CRITICAL CARE

LETTER

Using anti-platelet therapy to prevent extracorporeal membrane oxygenator thrombosis without heparin resistance and with thrombocytopenia

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Anticoagulation is widely used for preventing membrane oxygenator thrombosis during extracorporeal membrane oxygenation (ECMO) therapy while anti-platelets are rarely used, especially in patients with thrombocytopenia [1]. Here we report a case of successfully using anti-platelet treatment to prevent recurrent extracorporeal membrane oxygenator thrombosis in a fulminant myocarditis (FM) patient.

A 32-year-old man was admitted to our hospital with FM, with 10% left ventricular ejection fraction. After his arrival, he had a sudden onset of ventricular fibrillation and developed cardiac arrest, so cardiopulmonary resuscitation was performed. With high doses of vasoactive medications, he was transferred to the ICU, where venoarterial ECMO was initiated. Continuous infusion of heparin was used for anticoagulation, and activated partial thromboplastin time (aPTT) was titrated up to about 70 seconds. The rotational speed of the pump was about 4,500 rpm, and the blood flow of the pump was 3.9 L/minute. On the second day of ECMO therapy, the membrane oxygenator developed thrombosis, so we replaced it with a new one. Furthermore, we intensified the systemic anticoagulation with a higher aPTT target, about 70 to 90 seconds. However, the membrane oxygenator still developed thrombosis on the fourth day of ECMO initiation, and the oxygenator was replaced again. In addition, there was no clinical evidence of hemolysis or thrombosis at other body sites, and the platelet count decreased to 31,000/mm^3. Thus, we inferred that the membrane oxygenator may have abnormally activated the platelets, resulting in thrombotic clot formation. As a result we combined the anti-platelet treatment (aspirin administered with a first dose of 300 mg and then 100 mg per day) with heparin therapy. After that, oxygenator thrombosis did not occur again, and the platelet count gradually increased to 123,000/mm^3 without bleeding complications. The patient was weaned from ECMO 15 days after his admission. The related data for coagulation and anti-platelet treatment are summarized in Table 1.

Ranucci and colleagues [2] reported that inadequate thrombin suppression by heparin may induce intravascular and extravascular thrombosis, which would result in coagulation factors and platelet consumption, and cause more bleeding and require larger transfusions. However, there was no evidence of insufficient anticoagulation and heparin resistance in our case presented here. Moreover, a higher target of anticoagulation was not effective for preventing oxygenator thrombosis, and the recurrent thrombosis happened only in the oxygenator but not at other sites of the body. Therefore, it seemed that the combination of anti-platelet treatment would be appropriate. However, thrombopenia is another dilemma. In our opinion, the primary cause of oxygenator thrombosis and thrombopenia was abnormal activation of the platelets in the membrane oxygenator, which deserved medical intervention. Lehot and colleagues [3] recently showed that oxygenator thrombosis occurred without heparin resistance in coronary artery bypass graft surgery with normothermic extracorporeal circulation in polycytemia vera. To the best of our knowledge, this is the first case report of the use of anti-platelet therapy to prevent oxygenator thrombosis without heparin resistance and with thrombopenia in FM.

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Table 1 Related data for anticoagulation and anti-platelet therapy during extracorporeal membrane oxygenation

	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11	D12	D13
Plt	202	113	63	36	31	37	39	40	54	66	73	92	123
aPTT	77	68	91	97	65	58	56	61	60	79	77	74	64
Aspirin	-	-	-	300	100	100	100	100	100	100	100	100	100
Event		Ex-*		Ex-*									

D1 is the first day of extracorporeal membrane oxygenation initiation. Plt, platelet count ($\times 10^3/ml^3$); Aspirin, aspirin dose (mg); aPTT, activated partial thromboplastin time (seconds); Ex-*, oxygenator exchanged.

Abbreviations

aPTT: activated partial thromboplastin time; ECMO: extracorporeal membrane oxygenation; FM: fulminant myocarditis.

Competing interests

The authors declare that they have no competing interests.

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Written informed consent was obtained from the patient for publication of his individual details in this manuscript. The consent form is held in the patients' clinical notes and is available for review by the Editor-in-Chief. The authors thank Jing Zhang for her contribution in reviewing the manuscript for spelling and grammar.

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