

Platelet mapping in postoperative management of acute aortocoronary bypass thrombosis

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Abstract

Platelet mapping has been introduced recently to help with the assessment of the antiplatelet drug therapy, which is necessary in patients who underwent coronary bypass surgery. A case of acute bypass thrombosis in the immediate postoperative period is presented, and the management of evolving myocardial ischemia is described. The efficacy of thromboelastography platelet mapping in appropriate postoperative antiplatelet therapy is highlighted.

Keywords: bypass thrombosis, thromboelastography, platelet mapping

Streszczenie

W celu oceny skuteczności terapii lekami przeciwplatetkowymi, niezbędnymi w leczeniu chorych po operacjach pomostowania tętnic wieńcowych, wprowadzono badanie zwane mapowaniem płytek. Praca przedstawia przypadek ostrego zakrzepu pomostu w bezpośrednim okresie pooperacyjnym, opisując postępowanie przy rozwijającym się zawale serca z wykorzystaniem mapowania płytek do ustalenia właściwej terapii przeciwplatekowej.

Słowa kluczowe: zakrzepica pomostu, tromboelastografia, mapowanie płytek

Introduction

Thromboelastography (TEG) represents a standard method for the postoperative evaluation of hemostasis [1]. It helps to differentiate surgical from nonsurgical bleeding [2] and minimize the rate of unnecessary transfusions [3]. Unfortunately, the examination is insensitive to the effects of aspirin [4].

TEG platelet mapping (TEGPM) has been recently introduced for assessing the efficacy of antiplatelet drug therapy. The examination consists of adding subsequent agonists to the platelet receptor to a blood sample in order to activate platelets that were not inhibited by antiplatelet drugs.

The participation of platelets in the clot formation process requires activation of glycoprotein IIa/IIIb complex, which is a receptor for fibrinogen. Both the activation and inhibition of this receptor may be direct or indirect. Adding adenosine-5-diphosphate (ADP) activator or arachidonic acid to the blood of a patient treated with clopidogrel or aspirin, respectively, will activate noninhibited platelets. TEGPM tracing will show activated platelets and confirm or disprove the treatment efficacy.

Case report

A 64-year-old man was admitted to our department for elective coronary artery bypass surgery (CABG). Coronarography revealed a significant disease of the diagonal (Diag) and left anterior descending (LAD) arteries. Echocardiography showed good left ventricular function with no valve disease. A sequential mammary bypass to the Diag and LAD was performed on the beating heart. To check the quality of the constructed grafts, blood flows and their pulsatility indexes (PIs) were measured with a transit-time flowmeter (Medi-Stim, Oslo, Norway). A relatively good flow (20 ml/min) with a low PI (2.9) was recorded on the mammary artery proximally to the side-to-side anastomosis. Unfortunately, there was no measurable flow and a high PI on the distal anastomosis in the sequence. The topical application of papaverine to exclude a spasm as a cause of the insufficient flow did not improve the flow. The mammary graft was removed and replaced by a venous sequential aortocoronary bypass. The blood flow was 30 ml/min with a PI of 2.3 in the proximal segment of the graft, and 25 ml/min with a PI of 1.5 in the distal segment. The flow and PI values did not change after

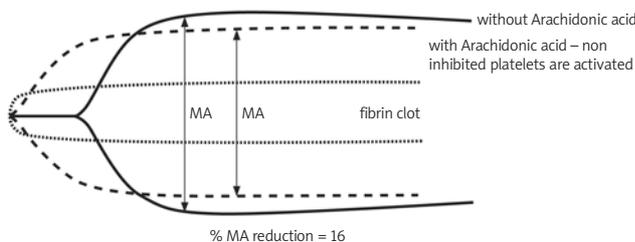
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protamine administration. The chest was closed and the patient was transferred to the ICU.

Two hours after the procedure, ST-segment elevations appeared in leads II and V5. Echocardiography revealed hypokinesis of the anterior wall of the left ventricle. Coronarography revealed a bypass thrombosis just upstream the side-to-side anastomosis and thrombosis of the entire Diag, and thromboelastography revealed hypercoagulability (the postoperative blood loss up to the repeat surgery was 220 ml over 5 h). The patient was returned to the operating room. Single off-pump aortocoronary bypasses to the LAD and Diag were performed using new vein conduits, and an endarterectomy was performed on the Diag. The blood flow through the bypasses was satisfactory (30 ml/min in the Diag and 33 ml/min in the LAD).

Accentuated anticoagulation therapy was initiated immediately with 400 mg of aspirin and low-molecular-weight heparin, with the good effect of the latter, confirmed by the anti-Xa activity test. TEGPM performed on the 1st postoperative day confirmed the efficacy of the aspirin treatment. However, TEGPM repeated on the 5th postoperative day showed aspirin resistance, and hence aspirin was swapped for clopidogrel. TEG platelet mapping confirmed good platelet inhibition with clopidogrel (Fig. 1). The subsequent postoperative course of the patient was uneventful. The left ventricular ejection fraction improved to normal (55%) with no segmental hypokinesis. The patient was discharged home on the 7th postoperative day.

A. Effect of Aspirin



B. Effect of Clopidogrel

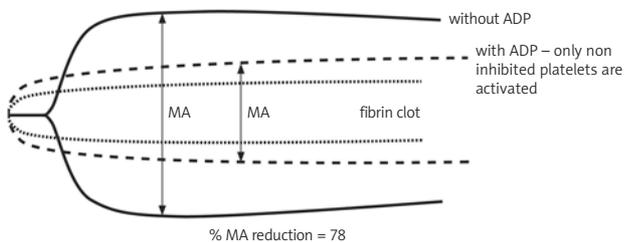


Fig. 1. Thromboelastography – platelet mapping tracings.

A. Ineffective treatment with aspirin. The maximal thromboelastography amplitude (MA) reduced by only 16% after the administration of arachidonic acid (most platelets could still be activated and detected after the administration of arachidonic acid).

B. Good effect of treatment with clopidogrel. The MA reduced by 78% after the administration of ADP (only 22% of platelets were detected, with the remainder well blocked by clopidogrel).

Four months after surgery he was asymptomatic, and spiral computed tomography confirmed that both grafts had good patency (Fig. 2).

Discussion

Antiplatelet therapy is required after CABG surgery in order to prevent bypass closure (thrombosis) and improve its long-term patency. The importance of this treatment is highlighted by the recommendation to begin antiplatelet therapy within 12 hours after the surgery. Most patients undergoing CABG routinely receive aspirin postoperatively. However, it has been shown that in 5–60% of patients, aspirin does not achieve adequate efficacy in various measures of platelet-activity inhibition [5]. Previous studies have estimated that 5–45% of the population does not achieve an adequate antiplatelet effect with aspirin [6], and that 20% of patients with stable angina are resistant to aspirin [7]. Especially in patients undergoing off-pump surgery, who are at an increased risk (6.5%) for early bypass closure due to hypercoagulability, it seems reasonable to monitor the adequacy of aspirin treatment [8].

TEGPM enables the effect of antiplatelet drugs to be quantified. Examination of blood samples with platelet-receptor agonists allows the effect of the chosen drug to be identified (with respect to the platelet receptor, which should be inhibited). If the treatment is effective the TEGPM trace will show a substantial (>50%) reduction in maximal amplitude of the curve after administration of the agonist. This is caused by the presence of only small number of activated, noninhibited platelets (Fig. 1).

Because aspirin resistance may not manifest immediately after surgery, it is mandatory to repeat TEGPM a few days

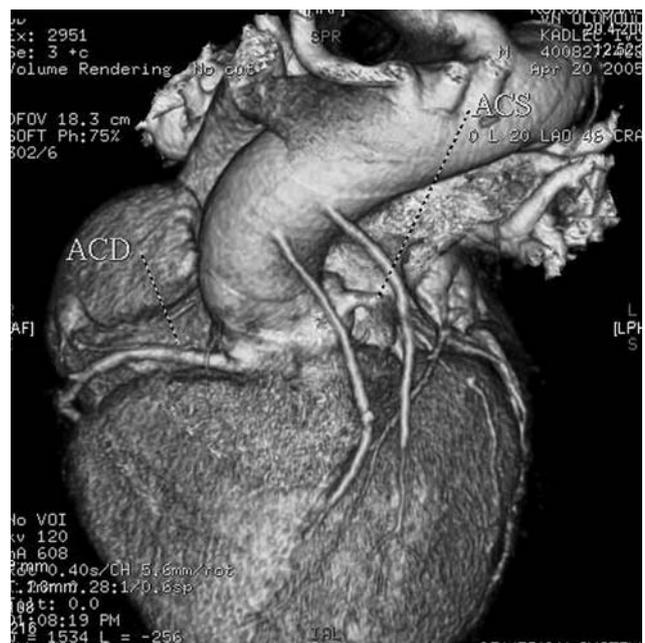


Fig. 2. Postoperative spiral computed tomography examination. Good patency of both venous grafts (Diag and LAD) at 4 months postoperatively

postoperatively. It is also important to be aware of the high probability of bypass thrombosis in an off-pump patient with a low postoperative blood loss.

Conclusion

Patients at an increased risk for bypass thrombosis should be closely monitored regarding their antiplatelet therapy, and TEGPM appears to be an indispensable tool in their management.

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