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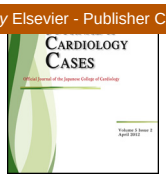

Case Report

A case of acute myocardial infarction during perioperative period of non-cardiac surgery in a patient with antiphospholipid syndrome and a history of coronary artery bypass surgery

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ABSTRACT

A 65-year-old woman underwent coronary artery bypass surgery and was diagnosed with antiphospholipid syndrome (APS) at the same time in 1985. She was admitted to our hospital to undergo mastectomy for left breast cancer in 2012. She was put on intravenous infusion of heparin and stopped receiving both antiplatelet agents and warfarin. The operation was performed without complications, and antithrombotic therapy was restarted one day after the operation. On day 6 postoperative, she complained of sudden chest pain and on examination she was diagnosed with acute myocardial infarction. The culprit lesion was in a saphenous vein graft and coronary intervention was performed.

<**Learning objective:** Antithrombotic therapy for patients with APS is complicated because of prolonged baseline activated partial thromboplastin time (aPTT). An effective perioperative antithrombotic therapy for APS patients who have a history of coronary artery disease and have undergone non-cardiac surgery has not yet been established. A safe strategy for such a therapy should therefore be discussed.>

Introduction

Antiphospholipid syndrome (APS) increases the risk of coronary artery disease [1,2]. Perioperative management of antithrombotic therapy for patients with APS is complicated because of prolonged baseline activated partial thromboplastin time (aPTT). We report here a case of an APS patient with a history of coronary artery bypass surgery suffering acute myocardial infarction during perioperative period of non-cardiac surgery.

Case report

A 65-year-old woman underwent coronary artery bypass surgery (– ascending aorta – saphenous vein graft – left anterior descending artery –) because of left main trunk stenosis and was diagnosed with APS at the same time in 1985. Prior to the surgery, she had been diagnosed with diabetes and deep venous thrombosis and had been receiving antithrombotic therapy with warfarin, aspirin and ticlopidine.

She was admitted to our hospital to undergo mastectomy for left breast cancer in September 2012. Laboratory data showed a prolonged aPTT of 79.5 s (reference 23.0–38.0 s). She was put on intravenous heparin infusion of about 10,000 units per day and stopped receiving warfarin, aspirin, and ticlopidine 7 days before the operation. The infusion of heparin was adjusted to keep the aPTT at approximately 100 s and was stopped 6 h before the operation. Warfarin, aspirin and ticlopidine were restarted 1 day after the operation. She received the infusion of heparin on days 1 and 2 after the operation. Six days after operation she complained of sudden chest pain and her electrocardiogram revealed ST-segment elevation in I, aV_R, aV_L, and V4–6 leads. She was then diagnosed with acute myocardial infarction. The PT-INR and aPTT of 6 days after the operation were 1.24 and 61.6, respectively. Emergent cardiac catheterization was performed. A 99% stenosis with delay was detected in the saphenous vein graft and a 90% stenosis was detected in the left anterior descending artery (Fig. 1). Under the support of intraaortic balloon pumping, percutaneous intervention was performed on the culprit lesion in the saphenous vein graft with a bare metal stent and plain old balloon angioplasty to the left anterior descending artery. The patient's maximum creatinine kinase was 7522 IU/L, and the presence of heart failure was determined. She was stabilized by intensive care and had no recurrence

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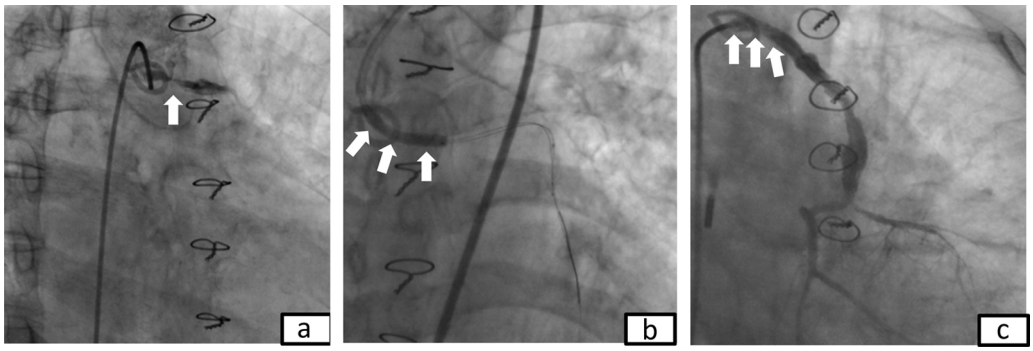

Fig. 1. (a) A 99% stenosis (white arrow) with delay was detected near the ostium of the saphenous vein graft. (b) Coronary intervention with a bare metal stent (white arrows) was performed in the saphenous vein graft. (c) Final angiography showed TIMI grade 3 flow. White arrows show the location of the bare metal stent.

of any thromboembolic diseases or bleeding complications. About 2 months after the onset of acute myocardial infarction she was transferred to a different hospital for rehabilitation.

Discussion

The prevalence of antiphospholipid antibodies is about 1–4% in normal controls [3]. Our patient did not have systemic lupus erythematosus, although about 11–38% of patients with systemic lupus erythematosus have antiphospholipid antibodies [3,4]. The prevalence rates of myocardial infarction, angina pectoris, and coronary bypass rethrombosis in patients with APS are 5.5%, 2.7%, and 1.1%, respectively [1]. Because of APS, our patient had a history of cardiovascular disease when she was young, and experienced recurrent cardiovascular events. The risk of such recurrent cardiovascular events is high in post-infarction patients with antiphospholipid antibodies [2,3]. We should be careful of cardiovascular events in patients with APS, especially those who have a history of coronary artery disease.

Diabetes, cancer, as well as surgery are risk factors of thrombosis [2], and were related to recurrent myocardial infarction in our case. Furthermore, antiphospholipid antibodies are associated with graft failure [5,6]. Safe preoperative management for patients with APS is important to prevent this.

Table 1
Antithrombotic strategies during perioperative period of cardiac surgery.

Patient	Preoperative anticoagulation	Intraoperative anticoagulation with heparin	Baseline aPTT (s)	Baseline ACT (s)	Actual ACT (s)	Complications	Report
69-year-old man (redo CABG)	None	ACT	48.7	None	540–660	Bleeding, renal failure, DVT, s/o PTE, ischemia of the toes	Sheikh et al. [13]
44-year-old man (MVR)	None	Double the baseline ACT	49.8	430	Over 999	None	Sheikh et al. [13]
47-year-old man (CABG)	None	Heparin concentration	47	236	Over 999	None	Ducart et al. [14]
51-year-old man (CABG + MVR)	None	(1) Celite ACT (2) Anti-Xa concentration	52	None	Over 550	Bleeding	East et al. [15]
49-year-old woman (MVR)	None	(1) Celite ACT (2) Anti-Xa concentration	45.8	None	Over	None	East et al. [15]
25-year-old woman (AVR)	None	Heparin concentration	68	249 (kaolin) 192 (celite)	Over 999 (kaolin) Over 1500 (celite)	Bleeding	Hogan et al. [16]
46-year-old woman (MVR)	None	Heparin concentration	116	219	Over 999	Cardiac tamponade rhythm disturbances	Brownstein et al. [10]
33-year-old woman (MVR)	Enoxaparin 100 mg/day	(1) Kaolin ACT (2) Anti-Xa concentration	44.5	147	388–517	None	Lennon et al. [11]
31-year-old woman (MVR)	i.v. heparin to double the baseline aPTT	(1) Kaolin ACT greater than 600 s (2) Anti-Xa concentration	49	168	603–819	Acute biventricular failure	Dorman [17]
40-year-old man (OPCAB)	None	(1) Celite ACT greater than 300 s (2) Heparin concentration	61.8	112	300	None	Maddali and Albahrani [12]
33-year-old man (MVP)	Enoxaparin	Celite ACT greater than 500 s	42	153	417–601	None	Weiss et al. [18]
68-year-old man (OPCAB)	None	(1) ACT (2) Heparin concentration	60.4	228	764–945	Arteriovenous fistula thrombus, atrial fibrillation	Jervis et al. [19]
70-year-old man (CABG + AVR)	None	(1) Celite ACT (2) Heparin concentration (3) Anti-Xa concentration	65	123 (celite) Over 200 (kaolin)	Over 600 (celite) Over 999 (kaolin)	None	Cartwright et al. [20]

aPTT, activated partial thromboplastin time; ACT, activated coagulation time; CABG, coronary artery bypass grafting; DVT, deep vein thrombosis; s/o, suspected of; PTE, pulmonary thromboembolism; MVR, mitral valve replacement; AVR, aortic valve replacement; OPCAB, off-pump coronary artery bypass.

There are only a few reports of perioperative antithrombotic therapies for patients with APS. We chose here unfractionated heparin therapy as a bridging therapy instead of warfarin, aspirin and ticlopidine. Management of heparin therapy in patients with APS is complicated because of prolonged baseline aPTT. In our case, aPTT was within therapeutic range determined by aPTT ratios of approximately 2.5 times the reference value [7] but myocardial infarction happened. The bridging therapy was an important factor of acute myocardial infarction due to saphenous vein graft failure 28 years after coronary artery bypass surgery. Previous reports suggested the possibility of underanticoagulation and recommended the use of unfractionated heparin or low molecular weight heparin [8,9]. Some reports show preoperative antithrombotic therapy using enoxaparin or heparin with doubling the baseline aPTT in cardiac surgery for patients with APS [10–12]. Intraoperative antithrombotic therapies by heparin have been reported to double the baseline activated coagulation time, to monitor anti-Xa concentration, and heparin concentration in cardiac surgery for patients with APS ([10–20], Table 1). However, the strategy of antithrombotic therapy during perioperative period of non-cardiac surgery for patients with APS has not been documented in literature. We propose that the strategy of perioperative antithrombotic therapy for cardiac surgery in APS patients should apply to the patients undergoing non-cardiac operation. It is worth using unfractionated heparin with doubling the baseline aPTT or using low molecular weight heparin.

Early discontinuation of heparin before enough blood concentration of warfarin might be one of possible reasons of myocardial infarction in this case. We should have used heparin until achieving therapeutic range of substitute antithrombotic medication like warfarin.

Some reports described perioperative management of patients with coronary artery disease during non-cardiac surgery. Using nitrate or nicorandil may be another option to reduce the risk of cardiovascular disease during perioperative period [21,22].

A safe strategy should be established regarding perioperative antithrombotic therapy for patients with APS and a history of coronary artery disease.

Conflict of interest

Authors declare no conflict of interest.

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