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Thromboelastogram showing an undesirable effect of platelet transfusion on blood coagulability and fibrinolysis in a patient with aplastic anemia

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Introduction

Aplastic anemia with reference to bone marrow failure is characterized by the destruction of the rapidly growing cells normally present in the marrow [1]. Pancytopenia is the most common symptom. Thrombocytopenia could introduce the risk of hemorrhage even with minor trauma or surgery. Preoperative transfusion of platelet concentrate has been regarded as a useful technique in the prevention of surgical bleeding. We describe a prophylactic platelet transfusion to a patient with aplastic anemia that caused an undesirable effect on blood coagulability and fibrinolysis. A thromboelastogram (TEG) was useful to detect the abnormality.

Case report

A 67-year-old, 43-kg woman was scheduled for mesopharyngeal tumor resection. She had been diagnosed as having aplastic anemia 2 years previously but had not been treated. Laboratory findings on admission included hemoglobin concentration, 7.6 $g \cdot dl^{-1}$; hematocrit, 24.9%; white blood cell count, 2.6×10^3 μl^{-1}; and platelet count, 4.2×10^4 μl. Coagulability and fibrinolysis were monitored by TEG perioperatively. The preoperative TEG showed no significant abnormality, even with thrombocytopenia (Fig. 1a). Standard coagulation tests, however, were not within normal ranges (e.g., prothrombin time, 16.0 s). To prevent excess perioperative bleeding, platelet concentrates (55 IU total) were transfused 1, 2, and 3 days prior to the operation.

On the day before the operation, the platelet count increased to 14.3×10^4 μl^{-1} and TEG monitoring indicated hypercoagulability [maximum amplitude (MA), 60 mm] and hyperfibrinolysis [fibrinolytic rate {FR: $(MA\text{-}A_{60})/MA \cdot 100$, A_{60}: amplitude 60 min after reaching MA}: 46.7%] (Fig. 1b). The patient was not given epidural anesthesia because of the risk of epidural hematoma. General anesthesia was induced by propofol (3 $mg \cdot kg^{-1}$) and maintained by continuous infusion of propofol (4–5 $mg \cdot kg^{-1} \cdot h^{-1}$) and intermittent administration of fentanyl (400 μg total) with 33% oxygen. Volatile anesthetics, including nitrous oxide, were not used. A sterile and disposable circle system with a bacterial filter was used to prevent infection. The pharynx and larynx were sterilized by povidone iodine before induction of anesthesia. The induction of anesthesia was uneventful. Thirty-five minutes after surgical incision, hyperfibrinolysis progressed (FR: 64.7%, Fig. 1c). The MA (51 mm) could have been underestimated due to fibrinolysis beginning before the amplitude achieved maximum. About three hours after surgical incision, the amount of bleeding had reached 800 ml and the hemoglobin concentration had decreased to 6.5 $g \cdot dl^{-1}$. Autologous blood was therefore transfused (800 ml) and hemoglobin concentration recovered up to 9.4 $g \cdot dl^{-1}$. The parameters of the TEG were normalized 30 min after blood transfusion (Fig. 1d). The total amount of bleeding was 900 ml. Postoperatively, platelet and red cell counts decreased gradually to the preoperative level.

Discussion

Routine coagulation tests are performed on isolated protein fractions and therefore could not assess the in-

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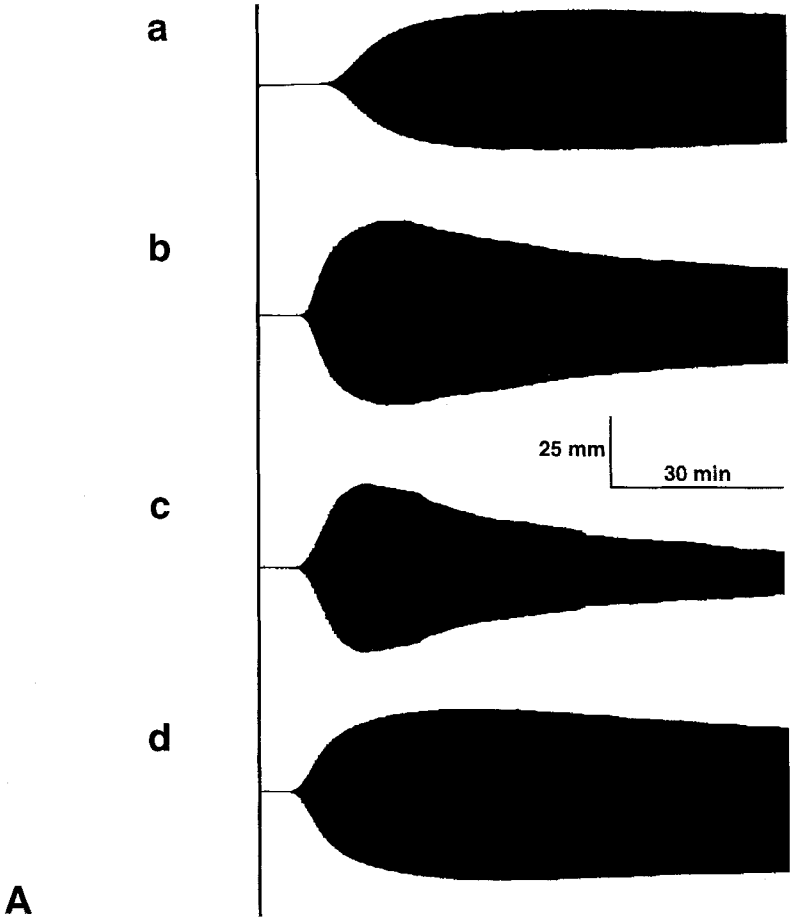

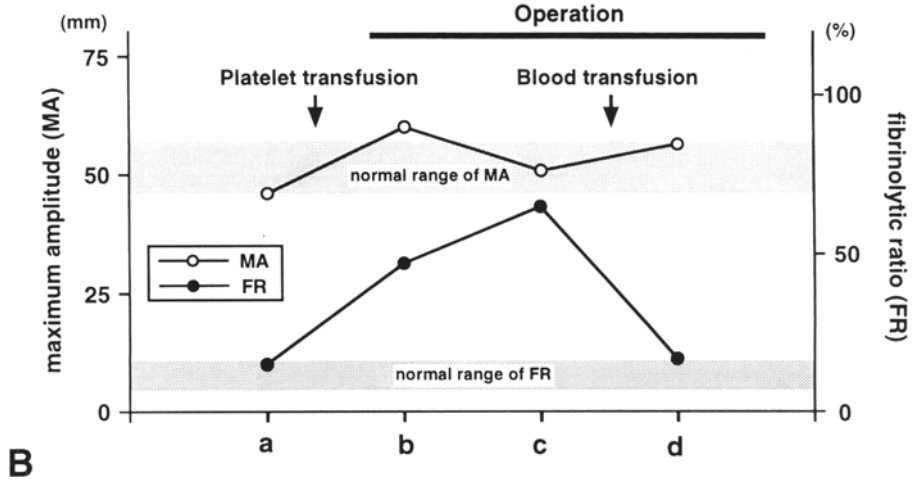

Fig. 1. Perioperative changes in **A** thromboelastographic patterns and **B** maximum amplitude (MA) fibrinolytic ratios (FR) of the patient with aplastic anemia. **a** Before platelet transfusion; **b** after prophylactic platelet transfusion (55 IU total); **c** 35 min after surgical incision; **d** after blood transfusion (800 ml total)

teraction of platelets with the protein cascade. Furthermore, conventional coagulation tests are often not compatible with the dynamics of the surgery, since time is required for data acquisition due to specimen transport and laboratory workload delays. TEG monitoring has advantages in the screening of blood coagulation and fibrinolysis perioperatively because of the simplicity and practicality of the technique [2–5]. The TEG assesses the entire process of the platelet surface interaction with the protein coagulation cascade, fibrin formation, clot retraction, and eventual lysis [6]. In an animal model using controlled levels of the fibrinolytic agent streptokinase, the TEG could detect clot destruction before changes were noted in plasma fibrinogen or plasminogen levels [7]. The TEG has also been used for intraoperative coagulation monitoring during liver transplantation, and it was extremely reliable in patients with hypercoagulability and hyperfibrinolysis [2]. MA archived by TEG tracing indicates the strength of the fibrin clot and directly reflects the function of the maximum dynamic properties of fibrin and platelet. MA is, therefore, regarded as a useful index of perioperative bleeding [5]. Recently, acceleration of fibrinolysis has also been reported to be a potential cause of perioperative bleeding [8]. Both MA and FR can be adequate parameters to detect bleeding diatheses.

This patient showed normal ranges of MA and FR in TEG measurement preoperatively, even with a platelet number of $4.2 \times 10^4\ \mu l^{-1}$. Platelet concentrates were prophylactically transfused to the patient. In this case hypercoagulability and hyperfibrinolysis were detected by TEG after transfusion of platelet concentrate. This acceleration of coagulability and fibrinolysis might have been caused by excess transfusion of platelet concentrate. The mechanism of this acceleration is uncertain; however, it is suspected that platelet surface receptor appearance was increased and that intraplatelet signal transduction systems could be easily activated by the transfusion of platelet concentrate in such patients with chronic thrombocytopenia.

Surgical stress per se could activate coagulability and fibrinolysis [9–12]. The mechanisms of this acceleration are thought to depend on pain and/or release of tissue factors by surgical incision. Blood coagulability could be elevated more in general anesthesia than in epidural anesthesia [11]. The mechanism of its effect depends on the increase in blood circulation caused by sympathetic nerve block and the inhibition of catecholamine release [13]. If we performed epidural anesthesia, the acceleration of intraoperative coagulability would be reduced. Such hypercoagulability and hyperfibrinolysis would lead to the consumption of platelets and coagulating factors, subsequently bringing about a tendency to bleeding. In this case, red blood cell transfusion containing anticoagulating agents seems to have improved this acceleration of coagulability and fibrinolysis during the operation. It is recommended that blood derivatives and anticoagulating or antifibrinolytic agents be used, under TEG monitoring, on patients with hemostatic abnormalities. Furthermore, where abnormalities of coagulation and/or fibrinolysis are demonstrated on the TEG during operation, the efficacy of using a particular pharmacological agent or a blood product can be assessed in vitro before administering it to the patient in order to conduct safe management of blood coagulability and to avoid excess transfusion [4,14].

This case illustrates that perioperative platelet transfusion does not always bring about desirable consequences; TEG monitoring is, therefore, extremely useful in operations on patients with hemostatic abnormalities.

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