Original Research Paper

June 1000 Karley

ROLE OF PLATELET COUNT AS A PROGNOSTIC INDICATOR IN PATIENTS WITH BURNS

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ABSTRACT Evaluation of platelet count and its significance in early detection of post burn septicemia for commencement of timely vigorous treatment against it. This study was undertaken to analyse various factors such as age, sex, cause of burn, percentage and depth of burn, pre-existing medical conditions, cause of death in patients and role of platelets in it. In non-survivors gradual decline in platelet count was observed and minimal platelet count was observed before death of the patient, while in survivors gradual rise in platelet count was observed. No significant variation observed in other laboratory parameters such as total neutrophil count and serum creatinine. In significant number of non survivor platelet count was low before their death and in significant number of survivor platelet count was normal before their discharge.[1][2]

KEYWORDS:

INTRODUCTION

Infection is the major challenge in the management of burn patients and is a leading cause of morbidity and mortality. It is estimated that infection is the primary or major contributing cause in 75% of deaths from thermal injury[3]. Sepsis occurs when the balance of interaction between the host and opportunistic organisms is altered unfavourably. Important determinants of sepsis in burn patients are factors such as creation of new portals of entry, altered host defences and exposure to potential pathogenic and opportunistic organisms [4].

Thrombocytopenia is caused by several factors including infection and sepsis. Normal to high platelet counts almost always occur in burn patients who are stable and are not an indication of the imminent likelihood of sepsis. However, thrombocytopenia is one of the major manifestations of infection[5].

STUDY MATERIALS AND METHODS

Present study was conducted on 50 burn patients admitted in surgical and plastic surgery units of Guru Gobind Singh Medical College and Hospital, Faridkot. The study was conducted to evaluate burn sepsis with reference to platelet count as a diagnostic and prognostic indicator of sepsis.

All the patients were chosen at random. The extent of burn injury was determined by 'rule of nines' (Wallace).

A quick brief history regarding mode of injury and general physical examination was done in every case. The patients were resuscitated with intravenous fluids and were given appropriate antibiotics and analgesics.

Fluid calculation during first 24 hours was done by using Parkland's formula ($4 \times body$ weight \times percentage of burns). Maintainance fluids were given after 24 hours according to daily losses of electrolytes, water, daily caloric needs and urine output.

Blood transfusion was given whenever it was needed. Patients were also fed orally or by nasogastric tube according to the condition of the patient. Any associated disease or illness was recorded. Routine investigations like Hb BT CT TLC DLC Urine-Complete Examination FBS Blood urea Serum Creatinine Serum Electrolytes were carried out

All the patients were monitored daily regarding general condition, temperature, pulse rate, respiratory rate, urine output and condition of the wound till the patients got completely stabilized.

Appropriate care was given to burn wound in the form of antiseptic dressings or antiseptic application with exposure. Patients were observed daily for any clinical evidence of sepsis. Pus for culture and sensitivity was sent when required. Blood culture was sent whenever there was any clinical evidence of septicemia.

Blood samples for estimation of platelets were collected on the day of admission, and on days 3, 7, 14 and 21. Platelet counts were estimated by BAIN's method as follows :

0.02 ml of EDTA anticoagulant blood was diluted and mixed with 1.98ml of diluting fluid with 1-2 drops of 1% brilliant cresyl blue. After 2 minutes improved Neubauer chamber was charged and placed under a petridish with moist filter paper for 20 minutes. This allowed the platelets to settle on the surface of the counting chamber. The moist filter paper kept the air moist and prevented drying of the chamber.

Platelet appeared as highly refractile particles under the high power lens. Platelets were counted in one or more square mm. Atleast 100 platelets were counted.

CALCULATIONS

Platelet count/l =<u>number of cells counted X dilution X 10⁶</u> Volume counted The results so obtained were analysed and compared so as to find out the relationship of platelet counts to burn wound sepsis and their efficacy in predicting morbidity and mortality in burn cases.

RESULTS: MEAN PLATELET COUNT IN SURVIVORS AND NON-SURVIVORS

| Day | Survivors | Non-survivors |
|-----|-----------|---------------|
| 1 | 2.59 | 2.01 |
| 3 | 2.38 | 1.56 |
| 7 | 2.28 | 1.32 |
| 14 | 2.49 | 1.17 |
| 21 | 2.61 | 1.05 |

Mean platelet count on first day was 2.59 lacs in survivors whereas in case of non-survivors mean platelet count on first day was 2.01 lacs. On day 3, mean platelet count in survivors was 2.38 and in non-survivors it was only 1.32. On 14th day, mean platelet count in survivors was 2.49 and in non-survivors it was 1.17. On day 21 mean platelet count in survivors was 2.61 and in non-survivors it was 1.05.

MEAN PLATELET COUNT IN DIFFERENT DAYS

| No. of | %age of | Mean platelet count on different | | | | |
|----------|---------|----------------------------------|-------|-------|--------|--------|
| patients | Burns | Day l | Day 3 | Day 7 | Day 14 | Day 21 |
| 7 | 20-30 | 2.67 | 2.45 | 2.49 | 2.62 | 2.71 |
| 11 | 31-40 | 2.67 | 2.48 | 2.46 | 2.64 | 2.73 |
| 4 | 41-50 | 2.7 | 2.55 | 2.46 | 2.56 | 2.7 |
| 6 | 51-60 | 2.4 | 2.14 | 1.96 | 2.15 | 2.35 |
| 13 | 61-70 | 2.25 | 1.81 | 1.60 | 1.66 | 1.99 |
| 3 | 71-80 | 1.96 | 1.56 | 1.20 | 1.9 | 1.9 |
| 3 | 81-90 | 1.63 | 0.99 | - | - | - |
| 3 | 91-100 | 1.7 | 1.21 | 0.96 | - | - |

Above table shows mean platelet count on different days against extent of burns. The value of platelet count is in lacs. X-axis shows days of hospital stay, Y-axis shows %age of burns

PLATELET COUNT AND SURVIVAL OF PATIENTS

| Platelet count | Survivors | Non-survivors | Total |
|----------------|-----------|---------------|-------|
| Low | 5 | 17 | 22 |
| Normal | 25 | 3 | 28 |
| Total | 30 | 20 | 50 |

Above table shows platelet count and survival of the patients. Amongst survivors, only 5 (16.7%) patients had low platelet count while most of the cases (83.3%) had normal platelet count. However, in case of non-survivors 17 patients (85%) had low platelet count and only 15% had normal platelet count.

PLATELET COUNT AND BLOOD CULTURE IN BURN CASES

| Platelet Count | Blood culture | | Total |
|----------------|---------------|----------|-------|
| | Positive | Negative | |
| Low (<1.5) | 20 | 5 | 25 |
| Normal | 2 | 23 | 25 |
| Total | 22 | 28 | 50 |

Above table shows platelet count and blood culture in burn cases. Eighty percent of the cases with low platelet count had positive blood culture while only 8% of the patients with normal platelet count had positive blood culture.

In the present study, mean platelet count decreased upto day 7 in both survivors and non-survivors. However from day 7 onwards, platelet counts steadily rose in case of survivors while maintaining a downward trend in case of non-survivors. Konickova (1979) also demonstrated the value of falling platelet count as one of the criteria in diagnosing endotoxaemia [6]. Housinger et al. (1993) established the relationship of falling platelet count in diagnosing sepsis and emphasized that the platelet count was an independent predictor of sepsis. The initial thrombocytopenia was related to the severity of the injury. As the extent of injury increases, there is an increase in the degree of thrombocytopenia [5]. Wolf et al. (1997) found enteral feeding intolerance, hyperglycemia and thrombocytopenia as indicators of sepsis in burn patients [7]. Konter – Thioulouse et al. (1985) also found close relationship between plasma fibronectin deficiency (protein derived from platelets) and sepsis in burn patients [8].

Thrombocytopenia is caused by several factors including infection and sepsis. Normal to high platelet counts almost always occur in burn patients who are stable and are not an indication of the imminent likelihood of sepsis. However thrombocytopenia is one of major manifestations of the infection [4]. Enuresis et al. (1971) had suggested that Initial thrombocytopenia was due to platelet destruction by extrinsic mechanism following which there is a marrow megaka ryocytic response which creates secondary thrombo cytosis. Jefferson (2007) reported that 42.9% of non-survivors had <100,000 platelets and 92% of them died[9]. Vasantharaja (2016) found that the fall in platelet count is associated with bad prognosis in burn patients [10].

In the present study, amongst survivors blood culture was positive in 7 cases (23.3%) while 76.7% cases had no growth in their blood culture whereas in case of non-survivors, 75% cases showed positive blood culture. Also 80% of the cases with low platelet count had positive blood culture while only 8% of the patients with normal platelet count had positive blood culture. Again the wound culture was positive in 16 survivors (53.3%), while 55% of the non-survivors showed positive wound culture

In the present study amongst survivors only 5 cases (16.7%) had low platelet count, while most of the cases (83.3%) had normal platelet counts. Kaukinen et al. (1985) found the relationship between platelet count, sepsis and survival in pediatric burn patients. Thirty two pediatric patients who sustained lethal burn injuries were compared with 32 patients with similar burns who survived. All but only one of the 32 non-survivors developed a low platelet count while only 10 of the survivors had similar occurrence.

Wolf et al. (1997) found thrombocytopenia as one of the indicators of sepsis associated mortality in burned children. Similarly Kaukinen et al. (1985) found thrombocyte numbers as one of the prognostic indicators in burned patients.

Unbelievable decrease in blood platelets in severely burned patients during the treatment of skin grafting caused two patients to unexpected death. Maduli et al. (1999) also found low platelet count in most of the non-survivors. Konterthioulouse et al. (1988) found value of depletion of circulating PFN in the development of infection and in determining prognosis. Vasantharaja (2016) concluded death as an ultimate effect of septicemia associated with fall in platelet counts. Beiming (2017) also concluded the use of platelet count as an early objective indicator of prognosis in burn casualties [12].

In the present study out of 50 cases, 30 cases (60%) survived while 40% died. Mortality was significantly higher in those cases where extent and depth of burns was more. It was also higher in those cases who had persistently low platelet counts and positive blood cultures. Delay in reaching emergency was also responsible for higher deaths. In a study on 310 burned patients 27 of whom died found that severity of burn injury, advanced age of patients, race of the patients, cause of the burns, preexisting medical problems, inadequate or inappr opriate early resuscitative measures and possible errors or over sights in the management of a few patients contributed to mortality in burn cases[13].

CONCLUSIONS

To conclude, it may be said that the factors which contribute towards increased morbidity and mortality in burn cases are advanced age of the patient, extent and depth of burns and delay in starting treatment. However, the development of sepsis with its complications is the most important cause of morbidity and mortality.

Mortality was found to be significantly higher among those patients whose blood cultures were positive. Thrombo cyto penia occurred in most of the cases with positive blood cultures indicating thereby that it is an important predictor of development of sepsis and outcome. Also thrombocytopenia preceded the development of early predictors of sepsis like temperature, loss of appetite, enteral feeding intolerance, tachycardia and leukocytosis. So it can be concluded that serial platelet counts can be independently used to identify those at risk to develop systemic infection, its complications and final outcome.

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