VOLUME - 11, ISSUE - 06, JUNE - 2022 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra				
Property Contraction		Original Research Paper	Haematology	
		DRUG INDUCED THROMBOCYTOPENIA (DIT)-A CLINICAL UPDATE		
Parmila Malik		PhD Scholar, Nims College Of Paramedical Technology, Nims Universitysthan, Rajasthan, jaipur		
Dr. Atul Khajuria*		H.O.D. Medical Laboratory Technology, Nims College Of Paramedical Technology, Nims University Rajasthan, jaipur *Corresponding Author		
ABSTRACT The drugs are the medicine used for the management of diseases. When the drugs used for therapeutic purpose, if it is revert the usage then leads to Drug induced thrombocytopenia (DIT), this condition shows				

purpose, if it is revert the usage then leads to Drug induced thrombocytopenia (DIT), this condition shows abrupt onset of disorder demanding immediate action of the health team members. The present article determines the cause, clinical findings, diagnosis and management of DIT.

KEYWORDS:

INTRODUCTION

Drugs are the substance used for the curative, preventive and promotion of the health of the diseased. The drugs on one hand helps in managing the disease and other hand develops a risk of adverse effects to the person. The risk and side effects of the drugs vary according effects of the drugs.¹⁴⁵

The most common side effects are minor and cause little harm, but there are few drugs that can cause adverse effects to the person and can be cause of the new disease or disorder one such is thrombocytopenia and when the decreased platelets caused by drugs it is termed as Drug Induced Thrombocytopenia (DIT), this condition can be vital and can harm the health of the patients^{23,67}

Drug induced thrombocytopenia is common disorder when used drugs which can cause decrease the platelet counts. DIT should be rapidly identified and managed by removing the causative agent, before the complication precipitate.⁸ The complications of DIT are bleeding, thrombosis, bone marrow suppression and increased platelet destruction.

Causes for DIT

DIT is caused due to decreased platelet production and increasing platelet destruction.

- Myelosuppression occurs when drugs like cytotoxic are used in chemotherapy.
- Suppression of megakaryocytes leads to isolated thrombocytopenia like thiazide, ethanol, tolbutamide.
- Increased in platelet destruction, caused due to use of bleomycin drugs.
- Used of beverages, foods and herbal remedies in treatment of certain diseases. $^{\rm 9.10}$

Pathogenesis of DIT

DIT is caused due to drugs used for therapeutic management, the idiosyncratic mediated reaction is the underlying pathology for DIT, where in the drug-dependent antibodies bind firmly to epitopes on platelet surface, this lead relatively weak reactive autoantibodies this affinates the epitopes on platelet surface which are insufficient to bind the normal circulation.^{11,12,14}

Sensitizing drugs contains charged or hydrophobic elements leads to bind both antibodies and platelet surface protein, leads to destructions of platelets.

Mechanism of DIT

DIT is caused due to drug dependent antibodies which bind to platelet and cause destruction. The antibodies bind to glycoprotein, on the cell surface of the platelet in presence of proactive drugs. quinine, quinidine, penicillin/ sulfonamides, NSIADs, anticonvulsions, antirheumatics, oral antidiabetic drugs, rifampicin, ranitidine.

- Hapten induced antibody observed in use of penicillin. Quinidine and anticonvulsion, mechanism is drug forms linkage in between membrane glycoprotein forming compound epitopes which leads to DIT.¹⁵
- Drugs such as tirofiban, eptifibatide, roxifiban comes under type of Ligand mimetic, act by drug reacts with GPIIb/IIIA and induce antibody reactions.
- Drug specific antibody seen in Abicximab usage due to antibody – specific to control GPIIb/ IIIaDrug induced autoantibody is seen in gold salts. Procaine amide.^{16,17}
- Immune complex is caused due to drug react with normal protein, forms as immunogenic complex forming immune complex.

Trigging factors for DIT

- Patients develop frequent DIT should be assessed for use of bevarion.ages such as quinine containing tonic or suspensions.
- Food like pulped sesame seeds, herbal tea, lupines termis beans.
- Drugs such as trimethoprim, sulfamethoxazole, acetaminophen, danazol, efalizumab, cimetidine, carbamazepine, methyldopa, nalidixic acid, vancomycin, hydrochlorothiazide.¹⁸

Diagnosis of DIT

The diagnosis is empirical, look for patients exposed to single dose of drugs leads to DIT.

- The radiolabeled and fluorescein labeled platelet immunofluorescence test.
- Anti-IgG to learn platelet bound immunoglobulin.
- ELISA for immunoassay
- Flow cytometry and immunoprecipitation western blotting
- Detecting specific antibodies test.¹⁰

Clinical findings of DIT

Patients with DIT sudden onset develops bleeding, thrombosis other associated clinical findings such as rashes, small patches, red spots, dizziness, disorientation, confusion, fatigue, loss of alertness, loss of consciousness.

Management of DIT

The DIT is always abrupt onset of thrombocytopenia, having platelets less than 20000/mL with bleeding and loss of consciousness, the initial treatment begins with in 1-2 days, after immediately disconnection of drug therapy.^{20,21}

 $Whole \ blood \ or \ platelet \ transfusion \ to \ manage \ bleeding.$

• Various drugs such as heparin, cinchona alkaloid like

Corticosteroids use to manage the complication.

CONCLUSION

Therapeutic treatment are the backbone of medical management, adverse effect of drugs leads to DIT, manifested by bleeding suddenly, the drug used for management is stopped immediately, watch on food intake and triggering factors. By understanding the course of effect of DIT its management is achievable in effective manner.

Conflict of interest -None to declare Financial Aid -No financial Aid.

REFERENCES

- Eisner EV, Crowell EB. Hydrochlorothiazide-dependent thrombocytopenia 1. due to IgM antibody. JAMA. 1971;215:480-482.
- Aster RH. Drug-induced immune cytopenias. Toxicology. 2005;209:149-153. 2
- 3 Aster RH. Drug-induced immune thrombocytopenia: an overview of pathogenesis. Semin Hematol. 1999;36:2-6.
- Chong BH, Du XP, Berndt MC, Horn S, Chesterman CN. Characterization of 4. the binding domains on platelet glycoproteins Ib-IX and IIb/IIIa complexes for the quinine/quinidine-dependent antibodies. Blood. 1991;77:2190-2199.
- 5 Curtis BR, McFarland JG, Wu GG, Visentin GP, Aster RH. Antibodies in sulfonamide-induced immune thrombocytopenia recognize calciumdependent epitopes on the glycoprotein IIb/IIIa complex. Blood. 1994;84:176-183.
- Gentilini G, Curtis BR, Aster RH. An antibody from a patient with ranitidine-6. induced thrombocytopenia recognizes a site on glycoprotein IX that is a favored target for drug-induced antibodies. Blood. 1998;92:2359–2365.
- Burgess JK, Lopez JA, Gaudry LE, Chong BH. Rifampicin-dependent 7. antibolies bind a similar or identical epitope to glycopotein IX-specific quinine-dependent antibodies. Blood. 2000;95:1988–1992. van den Bemt PM, Meyboom RH, Egberts AC. Drug-induced immune
- 8. thrombocytopenia. Drug Saf. 2004;27:1243-1252.
- George JN, Raskob GE, Shah SR, et al. Drug-induced thrombocytopenia: a 9. systematic review of published case reports. Ann Intern Med. 1998-129-886-890
- 10. Li X, Swisher KK, Vesely SK, George JN. Drug-induced thrombocytopenia: an updated systematic review, 2006. Drug Saf. 2007;30:185-186.
- Brinker AD, Beitz J. Spontaneous reports of thrombocytopenia in association 11. with quinine: clinical attributes and timing related to regulatory action. Am J Hematol. 2002:70:313-317.
- 12. Lee DH, Warkentin TE. Frequency of Heparin-Induced Thrombocytopenia. In: Warkentin TE, Greinacher A, editors. Heparin-Induced Thrombocytopenia. 3. New York: Marcel Dekker; 2003. pp. 107-148.
- 13. Christie DJ, Mullen PC, Aster RH. Fab-mediated binding of drug-dependent antibodies to platelets in quinidine- and quinine-induced thrombocytopenia. J Clin Invest. 1985;75:310-314.
- Batchelor FR, Dewdney JM, Gazzard D. Penicillin allergy: the formation of the 14. penicilloyI determinant. Nature. 1965;206:362–364. Weltzien HU, Padovan E. Molecular features of penicillin allergy. J Invest
- 15. Dermatol. 1998;110:203-206.
- Weltzien HU, Moulon C, Martin S, Padovan E, Hartmann U, Kohler J. T cell 16. immune responses to haptens. Structural models for allergic and autoimmune reactions. Toxicology. 1996;107:141-151.
- Garratty G. Immune cytopenia associated with antibiotics. Transfus Med Rev. 1993;7:255–267. 17.
- Murphy MF, Riordan T, Minchinton RM, et al. Demonstration of an immune-18. mediated mechanism of penicillin-induced neutropenia and
- thrombocytopenia. Br J Haematol. 1983;55:155–160. Salamon DJ, Nusbacher J, Stroupe T, Wilson JH, Hanrahan JB. Red cell and platelet-bound IgG penicillin antibodies in a patient with thrombocytopenia. Transfusion. 1984;24:395–398 19.
- Kunicki TJ, Russell N, Nurden AT, Aster RH, Caen JP. Further studies of the 20 human platelet receptor for quinine- and quinine-dependent antibodies. J Immunol. 1981;126:398–402
- Berndt MC, Chong BH, Bull HA, Zola H, Castaldi PA. Molecular 21. characterization of quinine/quinidine drug-dependent antibody platelet interaction using monoclonal antibodies. Blood. 1985;66:1292-1301.