CASE REPORT

DOI: 10.5114/kitp.2016.64883

Coronary artery spasm following on-pump coronary artery bypass grafting with 20 months follow-up

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Kardiochirurgia i Torakochirurgia Polska 2016; 13 (4): 361-365

Abstract

We report on a 69-year-old woman who demonstrated native coronary artery and grafted vessel spasm following on-pump coronary artery bypass grafting (CABG). Despite intraaortic balloon pump (IABP) insertion, electrocardiogram (ECG) abnormalities did not disappear. Emergency coronary angiography (CAG) was performed. The patient was successfully treated with systemic and intracoronary injection of vasodilator agents. ECG changes disappeared, with normalized and stable hemodynamic function. Intraaortic balloon pump was maintained for 48 h. The patient was discharged in good clinical condition. Coronary artery spasm (CAS) may result in life-threatening arrhythmias, circulatory collapse or death. The etiology of CAS is multifactorial and includes heart manipulation, exogenous vasoconstrictors, stress-related catecholamine release, hypoxia and oxidative stress. Postoperative CAS is most commonly manifested by ST-segment elevation and circulatory collapse without specific causes. The gold standard for revealing CAS is CAG. Infusion of vasodilators combined with IABP is adequate in most instances, but extracorporeal membrane oxygenation has been necessary for more extensive or resistant coronary spasm.

Key words: coronary artery bypass grafting, coronary artery spasm, coronary angiogram, on-pump cardiac surgery.

Streszczenie

W pracy przedstawiono przypadek 69-letniej kobiety, u której po zabiegu pomostowania aortalno-wieńcowego w krążeniu pozaustrojowym wystąpił spazm tętnic wieńcowych i pomostów naczyniowych. Pomimo zastosowania kontrapulsacji wewnątrzaortalnej, nieprawidłowości w zapisie elektrokardiograficznym (EKG) nadal się utrzymywały. Natychmiast wykonano koronarografię. Pacjentka była skutecznie leczona ogólnoustrojową oraz dowieńcową podażą leków naczyniorozszerzających. Zmiany w EKG zniknęły, a stan hemodynamiczny pacjentki się ustabilizował. Kontrapulsację wewnątrzaortalną utrzymywano przez 48 godzin. Pacjentka została wypisana z oddziału w stanie dobrym. Spazm naczyń wieńcowych może skutkować zagrażającymi życiu zaburzeniami rytmu, zapaścią krążeniową, a nawet zgonem. Do czynników, które mogą go wywołać, należą: manipulacja przy sercu, egzogenne wazokonstryktory, wyrzut katecholamin, hipoksja i stres oksydacyjny. Pierwszym objawem jest najczęściej uniesienie odcinków ST w EKG oraz dekompensacja hemodynamiczna bez uchwytnych przyczyn. Złotym standardem diagnostycznym jest koronarografia. Podanie leków naczyniorozszerzających w połączeniu z kontrapulsacją wewnątrzaortalną jest na ogół postępowaniem wystarczającym. W bardziej opornych na leczenie przypadkach niezbędne może okazać się zastosowanie pozaustrojowej oksygenacji krwi.

Słowa kluczowe: pomostowanie aortalno-wieńcowe, spazm naczyń wieńcowych, angiografia wieńcowa, krążenie pozaustrojowe.

Case report

We report a case of a 69-year-old non-smoking woman after ST-elevation myocardial infarction with stent implantation in the left artery descending (LAD) 11 years ago. No peripheral blood vessel diseases, such as Raynaud's phenomenon, were present in the patient's medical history. The patient presented with exertional angina despite optimal medical treatment. Admission coronary angiogram (CAG) showed ostial stenosis of LAD (70%), proximal obtuse marginal artery (OM) (70%), right coronary artery (RCA) segment 2 (60%) and ostial posterior descending artery (PDA) (70%). Preoperative echocardiography confirmed good left ventricle ejection fraction (50%). Coronary artery bypass grafting (CABG) procedure was performed with a saphenous vein graft to the PDA and a composite Y vein graft to the OM and LAD. The left internal thoracic artery (LITA) was harvested but not used as no satisfactory flow could be obtained. No injury or dissection of the LITA was observed. Lack of flow probably resulted from arterial spasm. The flow in coronary grafts was not assessed as the weaning from the cardiopulmonary bypass (CPB) was uneventful. The

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Received: 13.03.2016, **accepted:** 27.05.2016.

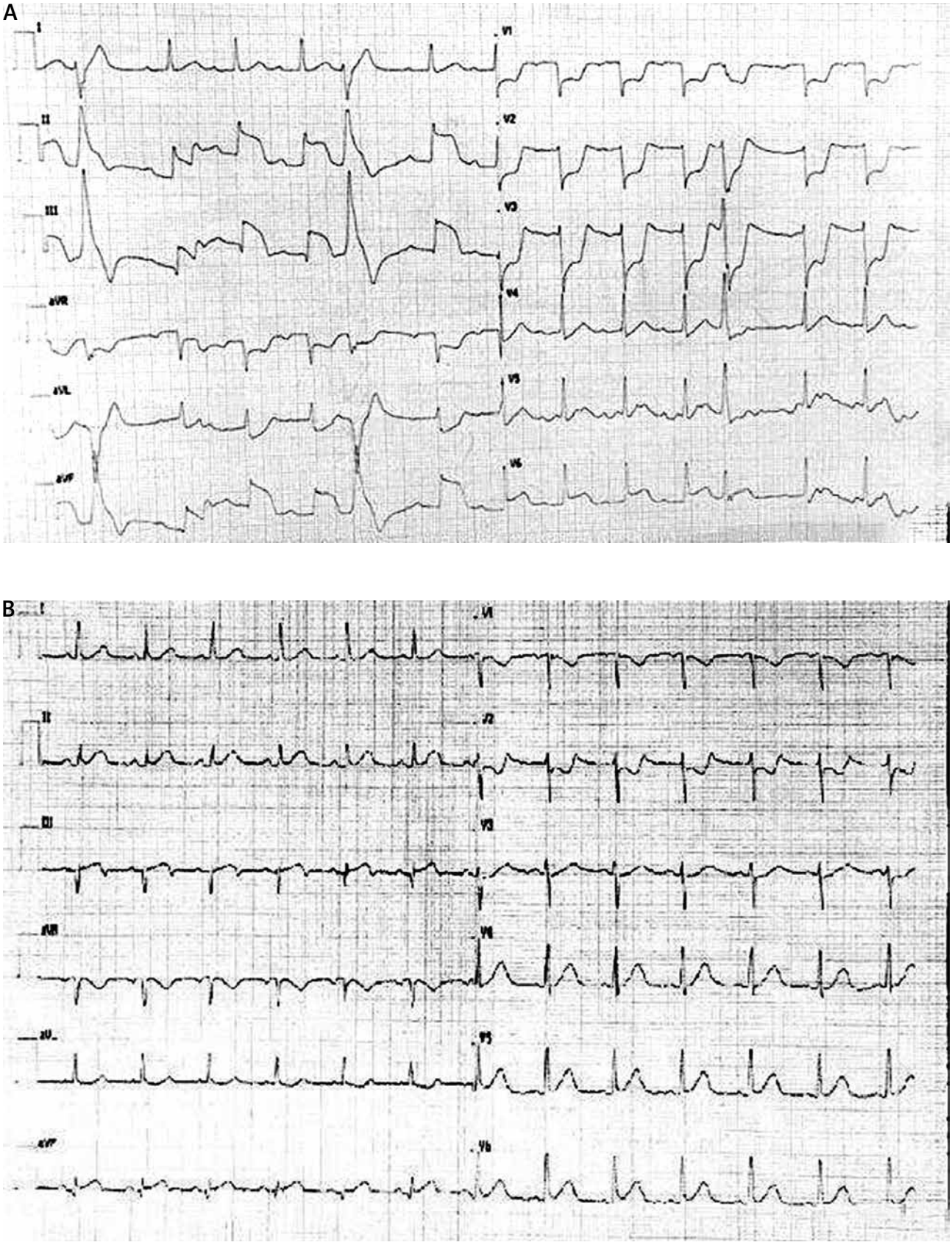

Fig. 1. A – Postoperative electrocardiogram (ECG) with ischemic changes, **B** – normalized ECG

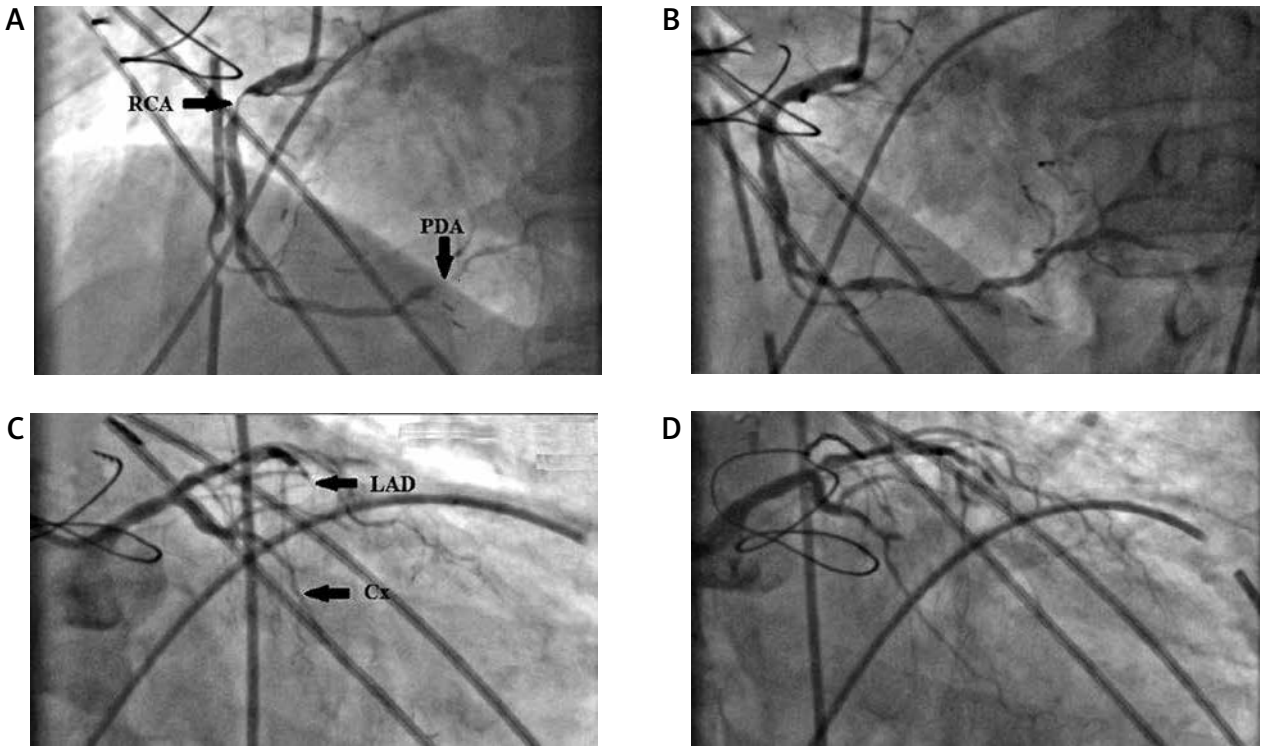

Fig. 2. Postoperative coronary angiogram – native vessels. **A** – Postoperative right coronary artery (RCA) and posterior descending artery (PDA) spasm, **B** – RCA and PDA after nitroglycerine infusion, **C** – Postoperative left artery descending (LAD) and circumflex (Cx) spasm, **D** – LAD and Cx after nitroglycerine infusion

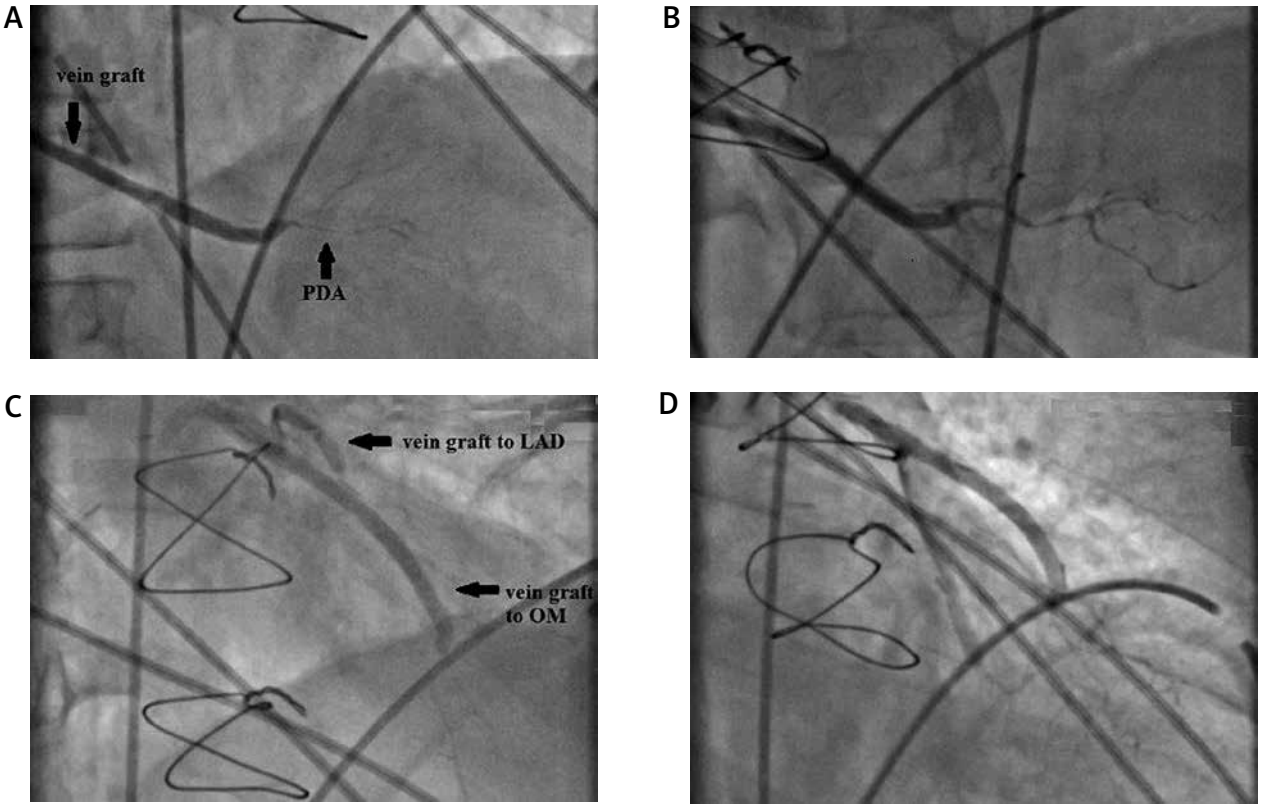

Fig. 3. Postoperative angiograms – coronary grafts. **A** – Patent graft to posterior descending artery (PDA) with spasm, **B** – graft to PDA after nitroglycerine infusion, **C** – Y-graft to left artery descending (LAD) and obtuse marginal (OM) with spasm, **D** – Y-graft to LAD and OM after nitroglycerine infusion

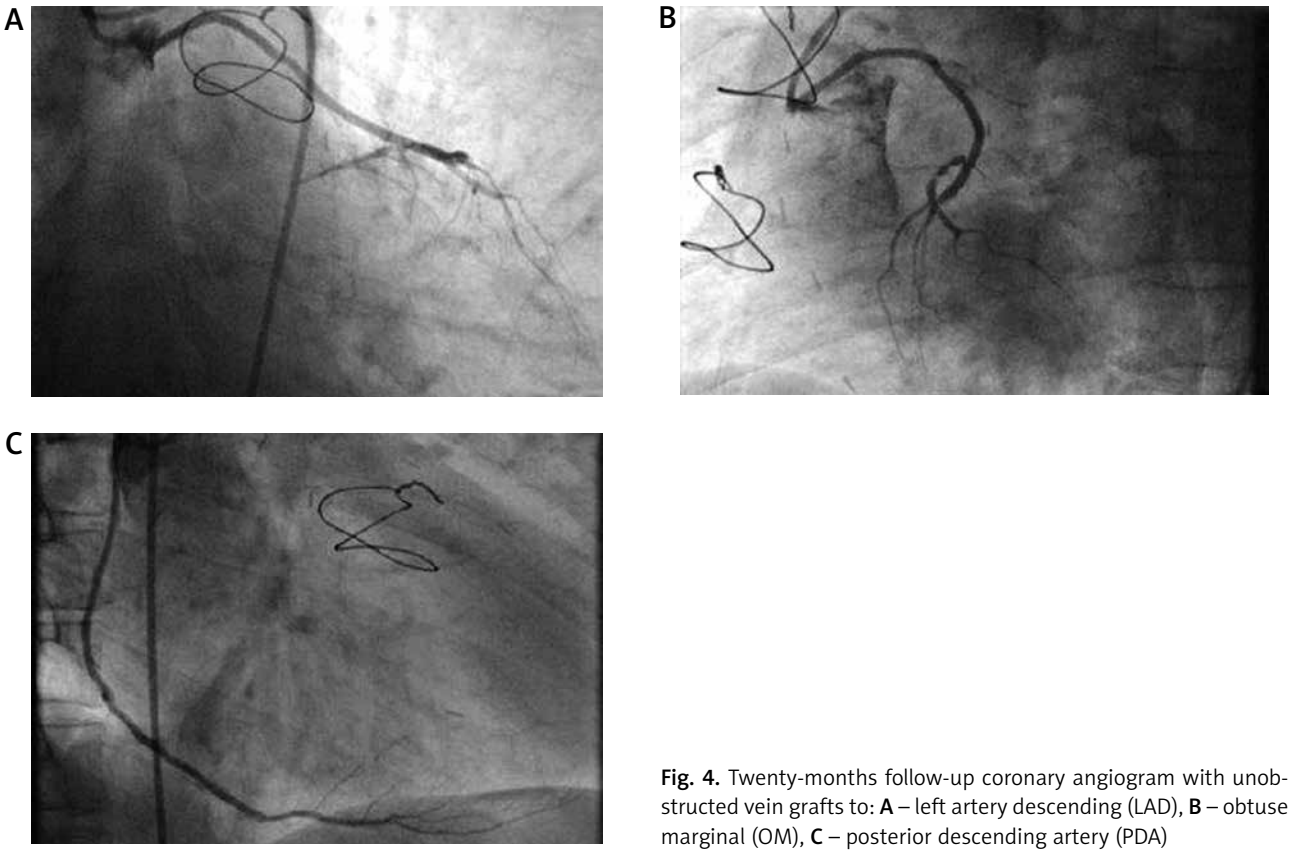

Fig. 4. Twenty-months follow-up coronary angiogram with unobstructed vein grafts to: **A** – left artery descending (LAD), **B** – obtuse marginal (OM), **C** – posterior descending artery (PDA)

patient returned to the intensive care unit (ICU) in a stable condition on dopamine. In the immediate postoperative period, ECG showed ST-segment elevation in leads II, III, aVF and depression in leads V1–V3 (Fig. 1 A). Then the intra-aortic balloon pump (IABP) was inserted at the ICU, but the electrocardiogram (ECG) abnormalities did not disappear. The patient was transferred immediately to the catheter laboratory. Two hours after the CABG troponin T was 0.122 ng/ml and creatine kinase (CK)-MB_{mass} 41 µg/ml. CAG revealed diffuse severe triple-vessel coronary artery spasm (CAS) (Figs. 2 A, C, 3 A, C). Systemic and direct infusion of nitroglycerine into the coronary arteries and grafts alleviated the spasm (Figs. 2 B, 2 D, 3 B, 3 D). Changes in the ECG disappeared (Fig. 1 B). On the next day the troponin T level was 0.380 ng/ml. Echocardiography showed an ejection fraction of 55%. The IABP was discontinued on postoperative day two. The patient was transferred in good condition to the cardiac unit on the ninth postoperative day and remains symptom-free at 20 months follow-up (Figs. 4 A, B, C).

The CAS after CABG is a rare (0.8–1.3%) but life-threatening complication with high morbidity and mortality [1]. It can occur during surgery or in the immediate postoperative period. It may involve manipulated or non-manipulated vessels, an implanted graft or native coronary arteries [2], but the RCA is most commonly involved [1]. The etiology of CAS after CABG is still undetermined, but several factors could induce vasospasm: vascular damage, oxidative stress, high levels of administered or endogenous vasoconstrictors, electrolyte abnormalities (hypomagnesaemia, hyperkalaemia), hypothermia, hypocapnia, preoperative use of β-blocking agents, calcium channel blockers (CCB) and high dosage of nitroglycerine [3]. Postoperative CAS is most commonly manifested by ST-segment elevation, hemodynamic instability, arrhythmia, circulatory collapse or cardiac arrest [3]. The gold standard for revealing CAS is CAG [4]. The best treatment for CAS is intravenous injection of vasodilator agents such as isosorbide dinitrate, adenosine triphosphate, papaverine, nicorandil and CCB. Several studies have reported that stent implantation is an option in cases of refractory CAS with focal lesions, but it is associated with many complications. The CAS is difficult to establish in patients hemodynamically. Preoperative recognition of high-risk patients, avoiding manipulation of the heart to minimize surgical trauma, minimizing the use of the carbon dioxide blower and avoiding hypothermia have all been proposed for the prevention of CAS. Postoperatively infusion of nitroglycerine, calcium antagonists and supplemental magnesium may decrease the risk of CAS [1].

Disclosure

Authors report no conflict of interest.

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