WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 14, Issue 3, 1077-1085. Review Article ISSN 2277–7105

EXPLORING THE ROLE OF PCT (PROCALCITONIN) IN BURN INJURIES – A REVIEW

Nidhi Sharma* and Anand Yadav

Student of M. Pharm (Pharmacology), GD Goenka University, Gurugram Road, Sohna Haryana, India.

Article Received on
19 December 2024,

Revised on 09 Jan. 2025,
Accepted on 29 Jan. 2025

DOI: 10.20959/wjpr20253-35439

***Corresponding Author**
Nidhi Sharma
Student of M. Pharm (Pharmacology), GD Goenka University, Gurugram Road, Sohna Haryana, India.

ABSTRACT

Damage to the skin from burns, especially in severe cases that involve more than 20% of the body's surface area, might hinder the skin's ability to act as a barrier and increase the likelihood of infection. This review looks at the effectiveness of using blood procalcitonin (PCT), a biomarker for systemic inflammation, to diagnose burn sepsis in adults as soon as it happens. There is an immediate need for dependable screening as existing diagnostic approaches have drawbacks such as sluggish blood culture and limited specificity. In the setting of severe burn injuries, our study seeks to help clinical decision-making and improve patient outcomes by examining the potential of PCT to give timely insights into bacterial infections.

KEYWORDS: Likelihood, inflammation, procalcitonin.

INTRODUCTION

Burns are prevalent injuries characterized by physical or chemical damage to the skin, resulting in diminished or complete loss of the skin's barrier function.[1] One of the most crucial physical barriers in protecting the body from outside invaders is the skin. There is an increased danger of infection after a severe burn because the skin's protective function is impaired; the bigger the burn area, the more severe the burn. Patients with burns affecting more than 20% of their body surface area have a 3-to-30% higher risk of developing sepsis.[2] In burn patients, a dysregulated response to infection can lead to subsequent systemic organ failure, a condition known as burn sepsis.[3] One of the main causes of death in patients with severe burns, burn sepsis has an ominous start, rapid development, and bad prognosis.[4] Thus, to decrease patient mortality and enhance patient

prognosis, it is essential to identify high-risk individuals who are susceptible to burn sepsis early on.

The most reliable way to diagnose burn sepsis in a clinical setting is using a blood culture. However, burn patients' prophylactic antibiotic usage, which can lead to antibiotic resistance, and blood cultures' long duration and low positivity all work against their accuracy.[5] A lack of specificity and sensitivity in the diagnosis of burn sepsis is caused by several variables that significantly impact other blood tests, including neutrophils, leukocytes, and liver and kidney function.[6] To help determine if burn patients have sepsis, which can improve prognosis, decrease mortality, and decrease morbidity, a blood test with high sensitivity and specificity is urgently needed.

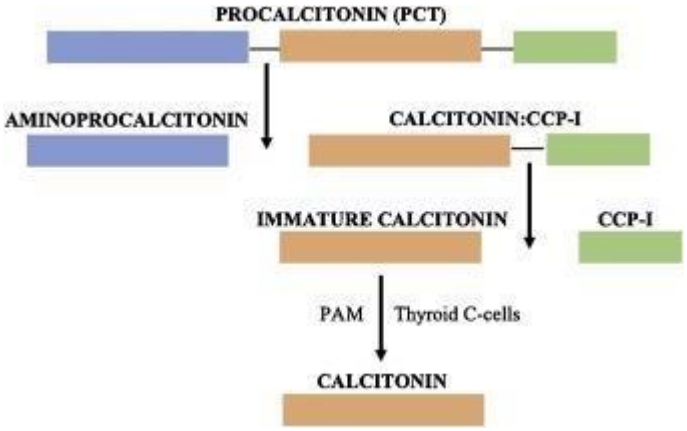
The 116-amino acid glycoprotein procalcitonin (PCT) is a building block for the hormone calcitonin.[7] Thyroid C cells normally release PCT, which acts in calcium metabolism by converting certain protein hydrolases into calcitonin. While PCT levels in the blood are very low in a healthy person, they can rise dramatically in response to systemic inflammation or a severe bacterial infection. This is because inflammation causes an upregulation of the expression of extrathyroidal non-neuroendocrine calcitonin genes, which in turn releases uncleaved PCT into the blood, increasing the plasma PCT concentration.[8] As a factual basis for clinicians to infer the duration of infection and subsequent management of patients, serum PCT levels in patients with combined severe bacterial infections are closely related to the duration of infection. Serum PCT typically rises rapidly within 6-8 hours and peaks at 24 hours.[9] In recent years, various studies.[10,12] have shown that serum PCT is useful for diagnosing burn sepsis early on. Having said that, there is currently no conclusive evidence about PCT's early diagnostic use for adult burn sepsis. The purpose of this meta-analysis was to give future clinical diagnoses and decision-making a solid foundation by methodically evaluating the diagnostic utility of serum PCT for burn sepsis in adults.

TRANSLATIONAL RELEVANCE: Medical imaging, immunology, inflammation, circulation, or coagulation researchers, biomarker researchers, and RCT developers will all find something of interest in this study.

CLINICAL RELEVANCE: Given these new developments, we take a close look at the diagnostic utility of existing evaluation tools and the security and effectiveness of existing therapies. Those doctors who treat patients who have suffered severe burns will find this

extremely useful. All members of the medical team, including surgeons who care for patients during admission, as well as those in the fields of emergency medicine, critical care, anesthesia, pain, internal medicine, pathology, nursing, and allied health, will find this information particularly useful.

BACKGROUND Burns have been studied for centuries.[13] "There is no more terrifying accident than a severe burn of a large area," said Haldor Sneve, an early practitioner of burn care, in 1905. These events are especially upsetting because of the severe pain the sufferer experiences and, even after recovery, the horrific disfigurement that remains''[14] Localised heat exposures can create life-threatening systemic consequences in the case of a severe burn, which is characterized by an acute wound.[15] Additional information on the treatment of radiation, friction, electrical current, and caustic chemical-induced acute wounds can be found elsewhere. Protein denaturation and coagulation are hallmarks of healthy skin deterioration.[16] Size, depth heterogeneity, behavior, and systemic repercussions are characteristics that distinguish severe burn wounds from other types of acute burns.



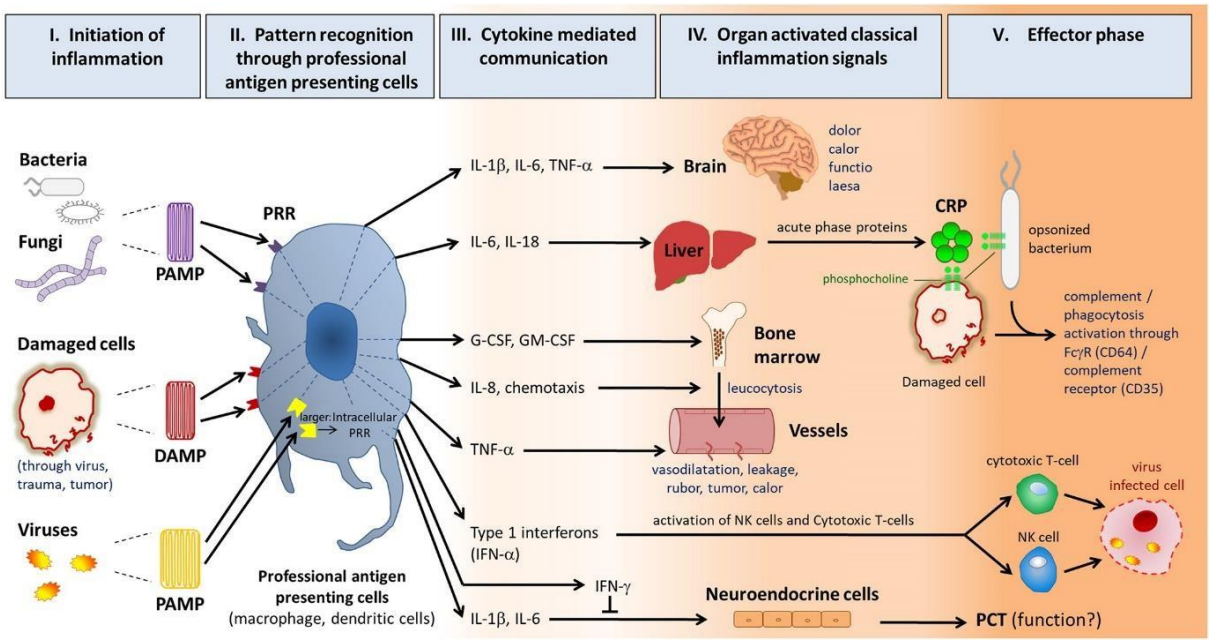
An example of a problem with wound size is the loss of healthy dermal and epidermal tissue equivalent to 1.5 square meters (m2) when a burn impacts 80% of the total body surface area (TBSA). There is a wide range of possible depths for any particular burn, and the severity of a burn can worsen over time, even after the initial injury has healed. Burn conversion describes this procedure.[17] Dermal thickness ranges from 1-4 mm and epidermal thickness from 0.1-1 mm in humans.[18] Poor healing outcomes and treatment are determined by the loss of both the dermis and the epidermis.[17] Hence, there is a want for a very accurate method of assessing burn depth.[12] Burns can be conceptualized by concentric zones.[16] The destruction of skin tissue occurs at the center of the hemorrhage, which is surrounded by a zone of stasis (lower skin perfusion) and a zone of hyperemia (increased skin perfusion). In theory, a fire might go

in any direction across these areas.[16] Lastly, there are systemic repercussions that are far-reaching after a burn. In this review, we will go over these consequences thoroughly. Wound treatment in intensive care units has been the subject of recent burn evaluations,[19] the metabolic response,[20] hypertrophic scarring,[21] smoke inhalation,[22] anesthetic, and hemodynamic considerations,[23] and developments in surgical techniques[24] yet this is the only evaluation that we are aware of that covers critical care, surgical, perioperative, and anesthetic treatment in recent years.

THE ACUTE MANAGEMENT OF PATIENTS WITH SEVERE BURNS

Care for victims of inhalation injuries There are several ways to lessen the chances of bad outcomes due to inhalational damage once bronchoscopy has verified it. Chest physiotherapy, coughing exercises, and early ambulation are all recommended based on the available evidence[22,25] Compared to ventilator modes with interrupted flow, continuous mechanical ventilation increases mortality and pneumonia incidence in intubated patients. These modes, which are covered in depth elsewhere, can involve varying frequencies of flow interruption from the ventilator to the patient while maintaining positive airway pressure.[26,27]

There is more research on pharmacological management alternatives. Nebulized N-acetylcysteine (NAC), salbutamol (a bronchodilator), and heparin (an anticoagulant) may shorten the time that patients need mechanical breathing following inhalational injuries, according to the available data.[28] There was no effect on coagulation markers from a dosage trial showing that 10,000 IU of nebulized heparin per dose improved lung damage ratings.[29] A growing body of research suggests that nebulized antithrombin can effectively lower the risk of pneumonia following inhalational insult.[30]



INTRAVENOUS FLUID RESUSCITATION

Early fluid administration following a burn has been linked to decreased mortality for more than a century.[31] It has been known for forty years that the mineral makeup and volume of the fluid compartments within blood vessels and between them are following a burn. Presents key ideas of intravenous fluid resuscitation and fluid compartments. A crystalloid solution, Hartmann's solution attempts to imitate the mineral makeup of extracellular fluid found in the body. The use of Hartmann's solution to several severely burned patients and the resulting decrease in the usual fatality rate was detailed in a landmark paper by Baxter and Shires.[32] Based on these results, a formula was developed (the Parkland formula) to determine the first volume of fluid resuscitation administered within the first twenty-four hours. The standard value is 4 milliliters per kilogram of body weight divided by the total body surface area (TBSA) burned.[33,34] Most burn centers utilize the Parkland formula,[33,34] However, serious side effects known as "fluid creep," including pulmonary edema and abdominal compartment syndrome, are linked to its usage.[35,37] Several equations may be used to calculate the initial volume and rate of Hartmann's solution for resuscitation; these include the Rule of Tens and the Modified Brooke formula, both of which take into account the patient's weight and the extent of the burn.[38] To ensure sufficient urine output, the Modified Brooke formula recommends administering 2 mL/kg/TBSA burn percent of Hartmann's solution intravenously over the first 24 hours, followed by 0.3-0.5 mL/kg/TBSA burn percent of colloid solution over the next 24 hours. Following the Rule of Tens, we estimate the burn size in TBSA percent to the closest

10%. For patients weighing 40-80 kg, multiply this figure by 10 to get the beginning fluid rate in mL/h.

CONCLUSION

Finally, extensive skin damage from burns increases the likelihood of infection and burn sepsis, making burns a very dangerous injury. If we want better patient outcomes and lower fatality rates, we must diagnose burn sepsis early. Results from early burn sepsis diagnosis using blood procalcitonin (PCT) levels are encouraging. Clinicians may learn a lot about the length of an infection and how to treat patients afterward by systematically evaluating serum PCT levels. The purpose of this meta-analysis is to lay a solid groundwork for the clinical diagnosis and decision-making of adult burn sepsis in the future.

To properly evaluate and treat severe burns, one must have a thorough understanding of their intricacies. When the dermis and epidermis are both severely damaged, as might happen with severe burns, the healing process isn't always smooth sailing. For appropriate therapy, it is essential to correctly determine the depth of the burn. The hemorrhage's center is the most severely damaged part of a burn lesion, which causes it to be classified into concentric zones. Severe burns can have far-reaching systemic consequences that affect many parts of patient care.

Care for inhalation injuries and intravenous fluid resuscitation are two of the most important factors in the acute management of patients with severe burns. Preventing complications after inhalation injuries requires a multipronged approach that includes early ambulation, coughing exercises, and chest physiotherapy. Patients suffering from inhalation injuries may be able to lessen their reliance on mechanical breathing with the use of pharmacological treatment alternatives, such as nebulized pharmaceuticals.

To lower death rates after a burn, it is critical to provide fluids quickly. Depending on the severity of the burn, many formulae may be employed to determine the correct volume of fluid resuscitation, one of which is the Parkland formula. But to avoid problems, possible adverse effects including fluid creep should be watched carefully.

Improving patient outcomes and lowering death rates requires breakthroughs in the detection and management of burn sepsis. Serum PCT levels and other evidence-based wound evaluation and treatment methods can help doctors provide better care for patients who have suffered

severe burns. Do your best and challenge yourself more.

REFERENCES

1. Dvorak JE, Ladhani HA, Claridge JA. Review of Sepsis in Burn Patients in 2020. Surg Infect (Larchmt), 2021; 22: 37-43.
2. Mannes M, Schmidt CQ, Nilsson B, Ekdahl KN, Huber-Lang M. Complement as a driver of systemic inflammation and organ failure in trauma, burn, and sepsis. Semin Immunopathol, 2021; 43: 773788.
3. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, Bellomo R, Bernard GR, Chiche JD, Coopersmith CM, Hotchkiss RS, Levy MM, Marshall JC, Martin GS, Opal SM, Rubenfeld GD, van der Poll T, Vincent JL, Angus DC. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA, 2016; 315: 801-810.
4. Ladhani HA, Yowler CJ, Claridge JA. Burn Wound Colonization, Infection, and Sepsis. Surg Infect (Larchmt), 2021; 22: 44-48.
5. Church D, Elsayed S, Reid O, Winston B, Lindsay R. Burn wound infections. Clin Microbiol Rev., 2006; 19: 403-434.
6. Rech MA, Mosier MJ, McConkey K, Zelisko S, Netzer G, Kovacs EJ, Afshar M. Outcomes in Burn-Injured Patients Who Develop Sepsis. J Burn Care Res., 2019; 40: 269-273.
7. Paudel R, Dogra P, Montgomery-Yates AA, Coz Yataco A. Procalcitonin: A promising tool or just another overhyped test? Int J Med Sci., 2020; 17: 332-337.
8. Wussler D, Kozhuharov N, Tavares Oliveira M, Bossa A, Sabti Z, Nowak A, Murray K, du Fay de Lavallaz J, Badertscher P, Twerenbold R, Shrestha S, Flores D, Nestelberger T, Walter J, Boeddinghaus J, Zimmermann T, Koechlin L, von Eckardstein A, Breidthardt T, Mueller C. Clinical Utility of Procalcitonin in the Diagnosis of Pneumonia. Clin Chem., 2019; 65: 1532-1542.
9. Hamade B, Huang DT. Procalcitonin: Where Are We Now? Crit Care Clin., 2020; 36: 23- 40.
10. Maisel A, Neath SX, Landsberg J, Mueller C, Nowak RM, Peacock WF, Ponikowski P, Möckel M, Hogan C, Wu AH, Richards M, Clopton P, Filippatos GS, Di Somma S, Anand I, Ng LL, Daniels LB, Christenson RH, Potocki M, McCord J, Terracciano G, Hartmann O, Bergmann A, Morgenthaler NG, Anker SD. Use of procalcitonin for the diagnosis of pneumonia in patients presenting with a chief complaint of dyspnoea: results from the BACH (Biomarkers in Acute Heart Failure) trial. Eur J Heart Fail, 2012; 14: 278-86.

11. Alba GA, Truong QA, Gaggin HK, Gandhi PU, De Berardinis B, Magrini L, Bajwa EK, Di Somma S, Januzzi JL Jr; Global Research on Acute Conditions Team (GREAT) Network. Diagnostic and Prognostic Utility of Procalcitonin in Patients Presenting to the Emergency Department with Dyspnea. Am J Med., 2016; 129: 96-104.e7.
12. Huo J, Wang L, Tian Y, Sun W, Zhang G, Zhang Y, Liu Y, Zhang J, Yang X, Liu Y. Gene Co-Expression Analysis Identified Preserved and Survival-Related Modules in Severe Blunt Trauma, Burns, Sepsis, and Systemic Inflammatory Response Syndrome. Int J Gen Med., 2021; 14: 70657076.
13. Sydnenham. On the treatment of burns. Lancet., 1844; 43: 153.
14. Sneve H. The treatment of burns and skin grafting. J Am Med Assoc., 1905; 45: 1–8.
15. Hettiaratchy S, Dziewulski P. ABC of burns: pathophysiology and types of burns. BMJ., 2004; 328: 1427–1429.
16. Jackson DM. The diagnosis of the depth of burning. Br J Surg, 1953; 40: 588–596.
17. Schmauss D, Machens H-G, Harder Y, Finck T, Rezaeian F, Wettstein R. Treatment of secondary burn wound progression in contact burns—a systematic review of experimental approaches. J Burn Care Res., 2015; 36: e176–e189.
18. Abadie S, Jardet C, Colombelli J, et al. 3D imaging of cleared human skin biopsies using light-sheet microscopy: a new way to visualize in-depth skin structure. Skin Res Technol., 2018; 24: 294–303.
19. Rowan MP, Cancio LC, Elster EA, et al. Burn wound healing and treatment: review and advancements. Crit Care., 2015; 19: 243.
20. Porter C, Tompkins RG, Finnerty CC, Sidossis LS, Suman OE, Herndon DN. The metabolic stress response to burn trauma: current understanding and therapies. Lancet., 2016; 388: 1417–1426.
21. Finnerty CC, Jeschke MG, Branski LK, Barret JP, Dziewulski P, Herndon DN. Hypertrophic scarring: the greatest unmet challenge after burn injury. Lancet., 2016; 388: 1427–1436.
22. Enkhbaatar P, Pruitt BA, Jr., Suman O, et al. Pathophysiology, research challenges, and clinical management of smoke inhalation injury. Lancet., 2016; 388: 1437–1446.
23. Anderson TA, Fuzaylov G. Perioperative anesthesia management of the burn patient. Surg Clin North Am, 2014; 94: 851–861.
24. Zuo KJ, Medina A, Tredget EE. Important developments in burn care. Plast Reconstr Surg 2017;139:120e–138e.
25. Gupta K, Mehrotra M, Kumar P, Gogia AR, Prasad A, Fisher JA. Smoke inhalation

injury: etiopathogenesis, diagnosis, and management. Indian J Crit Care Med., 2018; 22: 180– 188.

26. Allan PF, Osborn EC, Chung KK, Wanek SM. High-frequency percussive ventilation revisited. J Burn Care Res., 2010; 31: 510–520.
27. Miller AC, Ferrada PA, Kadri SS, NatarajBhandari K, Vahedian-Azimi A, Quraishi SA. High-frequency ventilation modalities as salvage therapy for smoke inhalation- associated acute lung injury: a systematic review. J Intensive Care Med., 2018; 33: 335–345.
28. McGinn KA, Weigartz K, Lintner A, Scalese MJ, Kahn SA. Nebulized heparin with N-acetylcysteine and albuterol reduces the duration of mechanical ventilation in patients with inhalation injuries. J Pharm Pract., 2019; 32: 163–166.
29. Elsharnouby NM, Eid HE, Abou Elezz NF, Aboelatta YA. Heparin/N-acetylcysteine: an adjuvant in the management of burn inhalation injury: a study of different doses. J Crit Care, 2014; 29: 182.e1–182.e4.
30. Kowal-Vern A, Orkin BA. Antithrombin in the treatment of burn trauma. World J Crit Care Med., 2016; 5: 17–26.
31. Fauntleroy AM, Hoagland AW. The treatment of burns: as exemplified in thirty-two cases. Ann Surg., 1919; 69: 589–595.
32. Baxter CR, Shires T. Physiological response to crystalloid resuscitation of severe burns. Ann N Y Acad Sci., 1968; 150: 874–894.
33. Baker RHJ, Akhavani MA, Jallali N. Resuscitation of thermal injuries in the United Kingdom and Ireland. J Plast Reconstr Aesthet Surg., 2007; 60: 682–685.
34. Gordon M, Marvin J, Greenfield E, et al. Regional and institutional variation in burn care. J Burn Care Rehabil., 1995; 16: 85–90.
35. Engrav LH, Colescott PL, Kemalyan N, et al. A biopsy using the Baxter formula to resuscitate burns or do we do it like Charlie did it? J Burn Care Rehabil., 2000; 21: 91–95.
36. Cartotto RC, Innes M, Musgrave MA, Gomez M, Cooper AB. How well does the Parkland formula estimate actual fluid resuscitation volumes? J Burn Care Rehabil., 2002; 23: 258–265.
37. Friedrich JB, Sullivan SR, Engrav LH, et al. Is supra-Baxter resuscitation in burn patients a new phenomenon? Burns., 2004; 30: 464–466.
38. Pham TN, Cancio LC, Gibran NS. American Burn Association practice guidelines burn shock resuscitation. J Burn Care Res., 2008; 29: 257–266.