Depending on the molecular context and cellular interactions, it may act both as a protective factor and a contributor to fibrotic progression. However, further studies are needed to determine the exact mechanisms and fully understand the role of PTX3 in fibrogenesis.

Experimental evidence from animal models strongly supports the role of PTX3 as a mediator and potential biomarker of lung injury, with clear translational relevance to PQ poisoning. In bleomycin-induced pulmonary fibrosis, PTX3 expression was shown to rise significantly during fibrogenesis, with PTX3-overexpressing mice exhibiting reduced fibroblast activation and collagen deposition, whereas PTX3-deficient mice developed severe fibrosis and poorer survival outcomes [11]. Similarly, in lipopolysaccharide (LPS)-induced acute lung injury, PTX3 levels in bronchoalveolar lavage fluid closely correlated with the severity of tissue damage and activation of inflammatory cascades [12]. Further, mechanistic studies demonstrated that PTX3 can drive fibrosis through CD44-mediated pathways, with PTX3 neutralization reducing fibrosis and improving survival. In models of viral-induced lung injury, PTX3 was protective, as its deficiency aggravated inflammation and tissue injury, while exogenous administration attenuated neutrophilic infiltration and improved lung pathology [22]. Hence, together, these studies underscore dual role of PTX3 as a marker of both inflammation and fibrosis, and a modulator of lung injury severity, providing strong justification for exploring PTX3 as a prognostic biomarker in PQ poisoning, where oxidative stress, inflammation, and pulmonary fibrosis are central to the pathogenesis.

Under normal conditions, the level of PTX3 in humans is extremely low (< 2 ng/mL), but it increases substantially in inflammatory conditions [13, 14]. A study by Yeo et al. observed that the mean maximal value of PTX3 in the lung fibrosis group was 7.6 ± 2.1 ng/ml [8]. The PTX3

level starts to rise gradually following ingestion of PQ and peaks between 12 and 24 h on day 1 [15]. This makes PTX3 an ideal and effective alternative to serum PQ levels in the diagnosis and prognosis of PQ poisoning.

The study by Zhang et al. demonstrated the prognostic value of PTX3 using AUC analysis to predict clinical outcomes in PQ poisoning. The findings indicated that PTX3 had limited predictive value on day 0, with an AUC of 0.48 (95% CI: 0.15–0.82). However, its prognostic accuracy improved significantly on day 1, with an AUC of 0.73 (95% CI: 0.61–0.81), suggesting that PTX3 may serve as a more reliable early marker of outcome when measured 24 h after exposure [15]. Additionally, a PTX3 cut-off value of 8.9 was identified for the non-survivor group, demonstrating a sensitivity of 67.7%, a specificity of 76.1%, and a Youden index of 0.44 [15]. The higher specificity and Youden index further support the potential of PTX3 as a reliable prognostic marker in PQ poisoning. Unlike serum PQ levels, which can decline rapidly due to fast elimination from the body, making them difficult to detect as time progresses, PTX3 may offer a more stable and practical indicator for predicting patient outcomes.

Yeo et al. explored the relationship between PTX3 levels and the severity of PQ poisoning by comparing plasma PTX3 concentrations with PQ levels in both survivors and non-survivors. The mean plasma PQ concentration was significantly higher in non-survivors (40.2 ± 35.6 μg/mL) compared to survivors (5.0 ± 6.6 μg/mL). Similarly, PTX3 levels were elevated in non-survivors (5.9 ± 3.7 ng/mL) compared to survivors (3.2 ± 2.6 ng/mL), suggesting a potential association between increased PTX3 expression and disease severity, as well as poor clinical outcomes [8]. This finding reinforces the observation by Zhang Y et al. [15], who reported that PTX3 levels were directly proportional to the severity of poisoning and serum PQ concentration, highlighting its potential as a valuable alternative biomarker when PQ levels are unavailable or unreliable.

Zhang Y et al. [15] observed that although all patients received standard treatment for PQ poisoning, PTX3 levels consistently increased from day 0 to day 3. Their study revealed a distinct temporal pattern, showing that PTX3 concentrations were consistently elevated in non-survivors relative to survivors at all assessed periods. In non-survivors, PTX3 levels were elevated on day 0, peaked on day 2, and then declined marginally by day 3. In contrast, survivors had a more gradual and moderate rise in PTX3 levels during the same timeframe. The continual rise in non-survivors indicates a significant correlation between elevated PTX3 levels and unfavourable clinical outcomes. The findings further support the potential of PTX3 as a predictive biomarker in PQ poisoning, especially when direct assessment of serum PQ levels is impractical due to its fast removal from the bloodstream.

In addition to PTX3, other prognostic scoring systems such as APACHE II have been evaluated in PQ poisoning. A systematic review and meta-analysis by Kaur et al. highlighted the significance of APACHE II in predicting mortality in paraquat poisoning [23]. While this study did not directly compare PTX3 with APACHE II, the findings reinforce the importance of exploring reliable prognostic markers in this context. Our review complements this evidence by highlighting the emerging role of PTX3 as a potential biomarker. Combining PTX3 with existing scoring systems may aid in improved prediction and early detection of adverse outcomes.

Clinical and medicolegal implications

PQ is rapidly but incompletely absorbed and then largely eliminated unchanged in urine within 12–24 h. So, there is a need for alternative diagnostic and prognostic methods for PQ ingestion [7]. Based on the studies reviewed above, PTX3 appears to be a reliable alternative to plasma PQ levels for assessing the severity of poisoning and predicting outcomes. This may be especially important in cases where the ingested dose is unknown, as PTX3 levels seem to correlate with the amount consumed and the associated tissue injury. Serum PTX3 level may provide significant clinical information regarding the treatment plans for PQ toxicity. Specifically, risk stratification using early PTX3 measurements may facilitate the identification of individuals at a higher risk for severe outcomes, enabling more prompt and targeted therapeutic measures. Moreover, continuous monitoring of PTX3 levels may prove beneficial in assessing disease progression or the efficacy of therapy, particularly when conventional chemical analysis yields ambiguous results.

Also, the case fatality rate is significantly high in PQ poisoning, with mortality ranging from 50% to 90%. In instances of deliberate self-poisoning with concentrated formulations, mortality reached nearly 100% [24]. Consumption of large amounts of liquid concentrate (> 50–100 ml of 20% ion w/v) leads to acute organ failure, including pulmonary oedema, cardiac, renal, and hepatic failure, as well as convulsions attributable to central nervous system involvement. Mortality occurs due to multiorgan failure, which occurs within hours to a few days. And consumption of minimal amounts typically results in toxicity in the kidneys and lungs, manifesting within the subsequent 2–6 days. Mortality rate in this group exceeds 50% [7]. This represents a significant proportion of autopsy cases related to deaths caused by poisoning. Since all poisoning cases are medicolegal in nature, an autopsy is routinely performed. An important challenge faced by forensic pathologists in such cases is a negative chemical analysis report of the viscera, particularly in delayed deaths involving rapidly eliminated toxins such as PQ. In these instances, measuring serum PTX3 levels

may serve as a valuable supplementary diagnostic tool alongside chemical analysis and histopathological examination in suspected PQ poisoning. However, to establish the postmortem utility of this biomarker, further studies are warranted.

Limitations

The role of PTX3 in PQ poisoning has not been adequately explored, as there are only a few studies available, the majority of which are constrained by small sample sizes. Despite PQ being one of the most frequently ingested herbicides in poisoning cases, there is a significant lack of human studies that explore the diagnostic or prognostic utility of PTX3 in this context. A multicentric study involving diverse patient cohorts and serial estimation of PTX3 levels with a large sample size is recommended to determine a cutoff level associated with poor outcomes accurately and to establish a universally accepted reference standard for assessing severity and prognosis in PQ poisoning. In addition to clinical evaluation, there is a critical need to explore the postmortem utility of PTX3 through well-designed autopsy-based studies. Such research could help validate PTX3 as a reliable biomarker in both the ante-mortem and postmortem settings, particularly in cases where chemical analysis yields negative results due to delayed presentation or rapid toxin elimination.

Conclusion

Paraquat poisoning continues to pose significant clinical and forensic challenges due to its high mortality and rapid toxicity. In the studies reviewed, maximal PTX3 levels were significantly higher in non-survivors compared to survivors (Yeo et al.: 5.9 ± 3.7 ng/mL vs. 3.2 ± 2.6 ng/mL), and plasma paraquat concentrations were similarly elevated in non-survivors (40.2 ± 35.6 µg/mL vs. 5.0 ± 6.6 µg/mL). PTX3 levels in non-survivors showed a distinct temporal pattern, peaking on Day 2 and remaining consistently higher than in survivors (Zhang et al.). Using a PTX3 cutoff of 8.9 ng/mL, the sensitivity was 67.7%, the specificity was 76.1%, and the Youden Index was 0.44, highlighting its prognostic relevance. These findings indicate that PTX3 may serve as a reliable biomarker for predicting severity and mortality in paraquat poisoning. Furthermore, combining PTX3 measurements with established scoring systems such as APACHE II could enhance early risk stratification and improve the prediction of adverse outcomes. Nevertheless, larger studies are required to validate its routine use in both clinical and forensic settings.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12245-025-01003-1.

Supplementary Material 1.

Acknowledgements

Data access statement

Research data supporting this publication were obtained from publicly available resources. Therefore, a dedicated repository was not applicable to this study.

Authors' contributions

DM - contributions to data collection, compilation, drafting the manuscript, and data analysisAPP - contributions to the conception, title, data analysis and interpretation of data; substantively revised the manuscript.All authors have approved the submitted version and APP as the corresponding author. APP has agreed to be accountable for the contributions, accuracy and integrity of the present work.

Funding

No funding available for this review.

Data availability

Research data supporting this publication were obtained from publicly available resources. Therefore, a dedicated repository was not applicable to this study.

Declarations

Ethis approval and consent to participate

This study does not breach data privacy and identification, and the risk level is less than minimal. Therefore, both institutional and ICMR guidelines state that no ethical approval is needed for conducting this review.

Competing interests

The authors declare no competing interests.

Received: 23 July 2025 / Accepted: 11 September 2025
Published online: 07 October 2025

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