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Original Article

Procalcitonin: the bacterial and non-bacterial sepsis marker

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Abstract

Introduction: The aim of this study was to evaluate the role of procalcitonin (PCT) in differentiating causative agents of bacterial sepsis, and sepsis from non-bacterial causes (viral, fungal, and parasitic).

Methodology: This study was conducted in the Department of Microbiology (April 2023 to March 2024) and included 1,346 clinically suspected cases of bacterial, fungal, viral, and parasitic infections confirmed through various diagnostic methods. Serum/plasma samples were collected from the participants and healthy controls, and PCT levels were measured. PCT concentration of < 2 ng/mL was classified as low, while levels ≥ 2 ng/mL were considered high, serving as threshold for sepsis diagnosis.

Results: PCT levels were significantly higher in sepsis caused by Gram-negative bacteria compared to Gram-positive bacteria, and showed a notable increase in *Plasmodium* infections ($p < 0.0001$). No significant association was observed between PCT levels and *Candida albicans* infections; however, cases involving non-albicans *Candida* species showed significantly elevated PCT levels ($p = 0.0265$). Infections with Cryptococcal species, hepatitis B, and hepatitis C showed a marked decrease in PCT levels. PCT levels were low in all cases involving skin commensals, with a more pronounced reduction in the case of coagulase-negative *Staphylococcus* compared to diphtheroids.

Conclusions: PCT levels showed a significant elevation in infections caused by non-bacterial agents, including *Plasmodium* and non-albicans *Candida*. A notable decline in PCT levels was observed in systemic infections caused by viruses and *Cryptococcus*. PCT is emerging as a universal biomarker for both bacterial and non-bacterial sepsis, making it a potential universal marker for sepsis.

Key words: procalcitonin; sepsis; biomarker.

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Introduction

Sepsis is one of the leading causes of mortality and morbidity in the world. It is a condition of systemic hyperinflammation initiated in response to an infection caused by microbes or their released endotoxins, which leads to multiple organ dysfunction syndrome and death [1]. Sepsis can be caused by various bacterial, fungal, viral, or parasitic pathogens [2]. Bacteria account for more than 70% of the documented sepsis [3]. Around 42% of sepsis cases are culture negative, suggesting a non-bacterial cause [4].

While bacterial sepsis rates have risen, it's essential to note a simultaneous increase in other causes of sepsis. Viral etiologies now represent approximately 1% of cases [5], while fungal origins have surged to 20% [6]. Parasitic infections are also at risk of developing and presenting as sepsis [7].

Therefore, diagnosing the specific causative agent of infection in sepsis is challenging, and laboratory confirmation methods for identifying the cause often have several limitations. Diagnosis of bacterial sepsis relies on blood culture, which usually takes up to 5 days to show positive, thus delaying treatment. Furthermore, blood specimens for bacterial/fungal cultures are usually sent only after initiating empirical antibiotics, resulting in culture-negative findings [8]. Contamination of the specimen while collection or transportation of culture often compromises the specificity of the obtained culture results, further complicating laboratory diagnosis. Hence, a diagnostic marker providing timely information about the probable cause of infection is necessary so that appropriate antibiotics can be administered on time.

Non-bacterial causes of sepsis, such as viral, fungal, and parasitic infections, are particularly challenging to diagnose using conventional methods like microscopy and culture. These methods are also labor and time-intensive, and they often suffer from low sensitivity and reproducibility [9]. Therefore, a comprehensive array of markers is necessary to diagnose the various causative agents of sepsis effectively. This underscores the need for a universal marker that is affordable, readily available, and capable of distinguishing between bacterial and non-bacterial causes, thereby aiding in accurate diagnosis and guiding appropriate treatment.

Biomarkers are crucial for the early diagnosis of sepsis as they can indicate its presence or absence, assess its severity, and differentiate between bacterial, viral, and fungal infections, as well as between systemic and local infections [10]. These biomarkers can be

classified into pathogen-specific and host-response biomarkers [11].

Widely used viral specific biomarkers are direct antigen tests like HBsAg and HBeAg [12] for hepatitis B and HCV antigen for hepatitis C [13]. Fungal sepsis markers are β-1-3-D-glucan (BDG) [11] for candidal infections, and the cryptococcal Ag test (CrAg) [11] for cryptococcal infections. In addition, parasite-specific markers like plasmodial lactate dehydrogenase and histidine-rich protein II (HRP II) [14] for malaria are commonly used for routine diagnosis.

Given the specificity of biomarkers for particular pathogens, there is a pressing need for a universal biomarker that can be used for the overall diagnosis of sepsis. Such a biomarker would need to be rapid, cost-effective, readily available, and capable of distinguishing between bacterial and non-bacterial causes of sepsis, thus guiding accurate diagnosis and treatment.

Traditional host-response biomarkers, like C-reactive protein (CRP) and procalcitonin (PCT), are used to distinguish bacterial from viral infections [15]. While significant research has focused on the roles of PCT and CRP in bacterial infections, their significance in non-bacterial infections remains less clear and warrants further investigation.

PCT is one of the most widely used and studied biomarkers for bacterial sepsis, but it also needs to be evaluated for non-bacterial sepsis.

To the best of our knowledge, this is a first of its kind study that evaluates all the microbial causative agents of sepsis—bacterial, viral, fungal, and parasitic. This study aimed to evaluate the role of PCT in differentiating various causative agents of sepsis, specifically in distinguishing bacterial sepsis from non-bacterial causes such as viral, fungal, and parasitic infections.

Methodology

This prospective study was conducted in the Department of Microbiology, utilizing routine laboratory data collected from April 2023 to March 2024. The study was time bound and utilized convenience sampling to enroll 1,346 clinically suspected cases of sepsis, including both bacterial and non-bacterial infections, and 50 healthy controls for baseline PCT reference.

Ethical approval was obtained from the institute's internal ethics committee (IEC 156/22) dated 18 October 2022, with a waiver for informed consent since there was no direct involvement of human subjects in this study.

Inclusion criteria

Patients with clinically suspected sepsis, confirmed to have bacterial, viral, fungal, or parasitic infections through relevant laboratory tests (e.g., blood cultures, polymerase chain reaction (PCR), or serology) were

Table 1. Distribution of all organisms that cause sepsis.

Groups		Name of causative organism	Total number of isolates (n = 1346)	Percentage (%)
Group 1 (34.39%)	**Gram-positive**	*Staphylococcus aureus*	56	4.1
		Enterococcus	48	3.56
	Gram-negative bacilli	*Escherichia coli*	66	5
		Enterobacter	12	1
		Klebsiella pneumoniae	77	6
		Pseudomonas aeruginosa	44	3.3
		Serratia	17	1.26
		Burkholderia	16	1.18
		Citrobacter species	9	0.66
		Stenotrophomonas maltophilia	5	0.37
		Salmonella	1	0.074
		Providencia	8	0.594
		Proteus	4	0.297
		Elizabethkingia species	2	0.148
	Gram-negative coccobacilli	*Acinetobacter*	*98*	*7.28*
Group 2 (6.83%)	**Fungi**	*Cryptococcus*	13	1
		Albicans candida	21	1.6
		Non albicans candida	58	4.30
Group 3 (2.30%)	**Malaria parasite**	*Plasmodium. vivax*	*31*	*2.30*
Group 4 (9.43%)	**Virus**	Hepatitis B	62	4.6
		Hepatitis C	65	4.8
Group 5 (47.02%)	**Skin commensals**	Micrococcus	46	3.41
		Diphtheriods	38	2.82
		CONS	549	41

CONS: coagulase-negative staphylococci.

included in the study. Only patients aged 18 years or older were included.

Exclusion criteria

Patients with concomitant infections that may affect PCT levels, those with non-infectious causes of sepsis such as autoimmune disorders or trauma, and pregnant women were excluded from the study.

Clinically suspected bacterial, fungal, viral, and parasitic infections were confirmed using various diagnostic methods, including automated blood cultures (BacT/ALERT 3D, Biomerieux Inc., Marcy l'Étoile, France) for bacterial and fungal growth, Cryptococcal Antigen Latex Agglutination test (CALAS, Pastorex Crypto Plus, Bio-Rad Laboratories, Gurugram, Haryana, India) for suspected cryptococcal meningitis, peripheral blood film examination, and rapid diagnostic tests for detecting *Plasmodium vivax*. Additionally, quantitative real-time polymerase chain reaction (RT-PCR) for the detection of hepatitis B and hepatitis C was performed using the Cobas TaqMan RT-PCR system (Roche Diagnostics, Basel, Switzerland). The cases were then divided into four groups based on the causative organisms, with a fifth group consisting of commensals typically found in routine skin flora (Table 1)

Serum/plasma samples of all the study subjects, including healthy controls, were collected, and their PCT concentrations were measured using the Vidas analyzer (enzyme-linked fluorescent assay (ELFA) technique; Biomerieux Inc., Marcy-l'Étoile, France).

PCT values of ≥ 2 ng/mL are likely to be suggestive of systemic infection based on the manufacturer's instructions in the kit literature [16]. This finding is also supported by other studies, such as Samsudin *et al*. [17], Sinha *et al.* [18], and Meisner [19], where it has been similarly reported that PCT concentration levels ≥ 2 ng/mL are more likely associated with systemic infection. Therefore, in this study, PCT concentrations < 2 ng/mL and ≥ 2 ng/mL were considered low and high, respectively, as a criterion for sepsis.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics version 25.0. Descriptive statistics were used to summarize the distribution of etiological agents. Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the diagnostic performance of PCT in differentiating between etiological categories, and based on the fixed PCT values as described above. The area under the curve (AUC) with 95% confidence intervals (CI), sensitivity, specificity, positive likelihood ratio (+LR), negative likelihood ratio (−LR), and diagnostic accuracy were calculated using a cutoff value of 2 ng/mL. The Chi-square test was applied to assess the association between different microbial pathogens and PCT levels, determining the significance of elevated PCT in various infectious groups. A p-value of < 0.05 was considered statistically significant.

Results

Out of the 1,346 clinically suspected cases of sepsis enrolled in the study, 463 cases showed growth of pathogenic bacteria. This constituted Group I, and the distribution was as follows: *Acinetobacter* was the most common (7.28% n = 98), followed by *Klebsiella pneumoniae* (6%, n = 77), *Escherichia coli* (5%, n = 66), *Staphylococcus aureus* (4.1%, n = 56), *Enterococcus* species (3.56%, n = 48), *Pseudomonas aeruginosa* (3.3%, n = 44), and others. Group II consisted of 92 cases of yeast and yeast-like fungi. This group included *Candida albicans* (1.6%, n = 21), non-albicans *Candida* (4.30%, n = 58), and *Cryptococcus* species (1%, n = 13). Group III included 31 clinically suspected cases of *Plasmodium* infections. Group IV comprised 127 confirmed cases of blood-borne viral hepatitis, hepatitis B (n = 62), and hepatitis C (n = 65), particularly those with high viral loads. Group V included 633 clinically suspected cases of sepsis where only skin commensal bacteria were isolated in automated blood cultures. Among these, coagulase-negative *Staphylococcus* species accounted for 41%, followed by *Micrococcus* species at 3.41%, and diphtheroid at 2.8%. (Table 1).

The diagnostic performance of PCT across different groups, comparing Group 1 (bacterial) individually with Groups 2 to 5 (fungal, parasitic, viral, and commensal, respectively), is summarized in Table 2 and Figure 1.

The PCT values of the different microbial species were calculated with their value of significance (Table

Table 2. Diagnostic performance of procalcitonin at a cut-off value of 2.0 ng/mL for differentiating between etiological groups in sepsis.

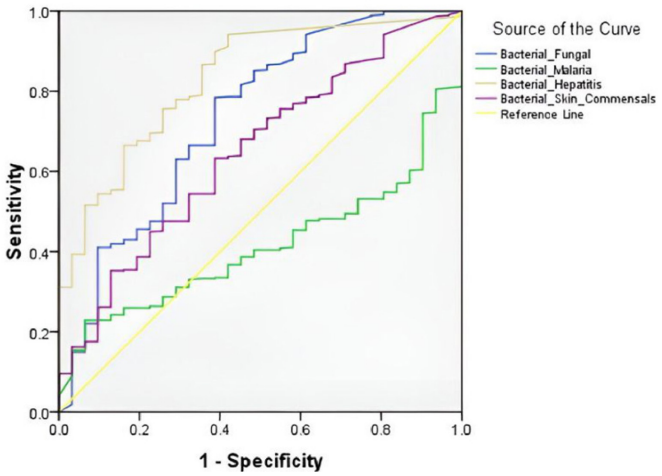
Comparison	Sensitivity (%)	Specificity (%)	AUC	95% CI	+LR	−LR	Diagnostic accuracy (%)
Group I vs II	61.1	50.0	0.609	0.545–0.672	1.22	0.78	55.6
Group I vs III	61.1	9.7	0.413	0.342–0.484	0.68	4.01	35.4
Group I vs IV	86.8	59.1	0.811	0.768–0.853	2.12	0.22	73.0
Group I vs V	87.9	31.6	0.664	0.632–0.696	1.29	0.38	59.8

AUC: area under the curve; 95% CI: 95% confidence interval; +LR: positive likelihood ratio; −LR: negative likelihood ratio.

3). In Group I, PCT values were significantly higher in the case of Gram-negative bacteria compared to Gram-positive bacteria, with a *p*-value of 0.004. In Group II, there was no significant correlation between PCT levels and *Candida albicans*. However, PCT levels were significantly elevated in cases involving non-albicans *Candida* species ($p = 0.0265$). Cryptococcal species exhibited minimal or no elevation in PCT levels, which was still statistically significant ($p = 0.0052$). In Group III, sepsis due to *Plasmodium vivax* infection showed markedly elevated PCT levels, with a highly significant *p* value of < 0.0001. In Group IV, cases of hepatitis B and C demonstrated a significant reduction in PCT levels ($p< 0.0001$). In Group V, PCT levels were low across all cases involving skin commensals, with a more pronounced reduction observed in coagulase-negative *Staphylococcus* compared to diphtheroid ($p < 0.0001$). A bar chart was constructed to compare the median PCT values across different organisms. *Enterobacter* species exhibited the highest median PCT value at 23.68, indicating a strong association with elevated PCT levels. This was followed by *Elizabethkingia s*pecies with a median value of 12.115. *K. pneumoniae* and non-albicans *Candida* showed median PCT values of 7.915 and 7.15, respectively. In contrast, coagulase-negative *Staphylococcus* (0.79), *Micrococcus* (0.93), and *Cryptococcus* (0.08) had the lowest median PCT values, indicating minimal PCT elevation (Figure 2).

Therefore, it was observed that PCT values were elevated in cases of bacterial sepsis, malarial sepsis, and sepsis caused by non-albicans *Candida* species, while they were decreased in viral and cryptococcal sepsis.

Figure 1. Receiver operating characteristic (ROC) curves of procalcitonin (PCT) at a cut-off value of 2.0 ng/mL for differentiating bacterial groups from fungal, malarial, viral, and commensal groups in patients with suspected sepsis.



Discussion

This study examined PCT as a biomarker for both bacterial and non-bacterial causes of sepsis to better understand its levels across various microbial etiologies, as most existing literature has primarily focused on the role of PCT in bacterial sepsis. In this study, we found that PCT levels were elevated in

Table 3. Procalcitonin (PCT) values of different microbial species with their values of significance.

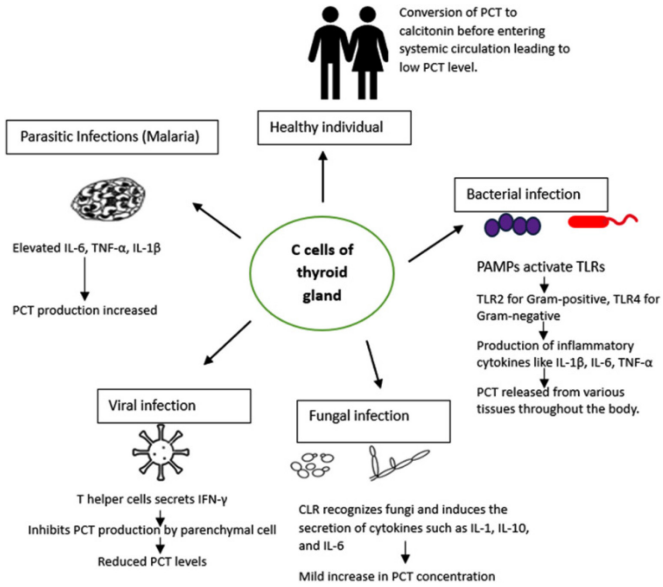
Groups	Pathogen	No of samples	PCT values (ng/mL)		*p* value Chi-square test
			< 2 ng/mL	> 2 ng/mL	
Group 1	Gram-positive	*Staphylococcus aureus*	28	28	0.351
		Enterococcus	22	26	0.2558
	Gram-negative bacilli	*Escherichia coli*	19	47	< 0.0001
		Enterobacter	3	9	0.0439
		Klebsiella pneumoniae	26	51	0.0003
		Pseudomonas aeruginosa	14	30	0.0029
		Serratia	10	7	0.6797
		Burkholderia	10	6	0.4857
		Citrobacter	6	3	0.4395
		Stenotrophomonas maltophilia	1	4	0.1281
		Salmonella	1	0	0.3545
		Providencia	4	4	0.8260
		Proteus	3	1	0.3957
		Elizabethkingia species	1	1	0.9127
	Gram-negative coccobacilli	*Acinetobacter*	36	62	0.0004
Group 2	Fungi	*Cryptococcus*	12	1	0.0052
		Albicans candida	11	10	0.5629
		Non albicans candida	23	35	0.0265
Group 3	Malaria parasite	*Plasmodium. vivax*	3	28	< 0.0001
Group 4	Virus	Hepatitis B	51	11	< 0.0001
		Hepatitis C	56	9	< 0.0001
Group 5	Skin commensals	Micrococcus	29	17	0.2038
		Diphtheroid	17	21	0.2523
		CONS	339	210	< 0.0001

CONS: coagulase-negative staphylococci.

bacterial sepsis, malarial sepsis, and sepsis caused by non-albicans *Candida* species.

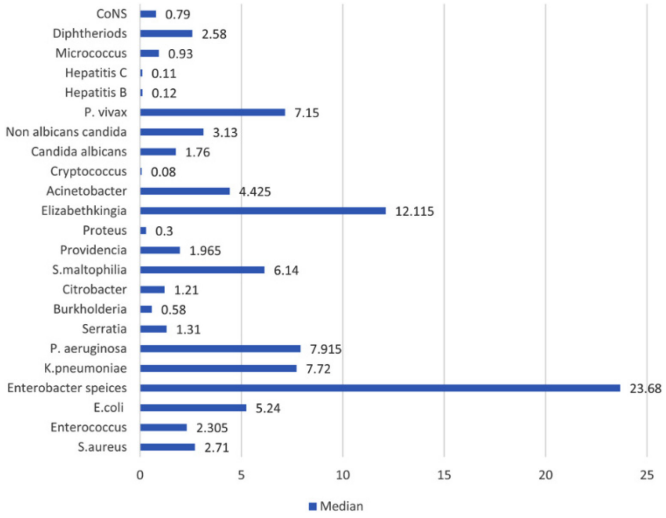
These findings are in line with existing evidence that PCT is a reliable biomarker for bacterial infections. PCT levels start to rise within 2–3 hours after a bacterial infection, showing a continuous dynamic change in serum with a plasma half-life of 24 hours [17]. Higher PCT levels correlate with the extent and severity of microbial invasion, indicating a poorer prognosis [20]. Although the exact mechanism behind the varying production of PCT in response to different bacterial pathogens is not fully understood, it may be due to the different ways Gram-positive and Gram-negative bacteria interact with host cells. Gram-positive bacteria, through lipoteichoic acids; and Gram-negative bacteria, through lipopolysaccharides (LPS); engage different pathogen-associated molecular patterns (PAMPs) [15]. These PAMPs activate different toll-like receptors (TLRs) in human cells: TLR2 for Gram-positive bacteria and TLR4 for Gram-negative bacteria [15]. This activation leads to the production of different inflammatory cytokines, such as interleukin-1β, interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α), which in turn stimulate the production and release of PCT from various tissues throughout the body [15] (Figure 3). This study aligns with a study conducted by Leli *et al.* [15] on bacteremia, which found that serum PCT levels were significantly higher in Gram-negative

Figure 3. Immunopathogenesis of procalcitonin (PCT) in healthy individuals and those with bacterial, viral, fungal, and parasitic infections.



IL-6: interleukin-6; TNF-α: tumor necrosis factor alpha; IL-1β: interleukin-1 beta; PAMPs: pathogen-associated molecular patterns; TLR: toll like receptor; IFN-γ: interferon gamma.

Figure 2. Bar chart comparing the median procalcitonin (PCT) values (ng/mL) across different organisms.



CoNS: coagulase-negative staphylococci; *P.* vivax: *Plasmodium vivax*; *S.* maltophilia: *Stenotrophomonas maltophilia*; *P.* aeruginosa: *Pseudomonas aeruginosa*; *K.* pneumonia: *Klebsiella pneumoniae*; *E.* coli: *Escherichia coli*; *S.* aureus: *Staphylococcus aureus*.

infections compared to Gram-positive infections. Another study conducted by Maigari *et al.* [21] had a similar finding that serum PCT concentration was significantly higher in bacterial sepsis. So PCT can guide in predicting the probability of bacterial infections and in determining the appropriate duration of antibiotic treatment.

To the best of our knowledge, there are only a few studies that have evaluated the role of PCT in cases of non-bacterial sepsis (Figure 3). Leli *et al.* studied the PCT level in Gram-positive, Gram-negative, and fungal bloodstream infections [15]. Another study conducted by Matur *et al.* evaluated the use of serum PCT levels in bacterial and viral infections [22]. Mahittikorn *et al.* described the importance of PCT in malarial infection and in severe malaria [7].

In this study, PCT did not show any significant difference in infections caused by *Candida albicans*, but was elevated in infections caused by non-albicans *Candida* species; and the findings were statistically significant ($p = 0.0265$). Cryptococcal infections also showed significantly low PCT levels. Several studies have shown that fungal infections result in low concentrations of PCT because the mechanism of PCT expression in fungal infections is different. Fungi are primarily recognized by C-type lectin receptors (CLRs) [23]. Activation of the CLR signaling pathway induces the secretion of cytokines such as interleukin-1 (IL-1), IL-10, and IL-6, resulting in a slight increase in PCT concentration following fungal infection [23]. A study

conducted by Cortegiani *et al.* found that a significantly lower value of PCT was observed in *Candida* species [9].

Malaria can mimic bacterial sepsis, with patients presenting with fever, chills, fatigue, and organ dysfunction. PCT levels are typically elevated in bacterial infections, and can also be elevated in malaria, though they tend to be lower compared to bacterial sepsis. A study by Mahittikorn *et al*. reported that patients with malaria exhibited increased PCT levels due to elevated proinflammatory cytokines such as IL-6, TNF-α, and IL-1β during infection [7]. Similarly, this study found that PCT levels were significantly elevated in cases positive for *Plasmodium vivax*. Thus, PCT can be used in conjunction with other diagnostic tools like rapid malaria tests, blood smears, and clinical assessments to rule out malarial causes, or if the clinicians suspect malarial sepsis.

In the present study, PCT showed a significant reduction in patients with hepatitis A and hepatitis B. The course of PCT production for viral infections differs when compared with bacterial infections. IFN-γ, secreted by T helper cells during a viral infection, inhibits PCT production by the parenchymal cells [22]. Therefore, decreasing PCT levels in viral infections can help to rule out viral sepsis and guide appropriate treatment decisions.

PCT levels were generally low across all skin commensals, with a more significant decrease observed in coagulase-negative *Staphylococcus* compared to diphtheroid. This is similar to a study conducted by Bassetti *et al*. [10]. Another study conducted by *Shomali et al*. showed that PCT may be useful in differentiating the pathogenic *S. aureus* from the skin commensal coagulase negative *Staphylococcus species* (CoNS) [24]. This information will prevent clinicians from overtreating such cases.

This study had several limitations. The small sample size for malaria and fungal infections prevented definitive conclusions about the value of PCT in these cases, and cryptococcal infections were diagnosed based only on CALAS. Hence, this reliance on rapid methods without a gold standard might have affected the PCT values. Detailed clinical history and follow-up of patients were not performed; thus, important factors that could potentially influence PCT levels, such as renal function, immunosuppressive status, and prior antimicrobial therapy, including its timing, were not recorded. In addition, the 633 clinically suspected cases of sepsis that showed growth of skin commensals on blood culture, were not followed up to determine the exact focus of their infection. The absence of this information may have impacted the interpretation of PCT levels. Furthermore, the study was conducted at a single center and followed a convenience-based sampling approach, which may restrict the generalizability of the findings to other regions and levels of healthcare.

Conclusions

The study concluded that PCT is an emerging universal biomarker for both bacterial and non-bacterial sepsis, showing significant elevation in infections caused by non-bacterial agents like *Plasmodium* and non-*albicans Candida*. Conversely, there was a statistically significant decline in PCT levels in systemic infections due to viral and cryptococcal causes. The presence of skin commensals during sample collection can be ruled out by identifying significantly low levels of PCT. This decrease in PCT levels can be attributed to specific underlying mechanisms related to these infections.

Although this is the first study of its kind to include a wide range of microorganisms, such as bacterial, viral, fungal, and parasitic, its findings require validation in a larger, multicenter study group to enhance reliability. Moreover, as the study was conducted at a single center using a convenience sampling approach, the findings may have limited generalizability to other regions and healthcare tiers. Such validation could establish PCT as a cost-effective, widely available commercial biomarker, potentially making it a universal marker for sepsis.

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Conflict of interest

No conflict of interest is declared.

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