

Post Electric Shock Reactive Thrombocytosis

RICHA KATIYAR¹, SHASHIKANT C.U. PATNE², PRANJAL PANKAJ³

ABSTRACT

Thrombocytosis is defined as a platelet count greater than $400 \times 10^9/L$. Electric shock may lead to tissue injury and marked thrombocytosis. We herein report a case of 45-year-old woman, who was hospitalized with progressively increasing weakness, tingling sensation, and numbness of the bilateral lower limbs. She had a history of receiving electric shock during household work, 15 days prior to her admission. Her laboratory investigations revealed a markedly increased platelet count ($1,570 \times 10^9/L$) along with increased level of serum uric acid (12 mg/dL), and mild increase in serum potassium (6.7 mmol/L), and serum alanine transaminase (50 U/L). She was treated with intravenous fluids, calcium, and multivitamins. Following hospitalization and treatment, condition of the patient improved, her symptoms resolved, and her platelet counts declined. We report this case because of rare presentation of marked thrombocytosis secondary to electric shock with a brief discussion on pathophysiology of this condition.

Keywords: Fluid therapy, Neuropathy, Platelets, Tissue injury

CASE REPORT

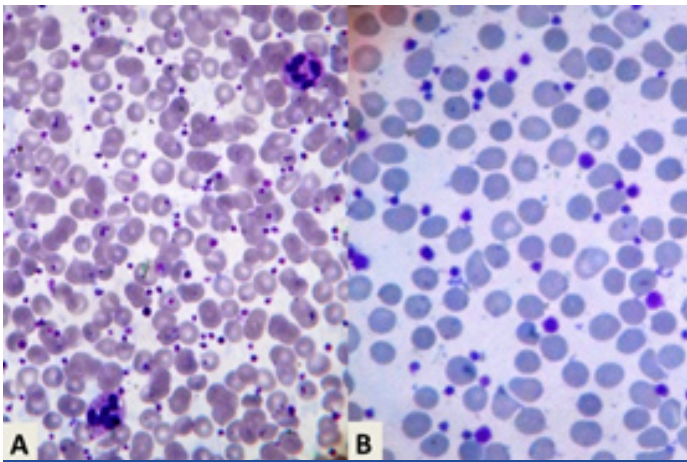
A 45-year-old woman presented in the emergency outpatient department with complaint of progressively increasing weakness, tingling sensation, and numbness of the bilateral lower limbs for last 15 days. Patient's past medical and drug histories were insignificant. There was a history of receiving mild electric shock while doing household work 15 days ago. Patient did not take medication or seek medical advice after the electric shock. Instead, she took rest and home remedies like massage with mustard seed oil to alleviate her symptoms. However, her complaint progressively deteriorated over a period of 15 days.

At the time of hospitalization, her pulse rate was 90/minute, blood pressure 110/70 mm of Hg, temperature 98.6°F and respiratory rate 18/min. The hematological and biochemical investigations at the time of hospitalization are given in [Table/Fig-1]. Serology for HIV and HBsAg were non-reactive. Ultrasound examination of the abdomen was unremarkable, except for an incidentally detected four mm sized gall bladder stone, which was revealed to the patient and her attendants. Peripheral smear showed normocytic normochromic red blood cells, and marked thrombocytosis [Table/Fig-2a]. There were no parasites or blast cells seen in the slides examined. Bone marrow examination showed normocellular marrow for the age, the myeloid: erythroid ratio 1:1.3, normoblastic erythropoiesis $400 \times 10^9/L$ and normally distributed myelopoiesis. Megakaryocytes were markedly increased in numbers with normal morphology. There were no suggestions of any myeloproliferative disorder or hemoparasite. With hematological diagnosis of reactive thrombocytosis and leucocytosis, patient was advised a close follow up.

Patient was treated with intravenous normal saline, intravenous ringer lactate, injectable calcium, and multivitamins for one week. On second day of admission, her platelet count was $1,530 \times 10^9/L$. One day before discharge, her hematological investigations were done, which are given in [Table/Fig-1]. After one week, patient improved symptomatically, her platelet count declined to $1,200 \times 10^9/L$ [Table/Fig-2b], and she was discharged in a stable condition. Patient was advised for regular follow up. However, she did not return in follow up and further investigations could not be done.

Laboratory Investigations	On Admission	On Discharge
Hematological		
Hemoglobin	12.8 g/dl	11.6 g/dl
Total leukocyte count	14000 /mm ³	9000 /mm ³
Platelet count	$1,570 \times 10^9/L$	$1,200 \times 10^9/L$
Red blood cell count	4.1 million/ mm ³	3.8 million/ mm ³
Packed cell volume	40 %	37 %
Mean corpuscular volume	97 fl	99 fl
Mean corpuscular hemoglobin	30 pg	30 pg
Mean corpuscular hemoglobin concentration	31 g/dl	30 g/dl
Red cell distribution width	14 %	14 %
Neutrophils	62 %	69 %
Lymphocytes	27 %	20 %
Monocytes	5 %	3 %
Eosinophils	6 %	8 %
Basophils	0 %	0 %
Immature cells	0 %	0 %
Erythrocyte sedimentation rate	32 mm at 1 hour	--
Biochemical		
Serum urea	28 mg/dl	--
Blood urea nitrogen	13 mg/dl	--
Serum creatinine	0.7 mg/dl	--
Serum uric acid	12 mg/dl	--
Serum sodium	135 mmol/l	--
Serum potassium	6.7 mmol/l	--
Total bilirubin	0.3 mg/dl	--
Direct bilirubin	0.2 mg/dl	--
Serum protein	7.7 g/dl	--
Serum albumin	4.3 g/dl	--
Serum alanine transaminase	50 U/l	--
Serum aspartate transaminase	34 U/l	--
Serum alkaline phosphatase	103 U/l	--
Random blood sugar	97 mg/dl	--

[Table/Fig-1]: Comparative table showing laboratory investigations of the patient at the time hospital admission and at the time of discharge



[Table/Fig-2]: (a) Peripheral smear at the time of hospitalization showing increased number of platelets, normochromic normocytic red cells and two neutrophils (Leishman stain, 400x) (b) Peripheral smear at the time of discharge, showing a decrease in platelet number (Leishman stain, 400x)

DISCUSSION

In the adults, thrombocytosis is defined as a platelet count greater than $400 \times 10^9/L$ (normal range: $150-400 \times 10^9/L$) [1,2]. Although accepted range of platelet count in normal adults is 150 to $400 \times 10^9/L$, upper limit of platelet count differs among various laboratories from 350 to $450 \times 10^9/L$ [2,3]. Nevertheless, a platelet count greater than $400 \times 10^9/L$ deserves further investigations to rule out neoplastic condition of essential thrombocytosis. Based on etiology, thrombocytosis is of two types- i) primary/essential and ii) secondary/reactive. Primary/essential thrombocytosis is seen in myeloproliferative disorders, myelodysplastic disorders, or rarely in hereditary conditions [4]. On the other hand, secondary/reactive thrombocytosis is a self-limiting normal physiologic response due to inflammatory conditions (e.g., infection, chronic inflammatory conditions), trauma, tissue damage, or surgery [1]. Electric shock injuries result in tissue damage due to direct effects of current on cell membrane and vascular smooth muscle [5]. It is proposed that overproduction of thrombopoietic factors because of tissue damage, inflammatory processes, etc. is responsible for reactive thrombocytosis. These factors, especially interleukin-6, act on megakaryocytes and their precursors and cause increased platelet production [1]. With resolution of the underlying condition, reactive thrombocytosis generally settles down. Therefore, it is important to distinguish between primary and secondary thrombocytosis as primary thrombocytosis is associated with a significantly increased

risk for thrombotic or hemorrhagic complications [6]. In contrast, less than 1% patients of reactive thrombocytosis present with complications [7].

Approximately 120 years ago, first case of electric injury was reported. Today the numbers of cases due to electrical injuries are increasing [8]. However, the majority of patients who suffer from mild electric shock do not seek medical care, so the exact number is not known. Consequently, very few literature is available regarding tissue response after electric shock [5,8]. To the best of our knowledge, there is no published literature on post electric shock reactive thrombocytosis. The symptoms of electric shock injury may be attributed to alternating current (AC) induced damage to the conductive neurovascular bundles of the limbs, or tetanic contraction of the skeletal muscles [5]. Tetanic contraction is accountable for skeletal muscle damage and consequently, hyperkalemia. Along with these factors, we hypothesize that microvascular phenomenon secondary to thrombocytosis might also be responsible for producing symptoms. Prognosis of reactive thrombocytosis secondary to electric shock is good with supportive therapy, as seen in our case.

CONCLUSION

We have reported an unusual case of electric shock injury presented with reactive thrombocytosis and the neuromuscular symptoms. The patient responded well to supportive treatment. Her symptoms improved and platelet count started decreasing at the time of discharge. However, further investigations and follow up in this case was not possible, as the patient did not return in follow up.

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