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RESEARCH ARTICLE

HEPARIN INDUCED THROMBOCYTOPENIA AND THROMBOSIS SYNDROME (HITTS)

***Dr. Sarvajeet Khare, Nishi Sheth and Dr. Zeeshan Ali**

Department of Pharmacy, Sumandeep Vidyapeeth, Vadodara, Gujarat, 391760

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**Corresponding author:*
Dr. Sarvajeet Khare

ABSTRACT

Heparin induced thrombocytopenia and thrombosis is associated after several days of ongoing heparin treatment. It is an immune-mediated response which leads to life-threatening thrombosis. Thrombocytopenia is caused by presence of antibodies which activate the heparin and bind to platelet factor 4(PF4). HIT is associated with thromboembolic complications in both arteries and venous circulation. Complications such as pulmonary embolism, myocardial infraction, and death due to thrombotic occlusion in heart, lungs and brain. The main diagnostic criteria are to examine the thrombocytopenia during heparin treatment. HITTS mostly occur after 1-2 weeks after the HIT. A complete watch on platelet and laboratory parameters is essential. The treatment options for HITTS are low molecular weight heparin (LMWHs) and antiplatelet agent are provided. The main consequence would be only keeping eye at patient during thrombocytopenia as it leads to thrombosis and providing effective treatment.

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INTRODUCTION

Heparin induced thrombocytopenia and thrombosis is an immune mediated response to heparin which results in thrombosis which is associated with high morbidity and mortality (Murphy, 1998) Heparin is a heterogenous compound and has thrombotic activity. HIT is immunologic drug reaction associated with venous and arterial thrombosis. The peak onset of occurring is 5-8 days from administration of heparin therapy (Alving, 2003). Two forms of HIT are HIT type 1 is non idiosyncratic and nonimmunologic form (Bick, 1999). Thromboembolic complications are result of platelet activation via the heparin dependent IgG antibodies and endothelial cell derived platelet response. It is immune response produced by platelet factor 4 (PE4) bound to heparin which activates platelets and cause thrombin formation. High level of HIT antibodies leads to thrombocytopenia and leads to thromboembolic complications (Greinacher, 2005). The complications associated with it are disseminated intravascular coagulation, pulmonary embolism, cerebral thrombosis, myocardial infraction, venous thrombosis and ischemic injury (Schechter).

It has marked influence when occur in previously treated patients, higher with Iv than subcutaneous administration, heparin flusher (500U/day). Clinical feature of HIT is platelet count is usually between 30,000-60,000 or 50% decrease from baseline value (Bick, 1999). It can lead to arterial and venous thromboembolism due to formation of platelet aggregation, endothelial damage, pre-existing thrombus and intrinsic plateletactivity (Arthur**, 1985**). Itis also thought to occur by Fc receptor mediated plateletaggregation. It has high prevalence in patient with tissue damage, tissue factor exposure, surgery, sepsis or past thrombosis (Baglin, 2001). Patient who had HIT has a circulating HIT antibody for 120 days. They have high risk of forming HITTS if suspected is given heparin administration (Alving, 2003). Clinical events associated with HIT are platelet count fall, thromboembolic complications, skin reaction at the site of heparin injection site, systemic reactions (Greinacher, 2005). The main diagnostic criteria of HITTS are heparin antibody test, platelet count watch is necessary and heparin stoppage is essential (Arthur, 1985). [6]Prevention of HIT related thrombotic complications recommend to start of non-heparin anticoagulation treatment advanced to decrease the effect. For the treatment of HIT rivaroxaban is found to be potential, as it is given orally,

requires no coagulation monitoring. Although it does not have effect on reducing HIT antibody reduction (Linkins *et al*., 2016). Bivalirudin is approved for Hit with percutaneous coronary interventions. Low molecular weight heparins (LMWHs) are most widely used and preferred other than that antiplatelet and plasmapheresis. Systemic thrombolysis with TPA or streptokinase is essential for treatment of venous thromboembolism and arterial thrombosis. Lepirudin and danaparoid are also used in the treatment (Betrosian, 2003). Thromboembolic complications are result of platelet activation via the heparin dependent IgG antibodies and endothelial cell derived platelet response.

CASE REPORT

A 45-year-old male patient with history of sickle cell disease for 10-12 years came with the complaints of chest pain since the day itself which became severe in afternoon, acute abdominal pain and leg pain for 1 day. On patient examination, temperature was normal, pulse: 68 bpm, BP:110/60 mmHg. On examining ultrasonography, abdominal tenderness and palpable splenomegaly was present. The patient was reported with vaso-occlusive crises in sickle cell disease. For the treatment of vaso-occlusive crises heparin was provided with other medications of sickle cell disease. Heparin 25,000 IU + 45 (NS) 1.66cc/hr was given. Heparin was given to reduce vaso-occlusive crises. The patient was continuously provided with heparin up to 5 days until he was diagnosed with thrombocytopenia. The platelet count was found to be as low as 80,000. He was urgently provided with blood for 3 days. He was advised for pulmonary angiography and ECG as a precaution for any thrombosis. Then he was admitted to the ICU ward as the oxygen saturation goes low. As soon as the patient was identified with HIT, heparin was stopped. CT pulmonary angiogram was performed and the person was detected with pulmonary thromboembolism. Rivaroxaban was provided as an anticoagulant to break the clot formation. He was treated with INJ. Febrinyl (paracetamol), INJ. Avil 1 amp IV, INJ.hydrocort (100) IV, INJ. Tramadol 1 amp 8 hourly, INJ. Emset (4g) IV SOS, Cap. Hydroxyurea (500) 1-0-1, Tablet Folic acid (5) 0-1-0, Cap.autrin(325) 1-0-1, and T. Rivaroxaban 1-0-0.

DISCUSSION

A 45-year-old malecame to our multispeciality hospital, patient is a known case of sickle cell disease in the past 12 years back. On diagnosis patient he was found out with vaso-occlusive crises in case of SCD. On examination, patient body was in pain and had acute onset chest pain. On laboratory investigations, hemoglobin was decreased to 8.0. he was treated with heparin to reduce the pain arising from vaso-occlusive crises. The main treatment goal for the sickle cell disease includes alleviationof pain. Heparin is an anticoagulant which also reduces the formation of blood clots. One of the most fatal side effects of heparin is thrombocytopenia. The patient after being treated with heparin for one week was opted for the normal laboratory tests, X-ray and pulmonary angiogram. The condition worsens when thrombocytopenia was observed, platelet count was found to be as low as 50,000 with hemoglobin value of 6.0.Patient was shifted to ICU ward as the oxygen saturation was gradually low. Pulmonary angiogram showed that patient had pulmonary thromboembolism. Mostly the treatment options associated with HITTS includes anticoagulants such as Lepirudin, danaparoid and fondaparinux. Direct oral anticoagulants such as rivaroxaban, apixaban and dabigatran. Treatment options for HIT are antiplatelet agents such as aspirin and clopidogrel are used. Warning sign such as rash, injection site rash or sore, weakness, numbness should be examining from the start. Pulmonary thromboembolism is one such complication caused by HIT. If kept untreated, it can cause life-threatening condition.Patient was advised for pulmonary embolectomy as earlier as possible. The treatment with blood thinner was provided. Rivaroxaban is anticoagulant to prevent blood clots. The patient was given proper counselling and advice about the medication and condition. Effective treatment and proper monitoring are required for the safe and effective treatment.

CONCLUSION

Heparin induced thrombocytopenia and thrombosis syndrome (HITTS) is life threatening condition, if not treated accurately and on timely manner it can be fatal. The recognition of the adverse effect is most challenging. The main consequence would be on the watch of platelets. Cessation of heparin and switching it to other anticoagulants is the only suitable approach for it. Monitoring and effective treatment will cause less potential risk for morbidity and mortality.

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REFERENCES

1. Murphy KD, Galla DH, Vaughn CJ, McCrohan G, Garrisi WJ. 1998. Heparin-induced thrombocytopenia and thrombosis syndrome. *Radiographics*. Jan;18(1):111-20.
2. Alving BM. 2003. How I treat heparin-induced thrombocytopenia and thrombosis. Blood, *The Journal of the American Society of Hematology*. Jan 1;101(1):31-7.
3. Bick RL, Frenkel EP. Clinical aspects of heparin-induced thrombocytopenia and thrombosis and other side effects of heparin therapy. Clinical and Applied Thrombosis/Hemostasis. 1999 Oct;5(1_suppl):S7-15.
4. Greinacher A, Farner B, Kroll H, Kohlmann T, Warkentin TE, Eichler P. Clinical features of heparin-induced thrombocytopenia including risk factors for thrombosis. Thrombhaemost. 2005 Jul 1;94(7):132-5.
5. Schechter AN, Rodgers GP. Sickle cell anemia—basic research reaches the clinic.
6. Arthur CK, Isbister JP, Aspery EM. The heparin induced thrombosis—thrombocytopenia syndrome (HITTS): a review. Pathology. 1985 Jan 1;17(1):82-6.
7. Baglin TP. Heparin induced thrombocytopenia thrombosis (HIT/T) syndrome: diagnosis and treatment. Journal of clinical pathology. 2001 Apr 1;54(4):272-4.
8. Linkins LA, Warkentin TE, Pai M, Shivakumar S, Manji RA, Wells PS, Wu C, Nazi I, Crowther MA. Rivaroxaban for treatment of suspected or confirmed heparin-induced thrombocytopenia study. Journal of Thrombosis and Haemostasis. 2016 Jun;14(6):1206-10.
9. Betrosian AP, Lambroulis G, Kostantonis D, Balla M, Papanikolaou M, Georgiades G, Theodossiades G. Heparin-induced thrombocytopenia with pulmonary embolism and disseminated intravascular coagulation associated with low-molecular-weight heparin. The American journal of the medical sciences. 2003 Jan 1;325(1):45-7.

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