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Original Research Article

Correlative Study of C - reactive protein with Serum Procalcitonin in Organ Sepsis in Telangana Population

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Conflict of interest: Nil

Abstract:

Background: Sepsis is a life-threatening cause of death if not diagnosed properly and in time for medical treatment. The prognosis of sepsis is closely related to procalcitonin (PCT) and C-reactive protein levels.

Method: 60 patients aged between 50-68 years were classified into 4 groups as per the severity of organ dysfunction in sepsis according to sequential organ failure assessment (SOFA). Blood investigations included WBC count, platelet count, S. bilurubin, S. creatinine, arterial blood gas analysis, S. procalcitonin (PCT), and S. CRP, and results were studied with statistical analysis.

Results: SOFA score-I 0-6 had 27 (45%), score-II 9512 had 25 (41.6%), score-III 13-18 had 7 (11.6%), score-IV 19-24 had 1 (1.6%), and deaths were 13 (21.6%). Co-morbidities were 25 (41.6%) had type-II DM, 14 (23.3%) had HTN, 26 (43.3%) had pneumonia, and 17 (28.5%) had UTI. The causes of death were: 5 (18.4%) had pneumonia, 4 (30.7%) had UTI, 1 (7.69%) had pneumonia + UTI, 1 (7.69%) had SBP, 1 (7.69%) had diarrhoea, and 1 (7.69%) had DSS. In the comparison of SOFA with the variable SOFA scores studied except S. creatinine, all the parameters like GCS, total bilurubin platelet count, and SOFA score SPCT and SCRP had highly significant p values ($p<0.001$).

Conclusion: In the present pragmatic study, it is concluded that serum prolactin and CRP value levels are significantly correlated, and the elevation of these parameters predicts the prognosis of the severity of sepsis.

Keywords: SODA sequential organ failure, GCS (Glasgow coma scale), SPST (serum procalcitonin), sepsis Telangana.

Introduction

Sepsis refers to life-threatening dysfunction of the organ due to non-coronary ICU patients. About 18 million new cases of sepsis are reported each year, with a mortality rate of 30–50% [1].

The clinical symptoms of sepsis include tachycardia, tachypnea, and fever. Severe sepsis is associated with hypoperfusion organ dysfunction or hypotension [2]. In sepsis, invasion of the microorganisms into the blood stream occurs so that they localize, proliferate, and release their pathogenic factors into the blood stream. Currently, the diagnosis of such diseases is based on biochemical indexes or pathogen detection through bacterial culture.

Relevant biochemical tests lack high specificity, which leads to increased uncertainty in the diagnosis process and is challenging for clinicians. Rapid and accurate disease diagnosis, as well as timely medical intervention, can help the clinician to confirm the disease in an appropriate time frame and make the necessary treatment decisions. According to the specificity of the biochemical indicators that determine the severity of infections, Timely control of infection can be achieved through effective antibiotics or surgery to reverse the progress of the disease.

Procalcitonin (PCT), a pro-hormone of the calcitonin-I (CALC-1) gene on chromosome II, comprises 114–116 amino acids. C-reactive protein is an acute-phase reactive protein that can interact with the capsule C polysaccharide of Streptococcus pneumonia. Among the clinically useful biochemical detection indexes, PCT has shown superiority as an important reference marker for the infection, as well as antibiotic guidance [3]. It is also noted that changes in PCT and CRP concentrations are related to the prognosis of patients with sepsis [4]. Hence, an attempt was made to correlate PCT and CRP values in organ sepsis with various sequential organ failure assessments of various diseases.

Material and Methods

60 (sixty) patients aged between 50-68 years admitted to the intensive care unit of the Government Medical College, Bhadradri Kothaguedem, Telangana (507118) were studied.

Inclusive Criteria

The patients are over 50 years old and clinically diagnosed with sepsis or septic shock, and they gave their consent in writing that they were ready to undergo treatment.

Exclusion Criteria: Patients have cerebrovascular and cardiovascular disease. The patients had bone marrow irradiation, chemotherapy, or radiation therapy within six months. HIV-infected patients or those with viral hepatitis were excluded from the study.

Method: Every admitted patient's history was taken, and the investigation included WBC count, platelet count, serum bilurubin, serum creatinine, arterial blood gas analysis, serum procalcitonin (PCT), and serum C - reactive protein (CRP). The patients selected for the study were categorized into 4 groups according to the severity of organ dysfunction in sepsis according to squaretail organ failure assessment (SOFA) score: group I = 0-6, group II = 7–12, group III = 13–18, and group IV = 19–24.

The duration of the study was from June 2021 to May 2023.

Statistical analysis: Patients were classified as per the SOFA score with percentage. The cause of death was also classified with percentage. Comparison of SOFA groups with variable SOFA score were studied with AVOVA test. The statistical analysis was carried out in SPSS software. The ratio of male and female was 2:1.

Obstruction and Results

Table 1: Destitution of SOFA score in Sepsis patients -Total score of day 1: 0-6 score sofa group-I 27 (45%), II: 9.12 score sofa group-II 25 (41.6%), III: 13-18 score sofa group-III 7 (11.6%), IV: 19-24 score group-IV 1 (1.6%)

Table 2: Co-morbidities in sepsis patients: 25 (41.6%) had type-II DM, 14 (23.3%) had HTN, 26 (43.3%) had pneumonia, and 17 (28.5%) had UTI.

Table 3: Cause of death of the patient 5 (38.4%) had pneumonia, 4 (30.7%) had UTI, 1 (7.69%) had pneumonia + UTI, 1 (7.67%) had diarrhoea, 1 (7.69%) had SBP, and 1 (7.69%) had DSS.

Table 4: The GCS score had a ratio of 26.54 and a $p<0.001$ difference.

- Creatinine (mg/dl) had a ratio of 1.716 and $p<0.017$ (the p value was insignificant).
- Total bilirubin of different scores had a 170.21 F ratio and $p<0.001$.
- Platelet count for different scores had an F ratio of 1964.07 and $p<0.001$.
- The SOFA score of different groups had an F ratio of 175.39 and $p<0.001$.
- PCT (mg/dl) of different score F ratios (17.2 and $p<0.001$)
- CRP (mg/dl) of different scores had a 7.085 F ratio and $p<0.001$.

Table 1: Destitution of SOFA score in Sepsis patients (Total No of Patient: 60)

Total SOFA score Day-1	SOFA group	Number of patient 60 (sixty)	Percentage
0 – 6	1	27	45
9 – 12	2	25	41.6
13 – 18	3	7	11.6
19 – 24	4	1	1.6

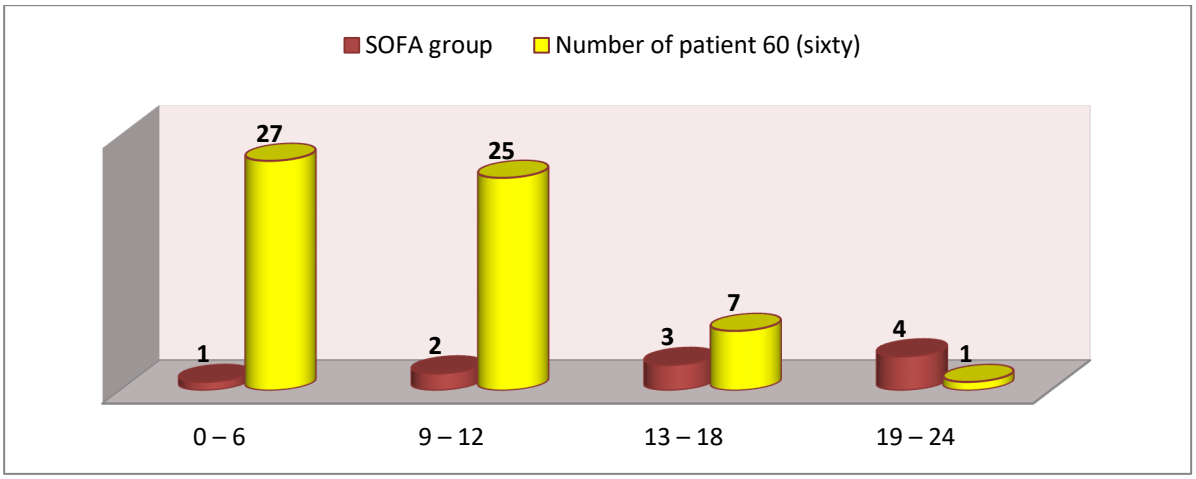

Figure 1:

Table 2: Co-morbidity factors in sepsis patients

Co-morbidities	No. of patients	Percentage (%)
Type-II DM	25	41.6
HTN (Hyper tension)	14	23.3
Pneumonia	26	43.3
UTI (urinary tract infection)	17	28.5%

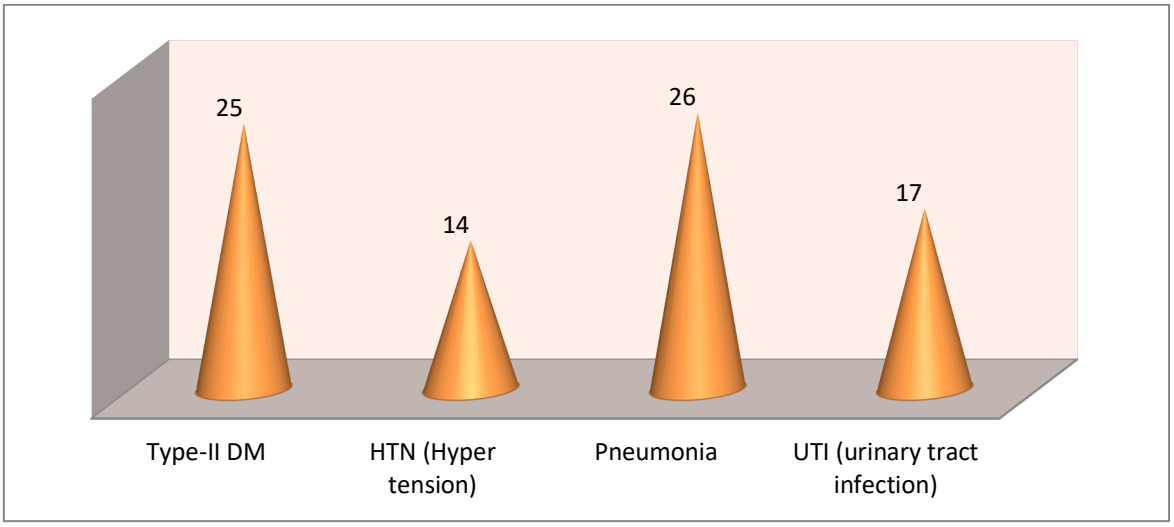

Figure 2: Co-morbidity factors in sepsis patients

Table 3: Cause of death of patients

Sl. No	Cause of death	Number (13)	Percentage (%)
1	Pneumonia	5	38.4
2	UTI	4	30.7
3	Pneumonia + UTI	1	7.69
4	Diarrhoea	1	7.69
5	SBP spontaneous Bacterial peritonitis	1	7.69
6	Dengue shock syndrome DSS	1	7.69

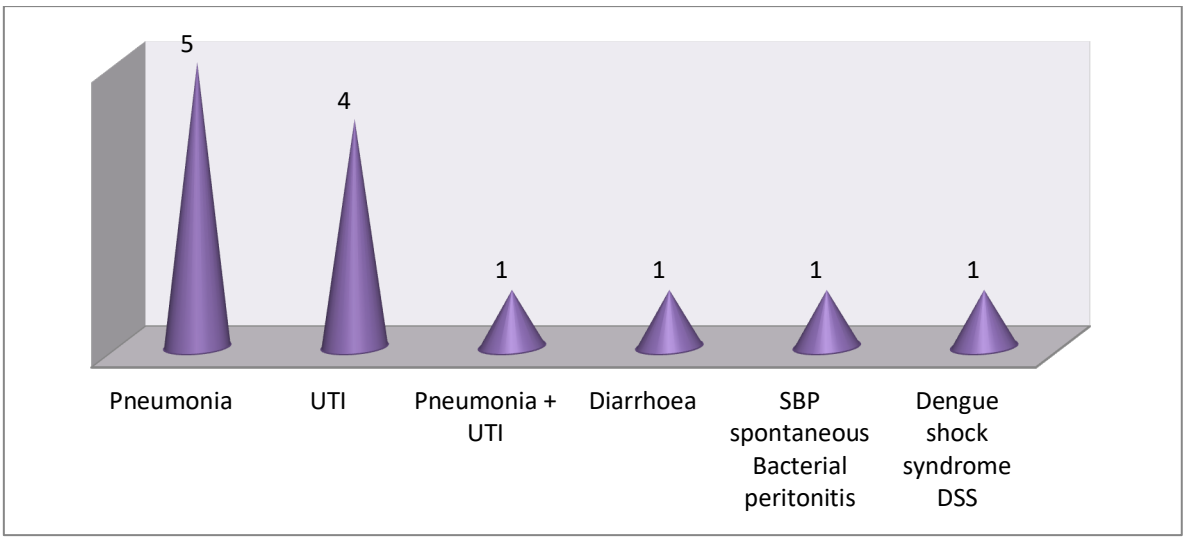

Figure 3: Cause of death of patients

Table 4: Comparison of sequential organ failure assessment (SOFA) scores with variables of SOFA score ANOVA TEST

Variables	SOFA group score-I (0-6)	SOFA group score-II (7-12)	SOFA group score-III (13-18)	SOFA group score-IV (19-24)	F ratio	P value
GCS	14.84 (±0.22)	14.32 (±1.80)	10.30 (±4.10)	7	26.54	P<0.001
Creatinine (mg/dl)	1.87 (± 0.62)	2.56 (± 1.23)	2.94 (± 0.92)	2.30	1.716	p>0.017
Total Bilurubin	1.43 (± 0.52)	4.33 (± 0.17)	2.42 (± 0.62)	2.08	170.21	P<0.001
Platelet count (per mm3)	241273.14 (±1152.62)	114240.04 (±56.21)	83140.20 (±35.65)	45.997	1964.0	P<0.001
SOFA score	3.46 (±1.70)	8.73 (±1.48)	13.80 (±1.03)	17.00	175.39	P<0.001
SPCT (ng/ml)	7.81 (±7.89)	28.12 (±28.02)	42.11 (±35.00)	86.91	17.21	P<0.001
S. CRP (mg/dl)	160.56 (± 50.02)	154.18 (±62.06)	145.10 (±55.20)	40.82	7.085	P<0.001

GCS= Glasgow coma scale, SPCT = Serum procaloitonin, SCRP = S. C-reactive protein

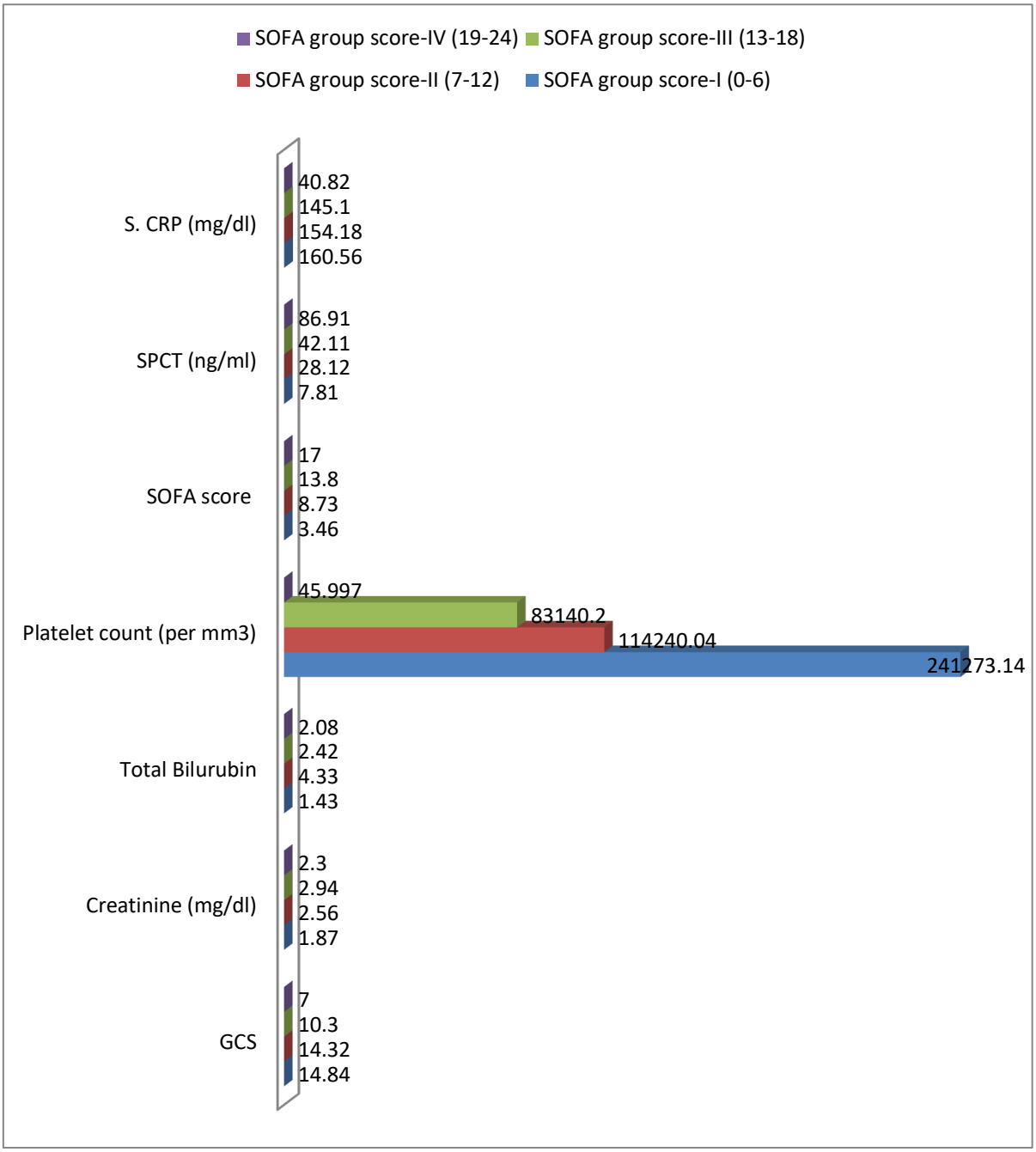

Figure 4:

Discussion

Present study of C-reactive protein with serum prolactin (SPCT) in organ sepsis in Telangana population SOFA group-I had 27 (45%) patients with a score of 0-6, group-II had 25 (41.6%) patients with a score of 9-12, group-III had 7 (11.6%) patients with a score of 13-18, and group-IV had 1 (1.6%) patient with a score of 19-24 (Table 1). The co-morbidity factors were: 25 (41.6%) had type-II DM, 14 (23.3%) had HTN, 26 (43.3%) had pneumonia and 111 (28.5%) had UTI (Table 2). In causes of death, 5 (38.4%) had pneumonia, 4 (30.7%) had UTI, 1 (7.69%) had pneumonia + UTI, 1 (7.69%) had diarrhoea, 1 (7.69%) had SAP, and 1 (7.69%) had DSS Table 3). In the comparison of SOFA scores with variables of SOFA scores except serum creatinine, all parameters were highly significant ($p<0.001$). The parameters were CGS, total bilirubin platelet count, SOFA score, serum procalcitonin (SPCT), and serum CRO (mg/dl) (Table 4). These findings are more or less in agreement with previous studies [5,6,7].

In healthy individuals, PCT is produced in thyroid **C** cells from the CALC-1 gene located on chromosome 11. The mRNA product is known as precalcitonin. It is further modified into the 116 amino acid procalcitonin. Finally, it is divided into three distinct molecules: active calcintonin (32 amino acids), ketocalcitonin (21 amino acids), and N-terminal procalcitonin (57 amino acids). The calcitonin hormone is involved in the homeostasis of calcium and phosphorous [8]. Practically all the PCT formed in the thyroid C cells is converted into calcitonin, so no PCT is released into the circulation. Hence, the PCT level in healthy subjects is very low (0.05 ng/dl), but the inflammatory release of PCT is independent of the above regulations. During inflammation, PCT is produced mainly by two alternative mechanisms: a direct pathway induced by liposacchride (LPS) or other toxic metabolites from microbes and an indirect pathway induced by various inflammatory mediators like IL-6, TNF-a, etc.

During sepsis, ubiquitous and uniform expressions of calcitonin (CT) mRNA in tissues were observed in hamsters (laboratory animals). It is reported that PCT is helpful for early detection of sepsis as well as monitoring the antimicrobial treatment regimen. In fact, PCT can be a useful tool for antimicrobial stewardship and its utilization. Exalted production of PCT during bacterial infection and its association with sepsis were reported, but the actual mechanism of production of PCT during infection is not known. It is assumed that bacterial lipopolysaccharides and sepsis-released cytokines modulate the liver and peripheral blood mononuclear cells to produce PCT [9]. Hence, in the early diagnosis of bacterially infected sepsis, PCT can be used for early detection of sepsis and prediction of outcome after major trauma, as well as in septic shock in patients with acute polynephritis secondary to ureter calculi [10].

CRP is useful in the detection of sepsis, and it is more sensitive than currently used markers such as BT and WBC count [11]. However, in the clinical context, it is often difficult to define CRP or PCT as an independent variable for the diagnosis of infection due to multiple causes of induction and co-morbidity of infection, systemic inflammation, organ dysfunction, endotoxinaemia, bacterial translocation, and tissue trauma in many critically ill patients.

Summary and Conclusion

The present correlative study examines C-reaction protein and PCT in organ sepsis in the Telangana population. PCT is a unique biomarker with a wide range of applications in the medical field compared to other conventional markers for sepsis. CRP is also closely related to PCT; hence, it will be a confirmatory biomarker in sepsis. It will be helpful to know the severity of sepsis, and these two biomarkers can predict the outcome or prognosis of sepsis. But the present study demands a patho-physiological, hormonal, nutritional, and genetic study because the actual mechanism of PCT production during infection is still unclear.

Limitation of study: Owing to the tertiary location of the research centre, the small number of patients, and the lack of the latest technique, we have limited results.

The present study was approved by the Ethical Committee of Government Medical College, Bhadradri Kothaguedem, Telangana, 507118.

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