ISSN- 2230-7346
Journal of Global Trends in Pharmaceutical Sciences

SUBDURAL HAEMORRAHGE DUE TO IDIOPATHIC THROMBOCYTOPENIC PURPURA – A CASE REPORT

Gowri Parvathy S R*, Anjana Sailan, Dr. Anusreeraj R S

Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Neyyatinkara, Thiruvananthapuram

*Corresponding author E-mail: gowrigp20@gmail.com

ARTICLE INFO

Key words:
Bruises, Burr-Hole Evacuation, Idiopathic Thrombocytopenic Purpura, Petechiae, Subdural Haemorrhage.

Access this article online
Website: https://www.jgtps.com/
Quick Response Code:

ABSTRACT

A 22 years old female patient was admitted in the general medicine department, NIMS, Trivandrum, Kerala 2018 with complaints of headache and paralysis on both legs. Patient was previously diagnosed with idiopathic thrombocytopenic purpura (ITP) with evident bruises and petechiae all over body. At present, patient found to have declined platelet count and diagnosed with subdural haemorrhage. Mainly ITP associated with intracranial haemorrhage when the platelet level fall 30,000 cells/mm3. Patient underwent burr-hole evacuation procedure and treated with corticosteroids and seizure prophylaxis medications. On the following days, patient became stable and discharged with oral corticosteroids and other medications.

INTRODUCTION

Idiopathic thrombocytopenic purpura (ITP) is a rare autoimmune bleeding disorder, caused due to decreased platelet count. It is characterised by bleeding, bruises (purpura), red coloured spots (petechiae) which is mainly seen in lower limbs. It can be acute or chronic. Acute ITP, which is the most common form, affects children of age group 2-6 years. ITP is self-limiting for 80% of children. Chronic ITP is the form that has sudden onset at any age. Chronic ITP is most commonly seen in adults. Adolescent females have high incidence rate than male. Intracranial haemorrhage is one of the serious complications that occur in about 1% of patients diagnosed with ITP [1, 2].

PATHOPHYSIOLOGY

The exact cause is unknown. Patients' cells produce autoantibodies against platelet Surface. Patient's body misidentify platelets as foreign substance. Antibody opsonization of platelets occurs in the blood stream and the opsonised platelets are phagocytosed by liver and spleen. Along with this antibody specific platelet precursor (megakaryocytes) suppression occurs in bone marrow. This leads to thrombocytopenia, leading to serious, mucosal bleed depending on the platelet count.

CAUSES

Auto antibody production against platelet by immune system can be due to congenital cause, autoimmune disorders (lupus, rheumatoid arthritis) viral infections such as HIV, Hepatitis. Secondary causes are medications (heparin, quinine), cirrhosis.

SYMPTOMS

- Gingival bleeding, nose bleed

- Bruises (<2mm)
- Petechiae (2-10mm)
- Ecchymosis (>10mm)
- Menorrhagia
- Blood in vomit, urine/stool
- Intracerebral/ subarachnoid haemorrhage, internal bleeding is the serious complication of ITP, which occurs when platelet count is <20,000cells/mm^3.

DIAGNOSIS

- Mucosal bleed, serious bleed, petechiae, purpura, ecchymosis.
- Blood test shows decreased platelet count (below 50,000cells/mm^3).
- Immature platelet in peripheral blood smear.
- Bone marrow biopsy shows increased or normal megakaryocytes.

TREATMENT

First line agents include corticosteroids, intravenous immunoglobulins (IV IgG), IV Rh anti-D. Corticosteroids reduce autoantibodies. Corticosteroids and IV IgG reduces the clearance of opsonised platelets. Emergency therapy includes administration of IV corticosteroids, IV IgG, IV Vincristine, platelet transfusion [3]. Splenectomy is preferred to those whose platelet count remains constantly below 20,000 or at high risk to control bleeding [4].

CASE REPORT

A 22 years old female patient was admitted in the tertiary care hospital with complaints of headache and paralysis on both legs. Patient had history of gingival bleeding, bruises all over the body and tiny red spots on both feet (Fig 1,2). Patient also had past history of menorrhagia and was diagnosed as idiopathic thrombocytopenic purpura (ITP). Past medication history suggest that she was on steroid therapy. She has no family history or social history of smoking /drinking. Also concluded that patient was not under any drugs that could induce ITP. On examination her vitals were found to be temperature [98.6˚F], respiratory rate [24 breaths/min], pulse rate [86 beats/min] and BP [130/70 mmHg]. On physical examination patient was found to be conscious and oriented. Through investigation it was found that patient's platelet count was declined. Her CRP was found to be elevated [81.8 mg/dL] and ESR was exalted than normal [55 mm/hr]. Haemoglobin was dropped to a value of 7.4 g/dL. Renal function test and liver function test was normal. On taking CT brain, impression was subdural haemorrhage (Fig 3). It was diagnosed as ITP induced subdural haemorrhage. Patient underwent burr-hole evacuation procedure. She was given 6-units of single donor platelet (SDP) and 3-unit platelet rich plasma (PRP) to elevate the platelet levels. She was treated with Inj. Mannitol [100mg, BD], Inj. Methylprednisolone [40mg, BD], Inj. Levetiracetam [500mg, TDS], Tab. Phenytoin [100mg, BD], Tab. Clobazam [5mg, 0-0-1]. Her platelet count was continually monitored. On discharge, she was advised to take Prednisolone [40 mg, OD], Clobazam [5mg, OD], Phenytoin [100mg, TDS], Levetiracetam [500mg, TDS].

Table 1: Platelet count of patient during the course at hospital

Day	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Platelet count [lakh cells/mm3]	15000	18000	18000	18000	35000	40000

Fig 1 : Bruises over arms

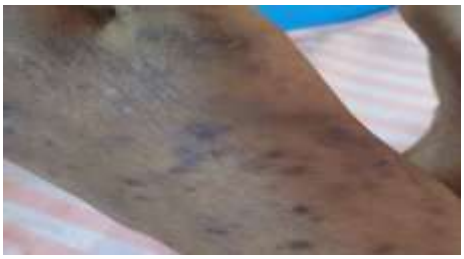

Fig 2: Tiny red spots on feet (petechiae)

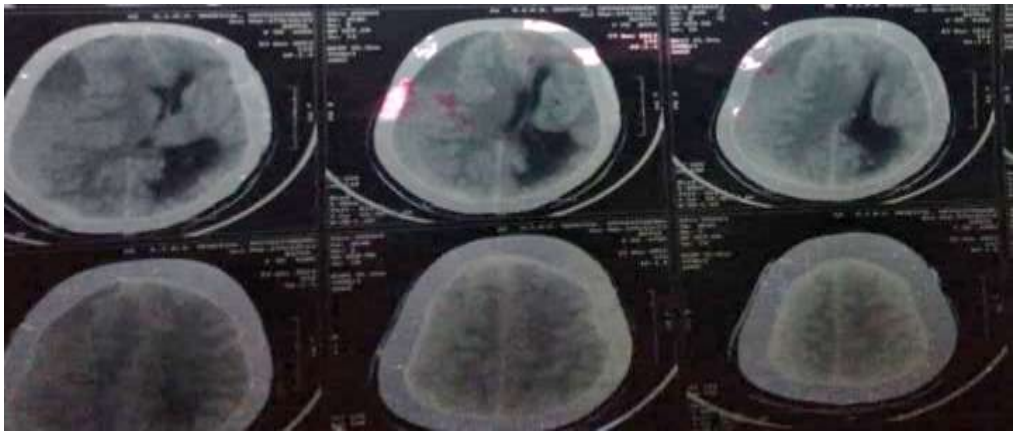

Fig 3: MRI showing subdural haemorrhage

DISCUSSION

Subdural haemorrhage is a detrimental complication which occurs when platelet levels fall below 30,000 cells/mm^3[3,5,6]. Platelet count for patients with ITP should be maintained at haemostatic level i.e., ≥30,000cells/mm^3[5]. In this patient, platelet level falls off to 15,000cells/mm^3, which increased the risk of serious bleeding. She was observed serially and her platelet count raised to 40,000 cells/mm^3 at the time of discharge. Her symptoms gradually resolved. Although bleeding risk depend on the platelet count, the enlargement rate of hematoma does not depend on it [7].

REFERENCES

1. Patnaik A, Mishra SS, Senapati SB, Pattajoshi AS. Management of chronic subdural haematoma in a case of idiopathic thrombocytopenic purpura. *J Surg Tech Case Rep*. 2012; 4(2):132-134.
2. Sebe, Ohshima, A case of chronic subdural hematoma associated with idiopathic thrombocytopenic purpura (ITP) *No Shinkei Geka*. 1990; 18:761–5.
3. Sreedharan, Subdural haematoma with spontaneous resolution--rare manifestation of idiopathic thrombocytopenic purpura. J Assoc Physicians India 2000; 48:432-4.
4. Cines, How I treat idiopathic thrombocytopenic purpura (ITP). *Blood*. 2005; 106(7):2244-51.
5. George.Idiopathic thrombocytopenic purpura in adults: current issues for pathogenesis, diagnosis and management. Hematol J. 2004;5:12-14
6. Cortelazzo, High risk of severe bleeding in aged patients with chronic idiopathic thrombocytopenic purpura. Blood. 1991; 77:31-33.
7. McMillan R, Bowditch RD, Tani P, Anderson H, Goodnight S. A non-thrombocytopenic bleeding disorder due to an IgG4-kappa anti-GPIIb/IIIa autoantibody. Br J Haematol. 1996; 95:747-49.