

Autotransfusion management during and after cardiopulmonary bypass alters fibrin degradation and transfusion requirements

INTRODUCTION

Alterations of the coagulation-fibrinolytic profile after retransfusion of cell saver-processed shed mediastinal blood during cardiac surgery have been widely documented. However, less information is available on whether these alterations also persist after perioperative autotransfusion management. The present study was designed to elucidate the impact of autotransfusion management during and after cardiopulmonary bypass (CPB) on intravascular fibrin degradation and postoperative transfusion requirements.

METHODS

Thirty adult patients, subsequently undergoing elective primary coronary bypass grafting, were randomly allocated either to a control group (n=15) or an intervention group (n=15) in which the mediastinal and residual CPB blood was processed by a continuous autotransfusion system before reinfusion. Intravascular fibrin degradation as indicated by D-dimer generation was measured and corrected for hemodilution. In addition, the chest tube drainage and need for homologous blood was monitored.

RESULTS

D-dimer generation increased significantly during CPB in the control group, compared to the intervention group (from 312 to 633 ng/mL in the control group versus 291 to 356 ng/mL in the intervention group, $P=0.001$). In contrast to the processed residual CPB blood, the unprocessed residual blood revealed an unequivocal D-dimer elevation (279 ± 103 ng/mL versus 4131 ± 1063 ng/mL, $P<0.001$). Consequently, in the first post-CPB period the intravascular fibrin degradation was significantly elevated in the

control group compared to the intervention group (1849 ± 299 ng/mL versus 580 ± 79 ng/mL, $P<0.001$). Twenty hours post-operatively no significant difference in D-dimer levels was detected between the groups. However, a significant intra-group D-dimer elevation was noticed in the postoperative period compared to the preoperative values (from 312 to 828 ng/mL in the control group and from 291 to 588 ng/mL in the intervention group, $P<0.01$ for both). This latter observation suggests physiological clot remodeling that occurs as part of healing. Postoperative chest tube drainage was higher in the patients from the control group, which also had the highest postoperative D-dimer levels. Perhaps early postoperative excess bleeding predisposes to increase clot formation and subsequent clot remodeling causing elevated D-dimer levels. Moreover, more patients in the control group received one or more packed red cells concentrate postoperatively.

CONCLUSION

These data clearly indicate that autotransfusion management during and after CPB suppresses the early postoperative fibrin degradation, and may therefore move haemostatic regulation back towards normal.



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