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Additional Thrombocytopenia after Cardiac Surgery with Extracorporeal Circulation: A Case Report

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ABSTRACT

Thrombocytopenia occurs almost systematically in cardiac surgery under extracorporeal circulation (ECC). Its usual causes are multiple and recognized, but sometimes uncommon mechanisms are added, posing the problem of etiological diagnosis and the dilemma of optimal adequate management.

The etiological diagnosis of thrombocytopenia after extracorporeal circulation requires a careful analysis of the chronology of the thrombocytopenia and also of the clinical and biological context.

The authors report the observation of a case of additional thrombocytopenia after cardiovascular surgery under extracorporeal circulation, detailing the diagnostic modalities and describing the different usual clinical and biological characteristics of platelet changes induced by extracorporeal circulation.

KEYWORDS: Thrombocytopenia, Extracorporeal Circulation, Cardiac Surgery

INTRODUCTION

Thrombocytopenia after cardiac surgery under extracorporeal circulation (ECC) has multifactorial origin, which poses a problem of etiological diagnosis (Table 1). We report a case of thrombocytopenia in a patient in septic shock.

OBSERVATION

Mrs K·F is 51 years old, diabetic, having undergone double mitral and aortic valve replacement by mechanical prosthesis and a tricuspid plasty. The preoperative biological workup showed a prothrombin rate (PT) of 78% and a moderate prolongation of the activated partial thromboplastin time (APTT) to 45.9 seconds, a fibrinogen level of 3.9 g/l, and a platelet count of 146 G/l. Weaning from the CEC was done by placing an intra-aortic balloon pump (IABP). Heparin in preventive dose was introduced on day 1 postoperatively. On day 2, the patient presented a pneumopathy with severe hypoxemia and hemodynamic instability with a blood pressure (BP) of 88/47 mmHg and a low central venous pressure of + 3 cm of water and oliguria.

The bacteriological study of the protected distal sample and blood cultures allowed the isolation of pseudomonas aeruginosa. The biological workup showed thrombocytopenia at 63,000/mm³, a Hb level of 10 g/l and hyperleukocytosis at 14000/mm³ associated with an inflammatory syndrome with a CRP at 169.3 mg/l and a fibrinogen level of 6.8 g/l. The hemostasis workup revealed a prothrombin level of 56% and a prolongation of the activated partial thromboplastin time (aPTT) of 65 seconds, and a level of D-dimer at 80.5 µg/ml. The diagnosis was septic shock with a pulmonary onset and thrombocytopenia. The patient was put on antibiotic therapy and vasoactive amines. The evolution was marked by the onset of multivisceral failure leading to the patient's death at D30.

DISCUSSION

Thrombocytopenia in postoperative cardiac surgery under extracorporeal circulation (ECC) has multifactorial origin, which poses a problem of etiological diagnosis. In our patient, several etiologies can be discussed, the mechanisms of which may be due either to a decrease in production, or to destruction and/or increased sequestration of platelets. Thrombocytopenia in relation to bypass surgery is transient, appearing from the first minutes of bypass surgery and normalizing in 3 to 4 days postoperatively, resulting from platelet activation leading to the formation of aggregates, which are immobilized secondarily in the extracorporeal circuit [1].

In our case, the use of the intra-aortic balloon pump and the unfractionated heparin (UFH) necessary for its implantation may also be incriminated in the consumption of platelets; according to the literature, a decrease in platelet count >50% has been reported in 26% to 58% of patients treated with IABP [2,3,4]. Another study found a 4.5% incidence of heparin-induced thrombocytopenia (HIT) in patients treated with IABP [5]. There is no critera to distinguish heparin-induced thrombocytopenia (HIT) from thrombocytopenia occurring frequently in a patient with IABP.

The usual evolution of the platelet count after IABP placement is a progressive decrease of about 50% with a maximum decrease between the third and fourth day of placement [5]. In a number of cases, the platelet count rises spontaneously even with IABP [5]. Sometimes the decrease in platelets persists as long as the IABP remains in place [5].

In the presented case, we observed an atypical course of thrombocytopenia, with an abrupt drop from 63 to 18000/mm³, while IABP removal had been performed, was suggestive of another cause for the thrombocytopenia.

In our patient, the presence of disseminated intravascular coagulation (DIC) does not rule out the diagnosis of heparin-induced thrombocytopenia (HIT), as they may be associated in 15 to 20% of cases. The non-resolution of thrombocytopenia after stopping heparin ruled out the diagnosis of HIT and the ELISA test for anti-PF4 antibodies was not performed because non-pathogenic anti-PF4 antibodies frequently exist in a post ECC context, 15 to 50% of patients may have a positive ELISA when there is no HIT [5]. Two different functional tests that could support the diagnosis in our case are the platelet aggregation test and especially the Labeled serotonin release test.

In the absence of an obvious drug-induced or reactive cause, the diagnosis of thrombocytopenia of septic origin was retained.

CONCLUSION

The etiological diagnosis of thrombocytopenia after extracorporeal circulation requires a careful analysis of the chronology of the thrombocytopenia and also of the clinical and biological context.

REFERENCES

1. A. watel.,D.Mathieu., A. Po., P. Dequied., G. Soot, A. Cosson. Modifications hématologiques induites par la circulation extracorporelle. Annales françaises d'anesthésie et de réanimation volume 4, issue 4, 1985, Pages 360-366
2. Vonderheide RH, Thadhani R, Kuter DJ. Association of thrombocytopenia with the use of intra aortic balloon pumps. Am J Med 105: 27-32
3. Roy SK, Howard EW, Panza JA, Cooper HA. Clinical implications of thrombocytopenia among patients undergoing intra-aortic balloon pump counterpulsation in the coronary care unit. Clin Cardiol 2010.33: 30-35
4. F. Stéphan. Thrombopénies en réanimation (2008) 17, 339—347
5. J-C. Rigal. , X. Fournet. , M. Trossaërt. , B. Rozec., M. Treilhaud. , Y. Blanloeil. Contre-pulsion par ballonnet intra-aortique et thrombopénie induite par l'héparine non fractionnée. A. Fd'Anesthésie et de Réanimation 25 (2006) 1149–1152

Table 1: Clinical and biological characteristics of the main etiologies of thrombocytopenia in post-ECC

Thrombocytopenia	Sepsis	HIT	Drugs	IABP
Intensity	Variable	Drop in platelets of more than 50% of the initial value postoperatively	Severe $< 10\ 10^9$ /l	Gradual decrease of about 50% of the initial value after IABP
Appearing	May precede the infectious syndrome by 12 to 48 hours	Typically between the 5th and 10th day if exposed to heparin in the previous 3 months	At least 7 days after exposure (median 14 days)	Maximum drop between 3rd and 4th day of installation
Particularities	- Bleeding: little - Possible thrombosis (purpurafulminans)	- Thrombosis +++ - Rare bleeding	- Bleeding +++	
Mechanisms	● DIC ●Immunological ● MAS	● Anti PF4 antibodies	● Anti-platelet antibodies	● Destruction and/or consumption of platelets on the IABP

Additional tests	● DIC: D-dimer, PT,fibrinogen ● Immunoligic: Platelet coombs, MAIPA test ● MAS: Myelogram	● Elisa test: anti F4P antibody ● Platelet aggregation tests ● Labeled serotonin release test	● Elisa test	