International Journal of Medical and Health Sciences

Journal Home Page: http://www.ijmhs.net ISSN:2277-4505

Case Report

Escitalopram Induced Persistent Thrombocytopenia: A Rare Entity

Puneet Dhamija[1*], Ravi Kant[2]

[1]Assistant Professor, Department of Pharmacology, All India Institute of Medical Sciences, Rishikesh,

[2]Assistant Professor, Department of General Medicine, All India Institute of Medical Sciences, Rishikesh.

ABSTRACT

Escitalopram is a commonly prescribed anti-depressant belonging to selective serotonin reuptake inhibitor class with a good safety profile. Hematological abnormalities with Escitalopram are not common and not usually suspected. This is the case of persistent thrombocytopenia following administration of Escitalopram where no other possible etiology could be identified and management of condition on the basis of known mechanisms of thrombocytopenia did not improve the laboratory abnormality. The application of causality assessment scales to present case of thrombocytopenia assigned it to the category of "possible" association with drug use. Escitalopram can cause persistent thrombocytopenia. The underlying mechanism needs to be explored further.

KEYWORDS: Escitalopram, ADR, Thrombocytopenia

INTRODUCTION

Escitalopram is one of the commonly prescribed selective serotonin reuptake inhibitors for management of a psychiatric disorders. The adverse reaction profile of Escitalopram comprises of generalized symptoms like insomnia, fatigue, anorgasmia etc [1]. Hematological abnormalities like cytopenia have not been described. Evidence is available regarding decreased platelet count with use of anti-depressants but duration of thrombocytopenia is not described [2]. Additionally, mechanism of thrombocytopenia following use of anti-depressants has not been described. This case report describes the evolution of and persistence of thrombocytopenia in a patient of generalized anxiety disorder managed on Tab. Escitalopram.

CASE REPORT

A 45 year old occasionally alcoholic, non-smoker male (Figure 1) reported to Pulmonary Medicine Out-patient Department (OPD) with complaints of non-specific central chest pain and cough with dyspnea. He was diagnosed to be suffering from acid peptic disease and depressive symptoms for which psychiatric consultation was advised. The patient provided history of complementary and alternative medicine (CAM) use. To psychiatrist, he complained of anxiety, irritability, palpitations & heaviness in stomach and was diagnosed to be suffering from anxiety not-otherwise specified. Treatment with Tab. Escitalopram 5 mg once daily and Tab. Clonazepam 0.25 mg twice daily was initiated along with multivitamin supplements and CAM was discontinued.

Follow-up after four weeks revealed some improvement in symptoms and same treatment was continued with increase in dose of Tab. Escitalopram to 10 mg once daily for another four weeks. Routine hemogram revealed platelet count to be 1.3 lakhs/mm^3 with no other cytopenia. All other biochemical and hematological parameters were in normal range. After 12 weeks of start of therapy, platelet count decreased to 69,000/ mm^3 and bone marrow biopsy ordered subsequently showed a hypocellular marrow with normal maturation of myeloid elements. There was no history of any systemic symptoms, illicit drug use, repeated CAM use, any other medical problem or family history of blood dyscrasias. Screens for viral infections were negative for Hepatitis B and C virus. With consistent improvement in anxiety symptoms the Tab. Escitalopram dose was increased to 20 mg once daily while other causes of isolated thrombocytopenia were being ruled out.

16 weeks after start of treatment the platelet count decreased to 56,000/ mm^3. Tab. folic acid and multivitamin capsules were added. Tab. Clonazepam was discontinued and after 20 weeks the count decreased to 48,000/ mm^3. Treatment on

the lines of Immune thrombocytopenic purpura (ITP) with corticosteroids did not improve platelet count. At this point Tab Escitalopram was discontinued. Two, four, eight and twelve weeks after stopping Tab. Escitalopram platelet count was 56,000/mm^3, 65,000/mm^3,42,000/mm^3 and 91000/mm^3respectively. In the absence of any medical illness, non-responsiveness to corticosteroids and negative response to folate or multivitamin supplementation and delayed positive response to de-challenge with Escitalopram, a diagnosis of drug-induced persistent thrombocytopenia was made.

Figure 1: Patient presenting with suspected drug induced persistent thrombocytopenia



CAUSALITY ASSESSMENT

Causality assessment done using Kach's and Lasagna [3] algorithm has been discussed in table 1.

Table 1. Causality assessment for suspected persistent thrombocytopenia with escitalopram using Kach's and Lasagna algorithm

Criteria	**Yes/No**
Temporal Association	Yes
Previous Reports	Yes
De-challenge	Yes
Re-challenge	No
Alternative Explanations	No
Causality Category	**Possible**

DISCUSSION

The case of persistent thrombocytopenia is unusual in many aspects and the finding if supported by other cases can have far reaching consequences in management of depressive episodes. Citalopram and its s-isomer Escitalopram are selective serotonin reuptake inhibitors used commonly in managing depressive episodes. They are among the first line agents for treatment of depression. The use of these agents is associated with a number of side-effects from mild symptomatic ADR like insomnia, fatigue and anorgasmia to life-threatening episodes like QT prolongation and hepatitis.[1]

Thrombocytopenia and idiopathic thrombocytopenic purpura have been reported during post-marketing experience with Escitalopram[1]. Effect of some anti-depressants on platelet counts when used for one month were evaluated in a recently published study [2]. Escitalopram treatment for one month led to a significant decline in platelet count from baseline. Other drugs except bupropion had result in similar direction. Mechanistic evaluation of thrombocytopenia has shown that drugs that have an affinity with surface of platelets can induce immunologic mechanisms leading to thrombocytopenia[4].

In the present case thrombocytopenia persisted long after the drug was stopped. This is unusual in case of agents directly leading to suppression of one or other cell line in bone marrow. Possibly, some long lasting immunologic mechanism is involved where antibodies persisted against megakaryocytes causing decreased maturation. Lack of response to corticosteroids in usual doses ruled out the possibility of ITP and that due to folic acid supplementation

ruled out the long-term suppression of one metabolic pathway.

CONCLUSION

Drug induced hematological abnormalities are in many cases idiosyncratic reactions which are overlooked during therapeutic monitoring. There is a need to monitor patients for blood counts and suspect drug-induced cytopenia early in absence of any major evidence of underlying medical illness.

REFERENCES

1. Package Insert Tab. Lexapro; US Approval 2002; Revised July 2014. Available at http://www.accessdata.fda.gov/drugsatfda_docs/label/2009/021323s032,021365s023lbl.pdf. Accessed on 30/12/2015
2. Hoo Rim Song, MD, Young-Eun Jung, MD, Hee-Ryung Wang, MD Platelet count alterations associated with escitalopram, venlafaxine and bupropion in depressive patients *Psychiatry and Clinical Neurosciences* 2012; 66: 457–459
3. Karch FE, Lasagna L. Adverse drug reactions. A Critical Review.JAMA.1975;234:1236–41.
4. Greinacher A, Eichler P, Lubenow N, Kiefel V. Drug-induced and drug-dependent immune thrombocytopenias. *Rev. Clin. Exp. Hematol.* 2001; 5: 166–200.

*Corresponding author: Dr. Puneet Dhamija
E-Mail: drpdhamija@gmail.com