e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(1); 345-348

Original Research Article

Correlation between Level of Platelets Aggregation and Severity of Burns in Acute Thermal Burns Patient

Govind[1], R K Srivastava[2], Sunil Ranga[3], Ajay Pal[4*]

[1]Assistant professor, Dept. of Plastic and Reconstructive surgery, Mahatma Gandhi Hospital, Jaipur
[2]Professor and Consultant, Dept. of Burns, Plastic and Reconstructive surgery, VMCC and Safdarjung Hospital, New Delhi
[3]Professor and Consultant, Dept. of Pathology, VMCC and Safdarjung Hospital, New Delhi
[4]2nd Yr Mch Resident, Dept. of Plastic and Reconstructive surgery, Mahatma Gandhi Hospital, Jaipur

Received: 25-10-2023 / Revised: 23-11-2023 / Accepted: 26-12-2023
Corresponding Author: Dr. Ajay Pal
Conflict of interest: Nil

Abstract:

Introduction: In thermal burns patients the alteration of hemostatic mechanism is due to oxidative stress and sepsis.

Aim: To evaluate the level of platelets aggregation in acute thermal burns patient and correlation between level of platelet aggregation and severity of burns.

Method: Study conducted on 30 cases of thermal burns and 30 healthy volunteers as control aged between 20-50 years at the Department of Burns, Plastic and Maxillofacial surgery, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi from Jan. 2015-June 2016. Patients with thermal burns involving more than 20% of total body surface area, Patients with age between 15-50 years and who got Hospitalization within 24 hrs of burn injury were included in the study.

Result: Patients ranged from 16 to 45 years with a mean age of 30.9 yr. Comparison of the platelet aggregation after the addition of ADP in both case and control groups were done and the mean platelet aggregation values were calculated. Mean aggregation percentage in the case group was found to be 64.6± 13.23% while that in the control group came out to be 71.3± 4.18% (p value >0.05) but significant difference was seen on 3rd and 5th day.

Conclusion: Thermal burn injury causes severe abnormality of ADP and collagen induced platelet aggregation in acute phase. This abnormalities of platelet aggregation correlate consistently with severity of burn in form of increased TBSA.

Keywords: Platelets aggregation, Thermal burns, ADP.

Introduction

In thermal burns patients the alteration of hemostatic mechanism is due to oxidative stress [1] and sepsis [2]. Haemostasis disorders are an important element of burn disease pathogenesis and failure to understand their progress and therapeutic management can increase morbidity and mortality of acute thermal burns patient [3]. Impairment of skin structure and loss of skin functions is found in thermal trauma. Bleeding tendency is characteristic finding in early phase of acute burn, hypercoagulability is found in the late phase [4].

Released inflammatory mediators and cytokines migrate to the systemic circulation when the TBSA exceeds 30%, inducing systemic inflammatory response to injury. In patients with severe burns who survive the initial period of treatment; leading cause of death is infection as a consequence of impairments in cellular and humoral immune responses [5]. An inverse relationship is found in several studies between the severity of disease and the platelet counts [6]. In severely burned patients sustained thrombocytopenia is regarded as a poor prognostic indicator [7]. In intensive care patients it is observed that in the pathogenesis of organ failure, hematologic factors particularly platelets and coagulation system play an important role. To reduce morbidity and mortality in critically ill patients with coagulation abnormalities it is crucial to have a proper differential diagnosis and to initiate adequate (supportive) treatment strategies [8].

A similar association is found in burns patient; however, in acute thermal burns patient research concerning correlation between level of platelets aggregation and severity of burns are rare and mostly case reports. In this prospective

observational study, aim was to study level of platelets aggregation in acute thermal burns patient and its correlation with severity of burns.

Aim

To evaluate the level of platelets aggregation in acute thermal burns patient and correlation between level of platelet aggregation and severity of burns.

Method

This was a Prospective observational study conducted on 30 cases of thermal burns and 30 healthy volunteers as control aged between 20-50 years at the Department of Burns, Plastic and Maxillofacial surgery, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi from Jan. 2015-June 2016. Patients with thermal burns involving more than 20% of total body surface area, Patients with age between 15-50 years and who got Hospitalization within 24 hrs of burn injury were included in the study.

Written consent were taken. Evaluation of platelet aggregation in venous blood sample was done within 24hr, on 3rd and on 5th post burn day. Data was collected and statistical analysis was done.

Result

Table 1: Study population

	Number	Percentage
Case	30	50.0
Control	30	50.0

In this study we had taken 30 cases of thermal burns and 30 healthy volunteers as control

Table 2: Age and Sex distribution

Age Distribution of cases	Percentage
≤20	6.66%
21-30	46.67%
31-40	36.67%
41-50	10.00%
Age Distribution of control	
≤20	6.67%
21-30	40%
31-40	40%
41-50	13.33%
Sex Distribution of cases	
Male (15)	50%
Female (15)	50%
Sex Distribution of control	
Male (16)	53.33%
Female (14)	46.67%

The age of the patients in our study ranged from 16 to 45 years with a mean age of 30.9 and a standard deviation of 7.24. Maximum number of patients were in the age group of 21-30 years i.e. 46.67% followed by those in the age group of 31-40 years i.e. 36.67% followed by the age group 41-50 years i.e. 10% and the least number of cases belonged to the age group of <20 years i.e. 6.66%. Out of 30 cases that were taken in our study, 15 cases were females that came out to be 50% of the total number of cases and 15 were males that constituted 50% of the cases. The age of the control group i.e. healthy volunteer in our study ranged from 18-43 with a mean value of 31.23 and a standard deviation of 6.86. The maximum number of controls came under the age group of 21-30 and 31-40 years i.e. 40% both followed by those in the age group of 41-50 years that accounted for 13.33% and the least were in the age group <20 years that were 6.67% of the control population. As far as the sex distribution amongst control was considered, 14 were females that accounted for 46.67% of the control population and 16 were males that accounted for 53.33% of the control population.

Table 3: Comparison of ADP induced platelet aggregation between cases and control

Within 24 hr.	Mean	SD	Median	Minimum	Maximum	P Value
Case	52.5%	13.74%	55%	24%	76%	0.0018
Control	61.3%	3.34%	61.5%	55%	67%	
On 3rd day						
Case	49.6%	12.14%	49.5%	22%	70%	<.0001
Control	61.1%	2.65%	61%	54%	65%	
On 5th day						

Case	49.7%	13.45%	49.5%	21%	70%	<.0001
Control	62%	2.57%	62%	54%	66%	

Aggregation percentage in case within 24 hour ranged from 24-76% while in control group ranged from 55-67%. The mean aggregation percentage in the case group was found to be 52.5± 13.74% while that in the control group came out to be 61.3± 3.34% and it was found to be statistically significant (p value <0.05).

Aggregation percentage in case on 3rd day ranged from 22-70% where as in control group ranged from 54-65%. The mean aggregation percentage in the case group was found to be 49.6 ± 12.14% while that in the control group came out to be 61.1 ± 2.65% and it was found to be statistically significant.

Aggregation percentage in case on 5th day ranged from 21-70 % while on the other hand in control group ranged from 54-66%. The mean aggregation percentage in the case group was found to be 49.7 ± 13.45% while that in the control group came out to be 62 ± 2.57% and it was found to be statistically significant (p value <0.05).

Comparison of the platelet aggregation after the addition of ADP in both case and control groups was done and the mean platelet aggregation values were calculated. The mean aggregation percentage in the case group was found to be 64.6± 13.23% while that in the control group came out to be 71.3± 4.18% and it was found to be statistically insignificant (p value >0.05). Aggregation percentage in case on 3rd day ranged from 36-78% while in sample from control group ranged from 65-78%. The mean aggregation percentage in the case group was found to be 61.4 ± 10.13% while that in the control group came out to be 71.6 ± 3.52% and it was found to be statistically significant (p value <0.05).

Aggregation percentage in case on 5th day ranged from 34-82% while in control group it was observed to be ranging from 63-78%. The mean aggregation percentage in the case group was found to be 61.6 ± 12.23% while that in the control group came out to be 71.9 ±3.85% and it was found to be statistically significant.

Discussion

While using ADP as agonist, the mean value of platelet aggregation within 24 hours, on day 3rd, on day 5th in thermal burns patient was52.5± 13.7%, 49.6± 12.1%, 49.7± 13.4% respectively. Whereas in control group blood sample collected on corresponding days showed value at 61.3± 3.3%, 61.1± 2.6%, 62± 2.6% respectively. When this in vitro platelet aggregation study was done with the help of collagen as an agonist in normal healthy volunteer i.e. the control group, it was found that the mean value of platelet aggregation on corresponding days was 71.3 ± 4.2%, 71.6 ± 3.5%, 71.9 ± 3.8% respectively. The mean value for the platelet aggregation within 24 hours, on day 3rd, on day 5th with collagen as agonist in burns patient was 64.4 ± 13.2%, 61.4 ± 10.1%, 61.6 ± 12.2%.

Cowan DH et al[2]in their study on platelet aggregation as a sign of septicemia in thermal injury found that the maximal extent of platelet aggregation in response to adenosine diphosphate, epinephrine, and collagen tended to be subnormal, but the differences were not significant owing to the variation in results.

Another study by Yaguchi A et al [9] showed that platelet aggregation was more severely altered in patients with severe sepsis than in those with uncomplicated sepsis. In Woth G et al [10] to evaluated platelet aggregation in septic patients compared to healthy controls revealed a significant deterioration in the inducible aggregation among septic patients.

The ADP induced platelet aggregation was significantly deteriorated in patients with low platelet count in all 5 days (P<0.05) while adrenaline caused aggregations were lower in the 2nd, 3rd, 4th and 5th consecutive day (P<0.05). Collagen induced aggregation was significantly lower on the 1st, 2nd, 3rd days following admission (P<0.05). There was no difference between the two groups based on the saline aggregation.

A study to assess platelet function after trauma by Kutcher ME et al[11] observed below-normal platelet response to at least one agonist on admission, and 91.1% patients had platelet hypofunction at some time during their ICU stay.

we also studied the correlation between platelet aggregation and severity of burns in absence of sepsis and observed that a significant difference was present in both ADP and Collagen induced platelet aggregation at all-time points within group A, B, C (according to severity of burns). Within 24 hours, in group A, B, C mean value of platelet aggregation with ADP and Collagen was 64.9%, 52.2%, 39.2% and 77.7%, 63.1%, 51.8% respectively.

Similar findings were observed on day 3rd and day 5th.In group B and C, there was further reduction in platelet aggregation on day 3rd compared to within 24hour, as well as on day 5th compared to day 3rd. In contrast, in group A platelet aggregation decrease on day 3rd compared to within 24 hours but increase on day 5th compared to day 3rd.

In this study mean value of platelet count was significantly high during first 24 hours in burns pa-

tients (3.07 ± 0.54 lakh/mm3) than sample collected on corresponding day from normal control group (2.58 ± 0.62 lakh/mm3).When the 3rd and 5th day was compared to the within 24hour, a fall in platelet count was observed and it was significant only in burns patients.

This study confirmed previous observations that after thermal burns a temporary period of thrombocytosis is most likely caused by a reactive response to the thermal injury or a rebound effect of the bone marrow to its increased consumption and destruction[12,13].

In our study a significant difference was obtained in platelet count at all-time points observed within group A, B,C (according to the severity of burns) respectively ($P < 0.008$).

A Study by Pavic M et al[14]in which burn patients were divided according to severity of burns in groups A (mild burns ≤ 10% TBSA) and B (moderate/severe burns > 10% TBSA) and platelet count was observed on days 1, 4, 7, 14, 21, 28 after burn injury.

Peak platelet counts were recorded on the 14th day in group B (492 x 109/L), and on the 21stday in group A (536 x 109/L).

Conclusion

Thermal burn injury causes severe abnormality of ADP and collagen induced platelet aggregation in acute phase. This abnormalities of platelet aggregation correlate consistently with severity of burn in form of increased TBSA. It is suggested that the use of serial determinations of platelet aggregation in thermal burn patients can be a useful adjunct in the early detection of abnormality in platelet function and may enhance the survival of burn patients through early initiation of supportive treatment strategies.

References

1. Parihar A, Parihar MS, Milner S, Bhat S. Oxidative stress and anti-oxidative mobilization in burn injury. Burns 2008 Feb; 34(1):6-17.
2. Cowan DH, Bowman LS, Fratianne RB, Ahmed F. Platelet aggregation as a sign of septicemia in thermal injury. JAMA 1976; 235:1230-4.
3. Garcia AA, Lorente JA, Cesar PJ, Garcia LJ, Alvarado R, Arevalo JM. Degree of hypercoagulability and hyperfibrinolysis is related to organ failure and prognosis after burn trauma. Thromb Res 1998; 89:59-64.
4. Kao CC, Garner WL. Acute burns. Plast Reconstr Surg 2000; 105:2482-92.
5. Hettiaaratchy S, Dziewulski P. Pathophysiology and types of burns. BMJ 2004; 328:1427-9.
6. Baughman RP, Lower EE, Flessa HC. Thrombocytopenia in the intensive care unit. Chest 1993; 104(4):1243-7.
7. Housinger TA, Brinkerhoff C, Warden GO. The relationship between platelet count, sepsis, and survival in paediatric burn patients. Arch Surg 1993; 128(1):65-6.
8. Levi M, Schultz M. Hematologic failure. Semindsa Respir Crit Care Med 2011; 32(5):651-9.
9. Yaguchi A, Lobo FLM, Vincent JL, Pradier O. Platelet function in sepsis. J Thromb Haemost 2004; 2:2096-102.
10. Woth G, Varga A, Ghosh S, Krupp M, Kiss T, Bogár L, Mühl D. Platelet aggregation in severe sepsis. J Thromb Thrombolysis 2011; 31(1):6-12.
11. Kutcher ME, Redick BJ, McCreery RC, Crane IM, Greenberg MD, Cachola LM, Nelson MF, MPA RN, and Cohen MJ. Characterization of platelet dysfunction after trauma. J Trauma Acute Care Surg 2012; 73(1):13-9.
12. Akca S, Michael HP, De MA, Suter P, Levi M, Vincent JL. Time course of platelet counts in critically ill patients. Crit Care Med 2002; 30(4):753-6.
13. Chang P, Murray DJ, Olson JD, Pennell BJ, Lewis RW, Kealey GP. Analysis of changes in coagulation factors after postoperative blood loss in burn and non-burn patients. Burns 1995; 21(Sept):432-6.
14. Pavic M, Milevoj L. Platelet count monitoring in burn patients. Biochemia Medica 2007; 17(2):212-29.