

Heparin-induced thrombocytopenia type II with pulmonary embolism after cesarean section

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Introduction

Heparin-induced thrombocytopenia (HIT) type II is an immunoglobulin-mediated, drug-induced side-effect of heparin treated patients. With an incidence of 1-3%, a mortality of 20% and permanent disability of another 20% it is a clinically relevant disorder. With heparin treatment or prophylaxis frequent platelet count monitoring is necessary. Thus, with heparin-induced thrombocytopenia type II the thrombocytopenia is often a harbinger of thromboembolic complications in the venous or arterial system [1, 2].

Case Report

A 36-year-old woman, I gravida, 0 para was admitted to hospital in the 30th week of pregnancy because of severe gestosis with intrauterine growth retardation and pathological Doppler flow. First, the symptoms of gestosis improved under adequate therapy. Eighteen days after admission the CTG revealed deep deceleration of type II. The decision for cesarean section was made. The operation was without any complications. The birth weight of the child was 1100 grams, Apgar 3-7-7 and Nsphart. 7.27. The child had to be intubated and was admitted to the Neonatal Care Unit. Two days after cesarean section the patient developed a severe pulmonary embolism stage III-IV and had to be reanimated. A sternotomy with following pulmonary embolectomy of both pulmonary arteries was performed. The patient recovered well without any problems. Retrospective analysis revealed that under 2x7500 U of unfractionated heparin s.c. preoperative through 18 days the platelet count fell from initial values of 180×10^9 (9) at admission to 87×10^9 (9) the day after cesarean section indicating a possible heparin-induced type II thrombocytopenia. The labor-chemical investigations showed immunoglobulins (Ab) against heparin and confirmed the presumption of HIT II. Thus, the anticoagulation was changed to cumarines.

Discussion

The incidence of venous thromboembolism has declined in recent years, possibly due to the successful use of prophylactic strategies. However, pulmonary embolism remains the most common preventable cause of hospital death in industrialized countries. A number of clinical

studies have shown that when used appropriately current antithrombotic therapies, such as unfractionated heparin, oral anticoagulants and low-molecular weight heparins, are effective in preventing thromboembolic events in the majority of patients. However, surveillance data indicate that in certain high risk clinical settings a significant proportion of patients still develop deep vein thrombosis despite use of the most effective prophylaxis methods available. Currently available antithrombotic therapies are also associated with high-risk treatment-specific adverse effects and various practical limitations. Risk of bleeding, drug interactions and heparin-induced thrombocytopenia are the most important safety concerns [3-6].

Heparin induced thrombocytopenia is a well-known complication of heparin administration but usually resolves upon discontinuation without sequelae. However a small proportion of HIT, patients develop thrombosis associated with HIT designated as HITT, which is often life-threatening and may lead to embolisms and gangrene with amputations [2, 5, 7, 8]. HIT II is clinically suspected when the platelet count falls to 50% of the initial values under heparin therapy for 5-20 days. HIT II is caused by IgG-Ab against a multimolecular complex of mostly unfractionated heparin and platelet factor 4. The Fc-RII-receptor (CD31) leads to an activation of the platelets. The risk of thrombosis is increased 27-times. Signs of the existence of IgG-Ab against heparin are redness and necrosis at the injection site of heparin [2].

Existing laboratory methods do not distinguish between HIT and HITT. Jy *et al.* [8] however reported a flow cytometric assay of platelet activation marker CD62P to distinguish the effects of the addition of HIT vs. HITT plasma to normal blood. This method may be clinically useful in the detection of HITT allowing early intervention and preventing catastrophic thrombosis.

The therapeutical management after diagnosis of HIT consists of changing the heparinisation to low-molecular weight heparin (LMWH) [1]. But in vitro studies give evidence for cross reactions in up to 90%. Thus, therapeutic strategies with heparinoids like orgaran® or recombinant hirudin like refludan® are better options because of missing cross-reactions with unfractionated heparin. After normalisation of platelet counts, anticoagulative therapy with cumarine can be induced. This option is not indicated in the first days after diagnosis of HIT because of the initial fast decrease of the inhibitors [1, 3, 4].

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Thus, heparin-induced thrombocytopenia type II is a severe complication of heparin therapy and should be regarded as possible harbinger of thrombosis and embolism. Therefore strict indications for perioperative prophylaxis with the use of low molecular weight heparin for routine prophylaxis should be undertaken.

Conclusion

Heparin-induced thrombocytopenia type II is a severe complication of heparin therapy and should be regarded as a possible harbinger of thrombosis and embolism. Therefore perioperative prophylaxis with the use of low molecular weight heparin for routine prophylaxis, should be strictly followed.

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