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Assessment of Platelet Count as a Prognostic Indicator in Burn Septicaemia: A Prospective Observational Study

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Abstract
Burn fatalities are mostly caused by septicemia. Only if septicemia is discovered early enough-which calls for an extremely sensitive prognostic indicator-can burn victims be spared. The platelet count was investigated in this work as a prognostic factor. Seventy patients with more than twenty percent burns, regardless of age or sex, who were admitted to the surgical wards of Hospital. (Raj), had their cultures studied for this research. The cultures were taken from the burn region 48 hours after the burn occurred, then again after 7 daysfinally, at most, up to the 21st post-burn day of hospitalisation. The patient's blood was drawn and processed simultaneously in order to separate the germs. For the first twenty-four hours, all patients received topical antibiotics. In non-survivors, a progressive decrease in platelet count was seen in the days that followed the burn until these individuals passed away. While a steady increase in platelet count was seen in survivors When post-burn septicemia is detected early, a particularly sensitive prognostic indicator is a declining platelet count.

INTRODUCTION

In burn patients with severe homeostatic abnormalities and impaired immune responses, platelets are crucial. Platelets are minute pieces of cytoplasm from megakaryocytes that are essential to both primary and secondary homeostasis because of the critical coagulation cascade processes that take place on their phospholipid surface. They perform inflammatory functions in addition to their main role in hemostatic control. By interacting with leukocytes and endothelial cells, releasing inflammatory mediatorsexpressing pro-inflammatory surface chemicals, they contribute to the development of both acute and chronic immunological responses[1].

According to Michael Peck in Epidemiology of Burn Injuries Globally, burns occur often, with an incidence of 1.1 per 1,000 people. Even though they hurt, most burn injuries are rather mild. On the other hand, a tiny percentage of people suffer severe, deep burns that result in either lifelong deformity or death. The patient's age and the burn area have historically been used as the main indicators of death after thermal damage. Additional variables found during hospitalisation may also be useful in properly identifying individuals who are at high risk of passing away[2].

When bacterial infections are linked to bacteremia, thrombocytopenia is almost always the outcome of elevated platelet consumption. The lowered platelet count might be a singular observation or it could be connected to a widespread intra vascular coagulopathy. In burn victims, thrombocytopenia often develops early and may be a sign of bacteremia[3].

For burn victims, sepsis continues to be the leading cause of mortality. The patient has septicemia as soon as bacteria enter the bloodstream. When a patient has septicemia, almost every organ system in the body is impacted, which may result in multiple organ dysfunction syndrome, systemic inflammatory response syndromeultimately death.

Therefore, the only time burn victims may be salvaged is during the early stages of septicemia, before several organs are irreversibly damaged. In septicemia, a declining platelet count happens extremely early on-even before clinical signs and symptoms manifest. To save the lives of burn victims, early identification and treatment may be instituted, but this needs the existence of sensitive measures that can identify septicemia in its early phase.

Burn injuries are linked to a high rate of morbidity and death, as well as unexpected and catastrophic trauma. Physical (friction, high temperature, cold, radiationelectricity) and chemical variables are among the many causal causes[4]. However, most burn injuries are thermal in nature, brought on by hot liquids, solids, or fire[5]. In 2018, the World Health Organisation released a study estimating that over 11 million burn cases occur globally each year, with burn injuries accounting for as many as 180,000 fatalities[6]. Looking back almost ten years, burn mortality has declined from the 300,000 deaths reported in 2011[7]. Advances in the treatment of burn patients in critical care units, as well as better wound care, infection control procedure she modynamic problem management, are partly responsible for the notable increase in the survival rate of burn patients[8,9]. But the death rate is still too high, especially for patients who have had severe burns.

MATERIALS AND METHODS

The current research will be conducted on 70 burn cases that occurred between 2013 and 2016 in the surgical departments.

Inclusion Criteria:

- All adult burn patients (age 15 and older), regardless of gender, admitted to the burn ward.
- 20%-70% of the body's surface area is burned. >70% and <20% of burns were disqualified from the research. The history of septicemia in burn victims informs our investigation.

Exclusion Criteria:

- Very few patients in fewer than 20% of burn cases develop septicemia, thus, these cases are eliminated. Very high and early mortality in more than 70% of burn cases is caused by hypovolemia, these patients pass away even before septicemia develops. They were so disqualified from the research.
- Children were not allowed to participate in the research due to the very small number of eligible patients, which made statistical inference challenging.

Investigations:

- **Platelet count:** Using an automated analyzer (XP100) or visual techniques.
- TLC and DLC (using an automated XP100 analyzer).
- Emphasise sensitivity and culture.
- Creatinine in serum.
- Blood sugar

Seventy patients with more than twenty percent burns, regardless of age or sex, who were admitted to the surgical wards of Hospital, had their cultures studied for this research. The cultures were taken from the burn region 48 hours after the burn occurred, then again after 7 daysfinally, at most, up to the 21st post-burn day of hospitalisation. The patient's blood

was drawn and processed simultaneously in order to separate the germs. For the first twenty-four hours, all patients received topical antibiotics.

In these instances, the next logical step for the diagnostic and therapeutic work-up was thought to be the swab culture, blood culture, full thickness burn tissue biopsy culturesculture sensitivity testing.

In each instance, a thorough clinical history was obtained. Additionally noted were the burn's proportion and intensity. A patient's prior medical history, personal history, family backgroundmental health were also recorded.

To learn more about the patient's problems, routine laboratory tests were performed, including Hb, TLC, DLC, ESRplatelet counts. Blood urea and sugar tests were also performed.

Depending on the problems that arise after a burn, other studies could be added.

Material:

- Petri dish
- Semi-Micro balance
- Sterile physiological saline solution (0.9%)
- Dry and sterile culture flasks and test tubes
- Disposable syringe (10 cc) and needles.
- Appropriate culture media
- Platinum Loop
- Bunsen Burner
- EDTA bulb
- Neubauer counting chamber
- Light microscope

Procedure: Patients were informed about the technique they had undergone after topical medications were initially removed using cotton pads soaked in sterile saline in order to get a culture of the burn area.

Specimen Collections Swab Culture: The chosen skin region was cleaned with saline gauzethe swabs were immediately removed from the wound surface using swab sticks. They were then promptly reinstalled in the dry, sterile test tubes.

Platelet Count: Two millilitres of venous blood were drawn, placed in an EDTA bulb (an anticoagulant)quickly mixed gently to estimate the platelet count. This was mixed with 1.9 ml of diluent (1 in 20 dilution) and 0.1 ml of blood. Ammonium oxalate (10 g/l) was the diluting agent utilised. After adding suspension to a wet petri dish, the Neubauer counting chamber was covered and the platelets were allowed to settle for 20 minutes. Under normal lighting conditions, platelets looked as tiny, extremely refractile particles under a light microscope, but not microscopic.

RESULTS AND DISCUSSIONS

In our study total 70 patients studied. Out of 70 patients, 36 patients were female and 34 were male patient. In survivors there is initial decline then gradual rise in platelet counts was observed on subsequent post burn days till discharge of these patients. [Table 1]

In non-survivors gradual decline in platelet count was observed on subsequent post-burn days till death of these patients. [Table 2]

In a significant number of non survivors (79.3%) platelet count was low before their deathin a significant number of survivors (87.8%) platelet count was normal before their discharge (P<0.001). [Table 3]

In a significant number of non survivors (62.0%) total Incidence abnormal value of leucocyte count was before their deathin a significant number of survivors (70.7%) total Incidence of normal value of leucocyte count was before their discharge (P<0.001).[Table4]

After thrombocytopenia, survivors showed a relative rise in platelet count, whereas non-survivors did not.4 This biphasic pattern was seen among survivors in our research, but only a steady drop was observed in non-survivors. Both investigations demonstrated a significant mortality rate in relation to thrombocytopenia. A 2002 research by Richard Strauss[10] shown that thrombocytopenia is often seen in patients in medical critical care units. There is an independent correlation between mortality in the critical care unit and a decrease in platelet counts of =30%, but not thrombocytopenia per se. Platelet count measurements performed on a regular basis are useful and easily accessible indicators for tracking a patient's health[10]. According to our research, a persistent decrease and a platelet count of <1.5 lakh/mm^3 are linked to a high death rate. According to a research by Vanderschueren, Steven[11] thrombocytopenia is a prevalent condition in intensive care units (ICUs) and is a straightforward and easily accessible risk marker for mortality that may be used in addition to or instead of recognised disease severity indicators. In adult ICU patients, both a low platelet count and a significant decline in platelet count are predictive of a poor vital outcomethe findings of this research are consistent with our own. A research was conducted by Shanti Prakash Kujur, Devpriya Lakraothers. 480 burn patients between the ages of 18 and 60 were examined, the proportion of burns ranged from 20% to 70%. For every patient, the platelet count was examined. Day 1, 3, 7, 1421 of the patients' investigations into their platelet counts were conducted. Estimations of serum

Table 1: Mean Platelet count (in lakh/mm3) in survivor

	1st day	3rd day	7th day	14th day	21st day
Mean Value±SD	344.11±77	276.95±78	266.81±95	304.36±88	326.30±77

Table 2: Mean Platelet count (in lakh/mm3) in non-survivor

	1st day	3rd day	7th day	14th day	21st day
Mean Value±SD	298.12±83	216.33±96	132.27±38	122.27±38	132.86±31

Table 3: Incidence low platelet count in survivors and non survivors

Platelet Count Lakh/Mm3	Survivor	Non- Survivor	Total
‹1.5	5(12.1%)	23(79.3%)	28(40%)
›1.5	36(87.8%)	6(20.6%)	42(60%)
Total	41	29	70

Table 4: Shows comparison of leucocyte counts in survival and non survival groups.

	Survivor	Non- Survivor	Total
Abnormal value >12,000/µl or <4000/µl	12(29.2%)	18(62.0%)	30(42.8%)
Normal value (12,000/µl -4000/µl)	29(70.7%)	11(37.9%)	40(57.1%)
Total	41	29	70

creatinine, neutrophil count other parameters were also performed. According to this research, the platelet count progressively climbed in survivors and remained there until their release, but it steadily decreased in non-survivors. The standard t test was used to examine the statistical significance of the differences in mean platelet counts between survivors and non-survivors on various post-burn days. In various post-burn days, i.e., (Day 1, 3, 7, 14 ans 21), it was found that the real difference between two means is more than double of the SED between two means. Thus, there is a substantial difference (P<0.05). Our investigation is supported by these findings. Other laboratory measures including TLC, neutrophil count serum creatinine do not substantially change with the onset and development of septicemia, according to research by Shanti Prakash Kujur, Devpriya Lakra[12] hence, their utility as prognostic indicators of septicaemia is less relevant. However, the findings of our investigation indicated that the entire incidence of abnormal leucocyte count value was before death in a considerable number of non-survivors (68.42%) and the total incidence of normal leucocyte count value was before discharge in a significant number of survivors (77.42%) (P<0.001). [Table 4] The findings contradicted the research conducted by Shanti Prakash Kujur, Devpriya Lakracolleagues. The research conducted by EI-Sonbaty M.A., EI-0tiefy M.A., *et al*. (1996) revealed that there was a considerable increase in haemoglobin concentrations just after the burn, particularly in the non-survivors. By day 4 post-burn in the non-survivors and by day 6 post-bum day in the survivors, this high level had progressively dropped to below the control level[13]. The results of our research demonstrated a significant difference in haemoglobin levels between non-survivors (73.68% pt. below normal) and survivors (54.84% pt. normal) before to discharge (P>0.05). Additionally, our research findings demonstrated that both survivors and non-survivors rhad noticeably elevated haemoglobin concentrations in the first several hours after the burn. The high level in both survivors and non-survivors eventually dropped to below normal on the next post-burn days. These observations align with the previous research. Our research findings demonstrated that, from the time of these patients' discharge or death, there were no appreciable differences in the mean creatinine levels between non-survivors and survivors that the mean creatinine value was within the normal range. These outcomes matched those of the research by Shanti Prakash Kujur, Devpriya Lakracolleagues[12].

CONCLUSION

Survivors get a rebound increase in platelet count in the days after their burn. If a patient does not survive, the pattern of decline continues until their death. When resuscitating and caring for patients with severe burns, it is crucial to keep an eye on the platelet count. Therefore, in burn patients, a serial decline in platelet count may be utilised as a prognostic indication to help diagnose septicemia early. It facilitates the start of septicemia therapy early on, improving the patient's prognosis. The serial platelet count in the post-burn period can be used as a prognostic indicator in burn patients because whenever the count starts to decline, all measures to support the general condition of the burned patient should be initiated. These measures include the administration of intravenous fluids and antibiotics, optimal care of the burn wound, debridement or escharectomyblood transfusion.

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