INTERNATIONAL JOURNAL OF ADVANCES IN CASE REPORTS

ISSN - 2349 - 8005

www.mcmed.us/journal/ijacr

Case Report

IMMUNE THROMBOCYTOPENIA PRESENTING WITH SUBARACHNOID HAEMORRHAGE

Nalla Monica, Tharika Shraddha R, Pradeep M. Venkategowda*, Himaal Dev

Department of Critical Care Medicine, Apollo Hospital, Sheshadripuram, Bengaluru, Karnataka, India. 560020.

ABSTRACT

Thrombocytopenia secondary to Immune thrombocytopenia can cause bleeding anywhere in our body. This is a case report of a 73year old female presented to our hospital with complaints of headache and petechiae all over the body. Routine blood investigation showed platelets of 3000 cells/cumm of blood and CT brain revealed right tentorial bleed and right SAH. She was diagnosed to have ITP related intracranial bleed and hence managed successfully with single donor platelets (SDP), injection methylprednisolone and Intravenous immunoglobulin (IvIg). Platelets count improved to 10,000 on 6th day and to more than 1 lakh on day 10. Patient condition improved without further worsening of intracranial bleed. This case report highlights the rare and devastating complication of ITP and successful management of it with steroids and IVIG.

Key words: Thrombocytopenia, Subarachnoid haemorrhage, Bleeding, Petechiae.

Access this article online	
Home page: http://www.mcmed.us/journal/ijacr DOI: http://dx.doi.org/10.21276/ijacr.2020.7.2.1	**Quick Response code**

Received:25.12.2019	**Revised**:12.03.20	**Accepted**:15.05.20

INTRODUCTION

Immune thrombocytopenia (ITP) is an autoimmune disease in which patient can present with mild skin bruises to life threatening intracranial haemorrhage due to very low platelet counts. It was first described in mid-1500s in a boy with dark macules and bloody discharges. Later the German physician Paul Gottlieb Werlhof named it as Morbus Maculosus Hemorrhagicus. Finally, it got changed to M. Maculosus and now as immune thrombocytopenia [1].

Case report

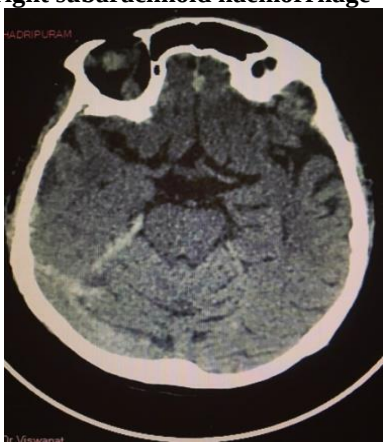
A 73years old female presented to our hospital with complaints of headache (sub occipital region) since 10 days. She was a known case of type 2 diabetes mellitus, hypertension, hypothyroidism and ischemic heart disease on treatment. She was initially evaluated in emergency. On examination she was conscious, oriented, afebrile with pulse rate of 78bpm and blood pressure of 134/74mmhg. Systemic examination revealed petechiae all over the body. Routine blood investigation showed haemoglobin of 9.2 g/dl, total leucocyte counts of 10400 cells/cumm of blood and platelets of 3000 cells/cumm of blood. Renal function tests, liver function tests and serum electrolytes were within normal limits. In view of severe headache CT brain was done which revealed right tentorial bleed and right SAH (Image-1). Haematologist opinion was sought and advise followed. She was admitted in ICU and she received 2 units of single donor platelets (SDP). Peripheral smear showed normocytic normochromic anaemia with thrombocytopenia. Bone marrow aspiration was done which revealed normocellular marrow with trilineage hematopoietic precursors and normoblastic maturation. She was immediately started on injection methylprednisolone 500mg once daily for 3 days. Since the platelet count didn't improve (3000 cells/cu mm) in spite of SDP transfusion and intravenous steroids, she was started on Intravenous immunoglobulin (IvIg) on day 5 of admission

Corresponding Author

Pradeep M. Venkategowda
Email:- drpradeepmarur@gmail.com

(1g/kg body weight). Platelet count improved to 10,000 on 6th day and subsequently it raised to 27000, 47000 and 77000 cells/cumm of blood. Patient was shifted to wards on 7th day and discharged on day10 with platelet count of more than 1 lakh with oral medication of tablet Prednisolone (10mg once daily).

Image-1: CT scan of the brain showing Right tentorial bleed and right subarachnoid haemorrhage



DISCUSSION

Platelets are also known as thrombocytes, are necessary for haemostasis [2], atherosclerosis, angiogenesis, tumorigenesis and antimicrobial host defence. In healthy individual the normal platelet count is around 150-450 X 10^9 / L of blood. They are produced in bone marrow and eliminated within 5-9 days by kupffer cells in the liver and spleen through phagocytosis. Thrombocytopenia is defined as platelet count in peripheral blood < 100 X 10^9 / L [3]. Immune or idiopathic thrombocytopenia (ITP) is an autoimmune disorder, having a platelet count of < 150 X 10^9 / L. In 50-60% of cases it is preceded by viral illness. ITP occurs due to immunological destruction (premature destruction of antibody coated platelets in the spleen) or decreased platelet production.

The pathophysiology is not well understood, it is proposed to be due to loss of immune tolerance by inflammatory T-cells, antiplatelet antibody production by the autoreactive B-cells [4] and destruction of the antibody coated platelets by cytotoxic T-cells or macrophages. Clinical features include- skin bruises, petechiae, purpura, hematoma, and bleeding from nose, gums and stools. The intracerebral bleeding occurs in 0.5% of children's and 1.5% of adults having ITP [5]. The study by Lacey et al [6] showed that the major bleeding occurred in patients with platelet count of < 10 X 10^9/L. Based on etiology, it is termed as primary ITP (the cause is not known - diagnosis of exclusion) and secondary ITP (due to autoimmune disease, viral infections, drugs and vaccinations). Based on duration of thrombocytopenia it is classified as acute ITP (In 80-90% of cases and thrombocytopenia resolves within 6 months) and chronic ITP (10-20% of cases where there is no remission within 6 months).

Diagnosis is based on clinical features and laboratory investigations. Routine blood investigation shows thrombocytopenia with normal white blood cell count, differential count and haemoglobin. Bone marrow examination is required in case of abnormal haemoglobin and white blood cells count and unexplained lymphadenopathy and hepatosplenomegaly. In case of ITP the bone marrow shows increased megakaryocytes with normal granulocytic and erythrocytic series. Only 40-60% of patients have antiplatelet antibodies [7]. It is a self-limiting disease and in 80% of cases it resolves spontaneously or with medications within 6 months. Platelet transfusion is recommended to keep more than 50 X 10^9/L when thrombocytopenia is associated with life or organ dysfunction. Medical management is with corticosteroids, intravenous immunoglobulin (IVIG) or Anti-D which are considered as first line drugs. If no response or chronic ITP then splenectomy, rituximab or thrombopoietin receptor agonists are considered.

The American society of haematology (ASH) guidelines [8] for ITP 2019 recommends hospital admission only when platelet count is < 20 X 10^9/L and Corticosteroid [Prednisone (0.5-2 mg/kg/day) or dexamethasone (40mg/day for 4 days)] is recommended when platelet count of < 30 X 10^9/L and who are asymptomatic or having minor mucocutaneous bleeding. Just observation is required if platelet count is > 30 X 10^9/L. If prednisolone is used, then it is recommended for a period of less than 6 weeks. If patient is dependent or unresponsive to steroids for more than 3 months, then other options includes rituximab, splenectomy or thrombopoietin receptor agonists. Rituximab is advised when patient wishes to avoid long term medications. When thrombocytopenia persists beyond 12 months and not responsive to initial medical management then splenectomy is considered as a definitive therapy [9]. Emergency splenectomy should be considered for patients with refractory thrombocytopenia and life threatening bleeding.

Acknowledgements

We acknowledge management of the hospital for their valuable support.

Declaration of Interest

None declared.

REFERENCES

1. Rodeghiero F, Stasi R, Bernheimer T, Terry Gernsheimer, Marc Michel, Drew Provan, Donald M. Arnold, James B. Bussel et al. (2009). Standardization of terminology, definitions and outcome criteria in immune thrombocytopenic purpura of adults and children: report from an international working group. *Blood*, 113, 2386-2393.
2. Ghoshal K, Bhattacharyya M. (2014). Overview of platelet physiology: its hemostatic and nonhemostatic role in disease pathogenesis. *Scientific World Journal*, 2014, 781-857.
3. Provan D, Stasi R, Newland AC, Blanchette VS, Bolton-Maggs P, Bussel JB et al. (2010). International consensus report on the investigation and management of primary immune thrombocytopenia. *Blood*, 115, 168-186.
4. Audia S, Rossato M, Santegoets K, Spijkers S, Wichers C, Bekker C et al. (2014). Splenic TFH expansion participates in B-cell differentiation and antiplatelet-antibody production during immune thrombocytopenia. *Blood*, 2014, 124, 2858-2866.
5. Kuhne T, Berchtold W, Michaels LA, Runhui Wu, Hugo Donato, Bibiana Espina et al. (2011). Newly diagnosed immune thrombocytopenia in children and adults: a comparative prospective observational registry of the Intercontinental Cooperative Immune Thrombocytopenia Study Group. *Haematologica*, 96, 1831-1837.
6. Lacey JV, Penner JA. (1977). Management of idiopathic thrombocytopenic purpura in the adult. *Semin Thromb Hemost*, 1977, 3, 160-174.
7. Warner MN, Moore JC, Warkentin TE, Santos AV, Kelton JG. (1999). A prospective study of protein-specific assays used to investigate idiopathic thrombocytopenic purpura. *Br J Haematol*, 104, 442-447.
8. Cindy neunert, Deirdra r. terrell, Donald m. arnold, George buchanan, Douglas b. cines et al. (2019). *American society of hematology 2019 guidelines for immune thrombocytopenia*, 3, number 23, 3829-3866.
9. Abadi U, Yarchovsky-Dolberg O, Ellis MH. (2015). Immune thrombocytopenia: recent progress in pathophysiology and treatment. *Clin Appl Thromb Hemost*, 21, 397-404.

Cite this article:
Nalla Monica, Tharika Shraddha R, Pradeep M. Venkategowda, Himaal Dev. Immune Thrombocytopenia Presenting With Subarachnoid Haemorrhage. *International Journal Of Advances In Case Reports*, 7(2), 2020, 15-17.
DOI: **http://dx.doi.org/10.21276/ijacr.2020.7.2.1**