A Role of Extracorporeal Circuit in the Post-Perfusion Thrombocytopenia: A Scanning Electronmicroscopic Observation

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ABSTRACT: Thrombocytopenia is one of the adverse effects of extracorporeal circulation (ECC) but the mechanism of which has not been fully understood. Blood-gas interface, mechanical agitation, rough surface of extracorporeal circuit and sequestration in the liver have been considered to be a cause of platelet loss. Extracorporeal circuit which provides large artificial surface for contact of blood has been blamed as the site of platelet destruction during oxygenation. However, the part of the oxygenator responsible for platelet loss has not been located. This study was designed to identify the sites of extracorporeal circuit responsible for platelet loss during ECC with scanning electron microscopy (SEM) of the post-perfusion circuit. The accumulation of platelet aggregates was most pronounced at the defoaming net and blood filter where a sudden changes in velocity of blood flow take place. The aggregates were considered to be formed locally at these sites. However, there were no accumulation and/or adherence of platelet aggregates of significant degree at the other parts of the circuit, namely venous and arterial tubings, venous column and arterial reservoir. Platelets seem to be removed from the circulation during each passage by defoaming net and blood filter. However the other parts of the circuit seem to be less blamed for the platelet loss. It was not possible to conclude whether the formation and trapping of platelet microaggregates at the defoaming net and blood filter or the destruction by oxygen bubbles is mainly responsible for the platelet loss during ECC.

KEY WORDS: scanning electron microscopy, extracorporeal circuit, thrombocytopenia, platelet aggregates, fibrin-like strands, microembolism, defoaming net, blood filter.

INTRODUCTION

Diverse effects of extracorporeal circulation (ECC) on the blood elements have been described by many authors. Among various many aspects of blood trauma during and following ECC, thrombocytopenia has been well recognized. However, the mechanism for this phenomenon has not been fully understood. It has been suggested that blood-gas interface of the bubble oxygenator, which is widely used, produces destruction of platelets.

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Recently, it has been recognized that platelet micro-aggregates are formed during ECC\textsuperscript{1,2}, but it is still a matter of controversy whether extracorporeal circuit is a site of aggregation or a trapping site of preformed platelet aggregates. This report is based upon the scanning electron microscopy of the post-perfusion plastic bubble oxygenators to determine the role of circuit in post-perfusion thrombocytopenia and to locate the sites in the circuit responsible for the platelet loss during ECC.

**MATERIALS AND METHODS**

*Extracorporeal circuit* (Fig. 1).

The extracorporeal circuit used for the open heart surgery at the First Department of Surgery, Tokyo Medical and Dental University Hospital, consists of disposable bubble oxygenator and tubings. The circuits used were produced and supplied either by Junken Co. Ltd. Tokyo, Japan, or by Bentley Laboratories, Inc., Irvine, California, USA. They were gas-sterilized and pre-packed. The material which constitutes the major part of the circuit is described as polypropylene (Junken’s circuit) and polycarbonate (Bentley’s circuit) by the companies. The same tubing system (Mera, Senko-Ikakogyo Co. Ltd., Tokyo, Japan) was used in both types of oxygenators. Blood filter using a sheet of square mesh with similar pore size to the regular transfusion filter is inset at the arterial tubing distal to the flexible pump part. Four oxygenators were submitted to the present study which had been used for open heart surgery. Length of perfusion, sites of sampling, and magnitude of thrombocytopenia on each oxygenator are given in Table 1.

*Priming*

Hemodilution was employed routinely, in which about 25 per cent hematocrit was predicted during ECC. Priming volume and solutions are listed below.

1. Total priming volume for adult: 2000ml
2. Blood: Following formula was used to determine necessity of priming
   \[
   \text{Blood} \ (\text{ml}) = 1.92 \times \text{Body weight (kg)} \times (25-\text{hematocrit in per cent}) + 1250
   \]
3. 20 per cent Mannitol: Body weight (kg) \times 5 ml
4. 7 per cent Sodium bicarbonate: 60 ml
5. Lactated Ringer's solution: 2000—(Blood+Mannitol+60) ml
6. Heparin: 100 mg
7. Chlorpromazine: 0.5 mg/kg of body weight
8. 8.5 per cent calcium gluconate: ACD blood/200 ml
9. Antibiotics: Sodium cephalothin, 4 g

*Extracorporeal circuit (Fig. 1) and method of perfusion*

Following completion of priming, pre-circulation was performed until all the bubbles within the arterial line were eliminated, which usually took about 5 minutes. After systemic heparinization, 3 mg/kg, patient was cannulated.

Venous blood was drawn from the superior and inferior vena cavae through right atrium by gravity and drained into the oxygenator. One hundred per cent oxygen was used for oxygenation. Oxygen was also utilized for the transport of oxygenated blood into the arterial reservoir in the Junken’s circuit. Oxygenated blood was pumped into the patient using a roller pump through arterial line, within which a blood filter was inserted for the removal of debris. After ECC was terminated, heparin was neutralized by protamine sulfate, two times of the dosage of heparin used.

*Processings of the plastic samples for SEM*

Following ECC, plastic samples were excised from venous tubing, venous column (Junken’s circuit), defoaming net, arterial reservoir, arterial tubing distal to the pump...