Comparison of procalcitonin levels with blood culture results and foci of infection in septic patients

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ABSTRACT

Background and aim: Large number of studies proved undisputable role of procalcitonin (PCT) in sepsis diagnosis. Moreover, potential of procalcitonin to predict blood culture results according to Gram staining, different types of pathogens and foci of infection is discussed lately. The primary aim of our study was to compare the PCT levels in septic patients with documented Gram-positive and Gram-negative bacteraemia. We also evaluated the PCT levels according to different foci of infection and with different types of pathogens.

Material and Methods: Procalcitonin levels measured at the time of sepsis diagnosis (PCT1) and after 24 hours (PCT2) in well-defined cohort of septic patients were statistically evaluated according to the results of blood cultures and foci of infection.

Results: Out of 258 patients, 180 had negative and 78 positive blood culture. The difference in PCT1 and PCT2 levels between gram-negative (GN) and gram-positive (GP) bacteraemia was not significant. The highest values of PCT1 as well as PCT2 in culture-positive cases were found in patients infected with *Streptococcus spp.* followed by *Escherichia Coli* in contrast to *Staphylococcus spp.* with the lowest PCT concentrations. Highest procalcitonin levels were observed in urosepsis with PCT2 concentrations significantly higher than in all other foci of infection.

Conclusion: PCT discriminatory power to differentiate between GN and GP bacteraemia in septic patients appears to be low. PCT concentrations correlates probably more closely to different type of pathogens with highest PCT levels in *Streptococci spp.* and foci of infection rather than result of the Gram stain. In our study population, urosepsis showed statistically significant higher PCT concentrations 24 hours following sepsis diagnosis when compared to other site of infection.

KEYWORDS
procalcitonin – sepsis – septic shock – bacteraemia – foci of infection

SOUHRN

Nejtek T., Müller M., Moravec M., Průcha M. a Zazula R.: Porovnání hladin prokalcitoninu s výsledky hemokultur a ložisky infekce u kriticky nemocných pacientů se sepsí

Východiska a cíle: Nespočet studií prokázal neoddiskutovatelný přínos prokalcitoninu (PCT) v diagnostice sepse. V poslední době je navíc diskutován potenciální vztah koncentrací PCT k různým patogenům a ložiskům infekce. Hlavním cílem předkládané studie bylo porovnání hladin PCT u septických pacientů s dokumentovanou grampozitivní a gramnegativní bakteremií. Vyhodnotili jsme také koncentrace PCT ve vztahu k různým ložiskům infekce a jednotlivým patogenům.

Metody: V dobře definované kohortě septických pacientů byly vyhodnoceny koncentrace PCT v čase klinické diagnózy sepse (PCT1) a za 24 hodin (PCT2) a porovnány s výsledky hemokultur a ložisky infekce.

Výsledky: U 258 pacientů byla pozitivní hemokultura zdokumentována v 78 případech. Rozdíl v PCT1 i PCT2 mezi skupinami pacientů s dokumentovanou grampozitivní a gramnegativní hemokulturou nebyl statisticky významný. Ve skupině bakteremických pacientů byly nejvyšší koncentrace PCT1 i PCT2 zaznamenány u pacientů se sepsí způsobenou streptokoky a *Escherichia coli* v kontrastu s infekcemi způsobenými stafylokoky s nejnižšími zaznamenanými hladinami. Nejvyšší koncentrace PCT byly zaznamenány u pacientů s urosepsí se signifikantně vyššími hladinami PCT2 oproti všem ostatním sledovaným ložiskům sepse.

Závěry: Schopnost PCT rozlišovat mezi grampozitivní a gramnegativní bakteremií u kriticky nemocných pacientů se sepsí je nízká. Koncentrace PCT korelují pravděpodobně spíše s jednotlivými typy patogenů a ložisky infekce než s výsledky Gramova barvení. Nejvyšší hladiny PCT byly zaznamenány u streptokoků. Signifikantně vyšší koncentrace PCT oproti všem ostatním ložiskům infekce byly zaznamenány u urosepsí 24 hodin od stanovení klinické diagnózy sepse.

KLÍČOVÁ SLOVA
prokalcitonin – sepse – septický šok – bakteremie – ložiska infekce

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INTRODUCTION

Sepsis, redefined in the third international consensus (Sepsis-3) as life-threatening organ dysfunction by a dysregulated host response to infection, is associated with an in-hospital mortality greater than 10%. For clinical operationalization organ dysfunction can be presented by an increase in the sepsis-related organ failure assessment (SOFA) score of 2 points or more. Septic shock as a subset of sepsis, with mortality rates greater than 40%, is identified by a vasopressor requirement to maintain a mean arterial pressure (MAP) at least of 65 mm Hg and serum lactate level greater than 2 mmol/L in absence of hypovolemia. Both are one of the most common causes of death worldwide [1, 2].

Early management of sepsis including rapid identification of pathogen and administration of appropriate antimicrobial therapy has a crucial importance for clinicians to reduce mortality, improve outcome of patients and enhance cost-effectiveness of delivered care [3]. However, in clinical practice, an effort to identify the pathogen is often delayed due to the availability of suitable microbiological tests. To achieve greater precision in the assessment of true etiology of illness, using biomarkers with high sensitivity and specificity, can improve and speed up diagnostic workup [4].

No specific test can reflect the whole clinical picture of organ dysfunction during sepsis. However, the host response associated with infection can be easily quantified, and indeed, more than 170 biomarkers have been studied for potential use in septic patients. Some of them are known to play key roles in the immune response while others are mere innocent bystanders [5, 6].

One of the well-established biomarkers in sepsis diagnosing is procalcitonin (PCT). Procalcitonin, acute-phase protein, is a 116-amino acid prohormone of calcitonin with a molecular mass of 13 kDa, that is primarily expressed in the C-cells of thyroid gland. Very low levels of circulating PCT in healthy individuals can be increased during sepsis by its production in multiple tissues in response to inflammatory cytokines and bacterial endotoxins [7, 8]. Dozens of studies proved undisputable clinical impact of the role of procalcitonin in sepsis diagnosis, especially in early stage of severe bacterial infection with PCT level rapidly increased in the first 2–6 hours and reaching its peak within 6–24 hours after septic stimulus [9]. Considering these characteristics, PCT has been proposed as a part of the initial diagnostic approach [10] and for monitoring of antibiotic treatment response in critically ill patients [11].

With a growing need to personalize and precise the therapeutic approach in complicated and complex condition like sepsis, it is necessary to search for new diagnostic methods and to improve the existing [12], including improvement of current diagnostic and therapeutic impact of PCT testing in different clinical settings [13].

Several studies showed clinically relevant potential of PCT to discriminate between gram-negative (GN) bacteraemia, gram-positive (GP) bacteraemia and fungaemia in septic patients [14]. Moreover, the potential of PCT to discriminate specific types of pathogens detected in bloodstream is discussed lately and there is ongoing expert debate about the relationship between magnitude of PCT response and different sites of infection [15, 16].

Although association between PCT levels and specific type of pathogen as well as different foci of infection as mentioned above are intriguing, the discriminatory power of PCT with all known limitations (i.e., falsely high levels in the absence of bacterial infection) is too low to guide therapeutic decision on its own [17].

The primary aim of our study was to compare the PCT levels in septic patients with documented Gram-positive and Gram-negative bacteraemia. We also evaluated the PCT levels according to different types of pathogens and different foci of infection.

MATERIALS AND METHODS

Study Population

Analysis of the patient dataset from December 2012 to July 2020 obtained from the Department of Anesthesiology and Intensive Care, First Faculty of Medicine, Charles University and Thomayer University Hospital, Prague, was performed.

The following criteria were required for study inclusion:

(1) Fulfilled criteria according to the Sepsis-3 definition.

(2) Available results of blood cultures (BC) – at least 1 aerobic and 1 anaerobic bottle drawn at the time of sepsis diagnosis.

(3) PCT level measurements at the time of sepsis diagnosis (PCT 1) and after 24 hours if patient still alive (PCT 2).

(4) Change in clinical status less or equal to 3 h prior to admission to ICU or start of sepsis treatment.

A total number of 258 patients met the inclusion criteria. The process of patient selection is demonstrated in figure 1.

Data Collection and Laboratory Diagnostics

For each patient, the following data were collected: demographics; history and comorbidities; results of blood cultures; PCT1 and PCT2; initial SOFA score and lactate; C-reactive protein (CRP); record of previous antibiotic treatment or administration; 30-day mortality; intensive care unit (ICU) length of stay (LOS) and foci of sepsis.

Blood cultures were drawn at the time of sepsis diagnosis under aseptic conditions, always at least 1 aerobic and 1 anaerobic bottle (Bactec Plus–Becton Dickinson, Franklin Lakes, NJ, USA). Then, 10 mL of whole

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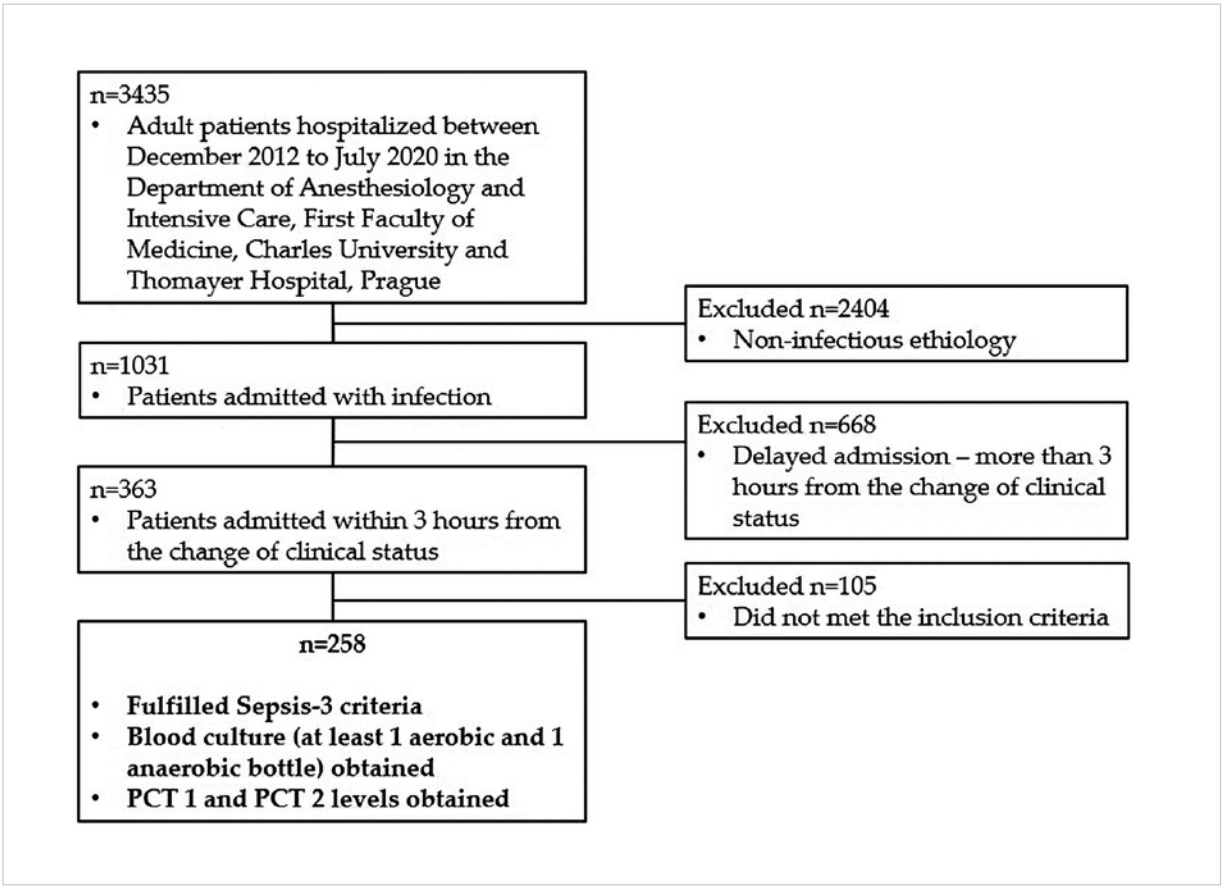


Figure 1. Process of patient selection and study inclusion criteria

blood was inoculated per bottle and processed in the department of clinical microbiology. Each bottle was incubated in the blood culture system (Becton Dickinson BACTEC FX40–Becton Dickinson, Franklin Lakes, NJ, USA) for five days. If positive, it was cultivated by standard microbiological methods.

Bacteraemia was defined as the presence of a causative pathogen found in blood culture(s), which were thoroughly evaluated by the physician in charge and experienced clinical microbiologist according to complementary investigations, presumed or confirmed focus of infection, other culture specimens (sputum, urine, etc.), and number of positive bottles (sets). All potential contaminants were ruled out and were not evaluated further, unless recognized by additional clinical and laboratory findings as a true pathogen.

Identification of the foci of infection was determined by a combination of imaging and laboratory findings and clinical judgement of the experienced interdisciplinary team. Groups were designated as follows: (1) Respiratory; (2) Abdominal; (3) Soft Tissues; (4) Urogenital; and (5) Catheter-Related Blood Stream Infection (CRBSI) and Infective Endocarditis (IE). If more potential sources of infection were probable, they were marked as group (6) Multiple. If no conclusive source of infection was found, foci remained (7) Unknown.

Lactate, partial pressure of oxygen in arterial blood, platelet count, bilirubin, creatinine, PCT, and CRP were investigated by standard methods available bedside or in the hospital laboratory.

Patients' histories and comorbidities were assigned to the Charlson comorbidity index (CCI) [18].

Statistical Analyses

Continuous data are presented as median (1st quartile–3rd quartile), and categorical data are presented as number (percentage), unless otherwise noted. For comparison of continuous data, the Wilcoxon/Kruskal-Wallis test was used. For pairwise comparisons after Kruskal-Wallis test, Wilcoxon rank sum test with continuity correction was used. For comparison of categorical data, the Chi-square test was performed. Differences in survival in bacteraemic and nonbacteraemic patients were analysed using the Kaplan-Meier method. A Cox proportional hazards model was used to perform a multifactorial analysis of the influence of selected factors on survival times and p-value(s) ≤ 0.05 was considered statistically significant. R 4.1.2 (The R Foundation, Vienna, Austria) with extension R-Studio 2023.03.0 + 386 (Posit Software, PBC, Boston, MA, USA) was used to perform statistical analysis.

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RESULTS

Out of 258 samples, 180 (69.8%) was blood culture negative (BC negative). Out of 78 (30.2%) bacteremic patients (BC positive), there was 34 (43.6%) with gram-positive flora (GP), 36 (46.2%) with gram-negative flora (GN) and 4 (5.1%) with mixed flora. In four (5,1%) positive BC fungi were found, and these were not further evaluated.

More frequent positive blood culture result was observed in group without previous antibiotic treatment ($p < 0,001$).

Other selected patient characteristics such as: age; sex; CCI; initial SOFA score; occurrence of septic shock; C-reactive protein; lactate; the record about previous antibiotics treatment or administration; 30-day mortality; ICU LOS and focus of infection are shown in table 1.

Difference in PCT1 and PCT2 levels in BC negative group compared to BC positive group was not statistically significant.

In BC positive samples levels of PCT1 in GN group were higher than concentrations of PCT1 in GP group, but the difference was not statistically significant. Similarly, PCT2 levels in GN group were also higher than concentrations of PCT2 in GP group, but the difference was not statistically significant neither.

Individual procalcitonin concentrations in the groups and subgroups according to the blood culture results are shown in table 2.

The representation of different microbial strains found in blood cultures are shown in table 3.

Among the mostly represented pathogens ($n \geq 5$) the highest PCT1 as well as PCT2 was observed in *Streptococcus spp.* sepsis. In *Streptococcus spp.* group the levels of PCT1 and PCT2 were significantly higher than in *Staphylococcus spp.* PCT2 levels in *Escherichia spp.* group were significantly higher than in *Staphylococcus spp.* group.

The difference in PCT levels among the most commonly represented ($n \geq 5$) pathogens is illustrated in figure 2.

Table 1. Patient characteristics

Characteristic	All patients (N = 254)	Blood culture results			
		BC negative (N = 180)	BC positive		
			All (N = 74)	GP (N = 34)	GN (N = 36)
Age (years)	66 (58–72.8)	66 (58.8–72)	66.5 (56–73)	64.5 (48.3–70)	68.5 (60.5–74)
Sex – male	167 (65.7)	119 (66.1)	48 (64.9)	22 (64.7)	22 (61.1)
Sex – female	87 (34.3)	61 (33.9)	26 (35.1)	12 (35.3)	14 (38.9)
CCI	4 (2–6)	4 (3–6)	5 (2–7)	3 (1.3–6)	5 (3–7)
Initial SOFA (points)	10 (7–13)	11 (8–12)	10 (6–13)	11 (7–14)	9 (6–13)
Septic shock	98 (38.6)	68 (37.8)	30 (40.5)	13 (38.2)	16 (44.4)
C-reactive protein (mg/L)	155.4 (83–269)	159.5 (82.2–251.8)	155.4 (103.7–274.3)	154.1 (113.2–302)	143 (93.1–250.9)
Lactate (mmol/L)	1.8 (1.1–2.9)	1.8 (1.2–2.8)	1.8 (1–3.2)	1.7 (1.2–3.1)	2 (1–3.1)
Previous antibiotics	150 (59.1)	119 (66.41)	31 (41.9)	15 (44.1)	16 (44.4)
30-day mortality	30.6 %	30.3 %	31.6 %	36.4 %	28.3 %
ICU LOS (days)	36 (32–49)	36 (30–49)	38 (35–67)	63 (27–125)	36 (22–NA)
Focus of infection					
Abdominal	66 (26)	51 (28.3)	15 (20.2)	3 (8.9)	10 (27.8)
CRBSI	14 (5.5)	1 (0.6)	13 (17.6)	8 (23.5)	5 (13.9)
Respiratory	105 (41.3)	90 (50)	15 (20.3)	6 (17.6)	9 (25)
Soft Tissues	20 (7.9)	9 (5)	11 (14.9)	9 (26.5)	1 (2.8)
Urogenital	20 (7.9)	10 (5.6)	10 (13.5)	3 (8.9)	7 (19.4)
Multiple	18 (7.1)	9 (5)	9 (12.2)	5 (14.7)	3 (8.3)
Unknown	11 (4.3)	10 (5.6)	1 (1.4)	0 (0)	1 (2.8)

Data are displayed as number (percentage) or median (Q1–Q3). ICU LOS is displayed as median (95% Confidence Interval).

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Table 2. Procalcitonin levels (μg/L) and its change in 24 hours (ΔPCT) in individual groups and subgroups according to the blood culture results

	BC negative	BC positive	p
PCT1	2.55 (0.75–9.78)	1.95 (0.71–15.58)	0.77
PCT2	2.78 (0.84–16.11)	4.60 (1.07–21.09)	0.25
ΔPCT	-0.06 (-0.95–1.19)	0.2 (-0.49–4.29)	0.053
	GP	**GN**	**p**
PCT1	1.92 (1.00–7.35)	2.81 (0.69–20.43)	0.19
PCT2	2.54 (0.80–14.45)	7.79 (1.71–38.97)	0.31
ΔPCT	0.07 (-0.46-4.35)	0.39 (-0.99-3.98)	0.57

Values are displayed as medians (Q1 – Q3).

Table 3. Causative pathogen strains found in positive blood cultures and corresponding PCT levels (μg/L)

Agents	N	%	PCT 1	PCT 2
Staphylococcus spp.	27	32.9	1.16 (0.57–2.62)	1.79 (0.58–4.92)
Klebsiella spp.	16	19.5	2.13 (0.65–14.07)	4.74 (1.32–15.34)
Escherichia spp.	10	12.2	15.81 (1.43–25.23)	24.08 (10.10–57.23)
Streptococcus spp.	7	8.5	63.05 (5.15–83.82)	99.04 (18.25–252.00)
Enterobacter spp.	6	7.3	1.45 (0.55–218.21)	3.92 (2.23–67.63)
Enterococcus spp.	5	6.1	2.15 (1.77–7.29)	4.01 (1.70–10.07)
Proteus spp.	2	2.4	7.41 (4.42–10.41)	68.51 (39.72–97.31)
Pseudomonas spp.	2	2.4	0.55 (0.50–0.61)	0.60 (0.59–0.60)
Serratia spp.	2	2.4	0.73 (0.66–0.80)	2.37 (1.38–3.35)
Acinetobacter sp.	1	1.2	13.22	10.80
Moraxella sp.	1	1.2	1.50	0.99
Morganella sp.	1	1.2	9.53	7.63
Sarcina sp.	1	1.2	2.03	3.10
Stenotrophomonas sp.	1	1.2	2.37	2.91

Data are displayed as number and percentage or median (Q1 – Q3).

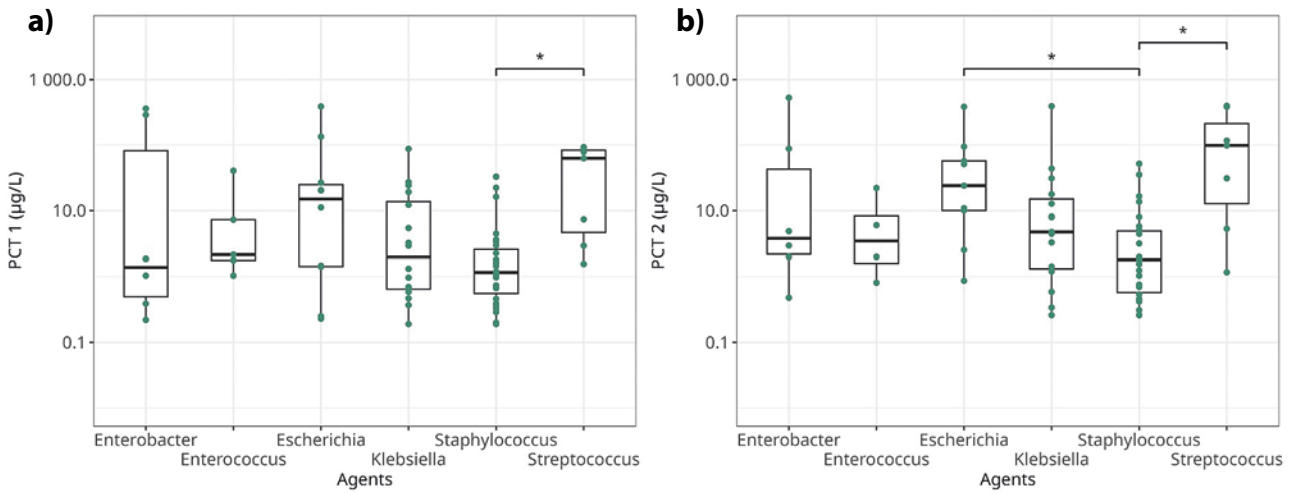


Figure 2. PCT1 (a) and PCT2 (b) concentrations (μg/L) in mostly represented (n ≥ 5) pathogens
Y axis is in logarithmic scale. *P* value(s) are demonstrated with horizontal line(s), if significant.

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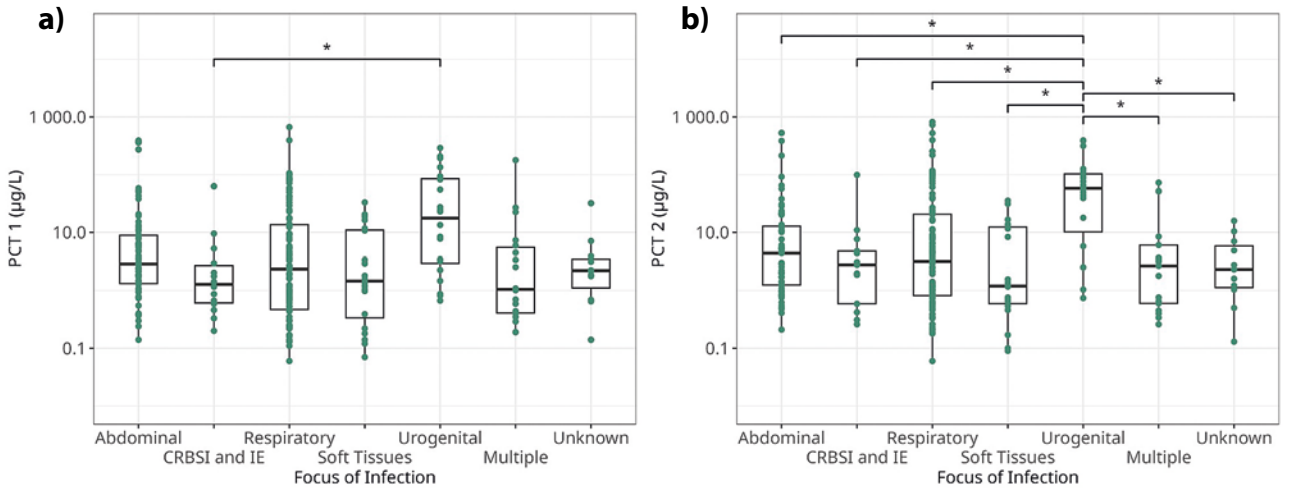


Figure 3. PCT1 (a) and PCT2 (b) concentrations (µg/L) according to foci of infection
Y axis is in logarithmic scale. *P* value(s) are demonstrated by horizontal line(s), if significant.

The highest PCT1 concentrations were recorded in group Urogenital 18.17 (3.00–85.26) µg/L followed by: Abdominal 2.90 (1.32–8.88) µg/L, Respiratory 2.35 (0.47–13.61) µg/L, Unknown 2.21 (1.23–3.52) µg/L, Soft tissues 1.47 (0.34–10.97) µg/L, CRBSI and IE 1.29 (0.61–2.74) µg/L and Multiple 1.05 (0.41–5.56) µg/L.

Similarly, PCT2 concentrations were highest in group Urogenital 57.99 (11.82–103.13) µg/L compared to other foci of infection: Abdominal 4.36 (1.25–12.81) µg/L, Respiratory 3.22 (0.82–20.56) µg/L, CRBSI and IE 2.79 (0.59–4.74) µg/L, Multiple 2.67 (0.60–6.03) µg/L, Unknown 2.32 (1.13–5.94) µg/L and Soft tissues 1.19 (0.60–12.34) µg/L.

The difference in PCT1 and PCT2 levels in individual groups according to foci of infection are shown in figure 3.

Difference in PCT1 levels in group without previous antibiotics compared to PCT1 in group with previous antibiotics was not statistically significant. PCT2 concentrations were significantly higher ($p < 0.0001$) in group without previous antibiotics compared to group with previous antibiotics.

Difference in PCT1 and PCT2 levels according to previous antibiotics are shown in figure 4.

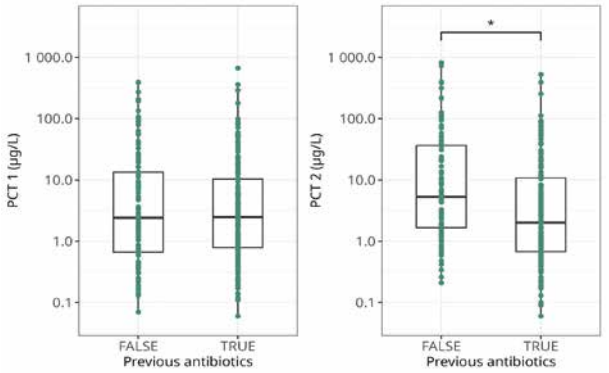


Figure 4. PCT1 and PCT2 levels (µg/L) according to previous antibiotics
Y axis is in logarithmic scale. *P* value is demonstrated by horizontal line, if significant.

DISCUSSION

The main finding of our study is, that although the levels of PCT1 and PCT2 in septic patients with GN bacteraemia were higher than PCT1 and PCT2 in GP bacteraemia group, this difference was not statistically significant. Similarly, some studies reported that PCT levels do not vary in GN or GP sepsis, nevertheless majority of studies have suggested that PCT levels are significantly increased in septic patients with documented GN bacteraemia [19].

The fundamental question whether PCT can distinguish between GP and GN in such heterogenous group as sepsis is difficult to answer. Many factors can influence PCT levels itself. Meisner et al. (1999) observed correlation between PCT concentrations and increasing SOFA levels in the critically ill patients with systemic inflammation [20]. However, initial SOFA did not statistically differ between septic patients with GN and GP bacteraemia. Antibiotics are cornerstone of bacterial sepsis treatment, and PCT level and its dynamic can be affected by antimicrobial treatment [21]. Nevertheless, the difference in PCT levels between GN and GP was not statistically significant neither in subgroup with nor without previous antibiotics. Similarly, the frequency of GP and GN bacteraemia with previous antibiotic treatment did not statistically differ from frequency in group GP and GN bacteraemia without previous antibiotics. Besides severity of illness and antibiotic treatment, several other factors such as patient characteristics,

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non-infectious systemic inflammatory response after major surgery, severe trauma, burns, inhalation trauma, pancreatitis, circulatory shock, and some type of cancer were found to be associated with high PCT levels, in contrast to localized infections causing sepsis (e.g.: mediastinitis, empyema, abscess and atypical pneumonia) with lower levels of PCT [22]. Although our study provides comprehensive data in well-defined cohort of septic patients, we do admit that unrecognised variables could have affected PCT levels and thus confound the overall results as stated in limitations. Contaminants, mostly presented by Coagulase-negative Staphylococci (CoNS), could be another possible explanation for failure of PCT levels to discriminate between GN and GP. Very wide range (up to 17%) of blood-culture contaminants (BCC) is reported among different institutions, including teaching hospitals [23]. Transient bacteraemia during routine care, instrumentation, diagnostic and therapeutic minor interventions, lack of ongoing training or staff work overload are the potential culprits [24]. However, this phenomenon was excluded by strict ruling out of contaminants applied for blood culture evaluation (as described in methods). Nevertheless, besides the appropriate algorithm in detecting BCC, other tools as time to cultivation, phenotyping, genotyping and use of new microbiological technologies as well as blood culture draw training is encouraged and strongly advised in an effort to reduce BCC in the future [25].

Comparable study (n = 166) of septic patients presented by Brodská et al. (2012) showed similar levels of PCT (median: 8.90; IQR: 1.88–32.60) µg/L in GN bacteraemia to our study cohort [26]. But the PCT concentrations in GP are remarkably lower (median: 0.58; IQR: 0.35–0.73) µg/L than PCT1 and PCT2 in the same group of our study population. Another study presented by Bilgili et. al (2018) with proportional study cohort of bacteraemic septic patients (n = 136) also reported similar median PCT values in patients with GN bacteraemia in contrast to much lower values in GP [27] than in our study cohort. So, it may be not appropriate to use PCT to differentiate between GP and GN sepsis with documented bacteraemia [13].

It appears that more than the results of Gram stain, specific pathogens are responsible for differences in PCT levels and the relationship between PCT and sepsis caused by various pathogens is discussed lately [17, 19, 28]. Some progress in knowledge of the specific mechanisms of PCT variations triggered by specific agents was achieved, however basic research is still lacking [13]. In our cohort, highest values of PCT1 and PCT2 were observed in Streptococci followed by *Escherichia coli* than in Staphylococci group where the PCT values were the lowest. These findings are consistent with observation of Rüddel et al. (2018) in well-designed prospective study cohort of septic patients, where highest concentrations were found also in *E. coli*, *Streptococcus spp.* and other Enterobacteriaceae [15]. The excessive concentrations in group of Streptococci are potential point of interest for further research. Evidence supporting claims about higher levels in α-haemolytic also ß-haemolytic Streptococci can be found in the literature. Adámková et al. in contrast to previously published data demonstrated higher levels of PCT in sepsis caused by *Streptococcus pyogenes*, when compared to other studied pathogens [29]. Similarly, Wang et al. presented interesting results of potential influence of microRNA (miR-497-3b) on PCT expression in pneumonia caused by *Streptococcus pneumoniae* [30]. In our study cohort the difference in PCT1 and PCT2 between α-haemolytic and ß-haemolytic Streptococci was not statistically significant. Unfortunately, we were not able to gather representative sample for each studied pathogen including Streptococci to evaluate possible consistent differences between all types of pathogens.

Another ongoing debate is drawing attention to PCT levels in different sites of infection [31]. In contrast to findings of recently published study by Kara et. al (2020) with highest PCT values detected in abdominal infections [16], our study, despite the lack of multivariant analysis, is one of the few with well-defined cohort of septic patients, which can support thesis about higher PCT values in urosepsis as suggested by Rüddel et al. (2018) [15]. Apart from possible influence of microbial load in different sites of infection, the spectrum of causative pathogens itself (Enterobacteriaceae) may be the main factor for magnitude of PCT level in urinary tract infections [32, 33]. And thus, more refine approach to improve diagnostic efficacy of PCT in different clinical settings for well defined subgroups of patients and for individual causative pathogens deserves further attention and confirmation in well designated controlled studies.

Our study has several limitations. First, the study was conducted retrospectively. Second, this study is single-center and the conclusion can be applied only for a survey population. Third, the insufficient information about patient baseline characteristics, comorbidities, and other factors, which could have influenced PCT levels. Fourth, in patients, admitted from other wards, sepsis diagnosis and enrolment into the study could have been delayed and confounded with nosocomial infections and previous antibiotics and thus affect the overall results. Fifth, relatively low number of positive blood cultures, especially in the group with previous antibiotics. Finally, the insufficient number of patients for each studied pathogen to confirm discriminatory power of PCT levels for individual bacteria.

CONCLUSION

PCT discriminatory power to differentiate between gram-negative and gram-positive bacteraemia in septic patients appears to be low. PCT concentrations

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correlates probably more closely to different pathogens and foci of infection rather than result of the Gram stain.

In our study population the highest values of PCT1 as well as PCT2 in culture-positive cases were found in patients infected with *Streptococcus spp.* followed by *Escherichia coli* in contrast to *Staphylococcus spp.* showing lowest PCT concentrations.

Urosepsis showed statistically significant higher PCT concentrations 24 hours following sepsis diagnosis when compared to other site of infection.

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