Acute coronary syndrome in patients with concomitant neoplastic disease and chemotherapy. Is clinical distinctness possible?

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ABSTRACT

We present 2 cases of acute coronary syndrome in patients with concomitant neoplastic disease during chemotherapy. In both patients, we found unusual angiographic image which showed that despite the presence of very large thrombi, there were no or hardly any atherosclerotic lesions in the vessel occlusion site. Aspiration thrombectomy was successfully performed in both cases to restore coronary flow and in the second patient, two bare metal stents were also implanted. After the patients were discharged from hospital, we decided to add enoxaparin to the routine dual antiplatelet therapy of both of them. So far, no guidelines have been developed on how to manage patients with acute coronary syndrome and concomitant neoplastic diseases while on chemotherapy.

KEY WORDS: acute coronary syndrome, chemotherapy, endothelial dysfunction, coronary thrombosis

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INTRODUCTION

Acute coronary syndrome (ACS) is usually caused by atherosclerosis which leads to endothelial dysfunction and secondary increase in coagulation and activation of platelets. Recently, due to population ageing and a better control of cardiovascular risk factors, the percentage of patients suffering from ACS and a concomitant neoplastic disease has been on the rise. It is well known that cancer and chemotherapy increase coagulation and risk of ACS respectively. In this group of patients, we may frequently find unusual clinical and angiographic image, which requires an individual approach. No guidelines exist on how to manage these patients (with ACS and neoplastic diseases) and usually they receive routine ACS treatment, i.e. primary percutaneous coronary intervention (PPCI) with stent implantation and 12 months of dual antiplatelet therapy (DAPT) [1, 2].

In the article, we present 2 patients with a neoplastic disease and ACS which occurred during chemotherapy. In both cases, a large thrombus formation was the cause of ACS. Both interventions were preceded by an administration of loading doses of heparin, DAPT, GP IIb/IIIa inhibitors infusion and aspiration thrombectomy. Later, two different clinical scenarios were carried out.

PRESENTATION OF THE CASES

Patient 1.

A 64-year-old man with a history of coronary disease and diabetes type 2, 16 years after coronary artery bypass surgery (CABG) and concomitant prostate cancer with bones metastases had been treated for 2.5 years with palliative chemotherapy which included leuprorelin 22.5 mg, zoledronic acid 4 mg and bicalutamide 50 mg.

The patient was hospitalized for inferior nSTEMI. Cardiac markers values were: TnT 133 ng/ml (normal value < 14 ng/l), CK-MB 139 ng/ml (normal value < 3.77 ng/ml).

Coronary angiography revealed a big fresh thrombus totally obliterating the saphenous vein graft (SVG) to right coronary artery (RCA) as a culprit lesion, and absence of significant atherosclerotic changes in other arteries and grafts requiring invasive treatment. Aspiration thrombectomy from the SVG was performed (Hunter Extraction Catheter, IBERHOSPITEX) with positive results and good TIMI 3 flow. Given the correct angiographic picture of the graft after thrombectomy, neither balloon angioplasty nor stent implantation was indicated.

The patient was discharged 2 weeks later with left ventricular ejection fraction (LVEF) 40%. Enoxaparin 1 mg/kg daily was added to the routine DAPT.

Patient 2.

A 35-year-old man presented with anterior STEMI 2 months after orchidectomy due to testicular seminoma and directly after the first course of chemotherapy with bleomycin 30 mg, etoposide 210 mg, cisplatin 42 mg and prednisone 10 mg. At admission, TnI was 3.6 ng/ml (normal value < 0.033 ng/ml), CK-MB mass 24.5 ng/ml (normal value < 6.6 ng/ml).

Coronary angiography revealed a large thrombus in the proximal and mid-left anterior descending artery (LAD) as a culprit lesion, and no changes in other arteries. Before the intervention, the patient was treated with abciximab i.c., heparin i.v., and

FIGURE 1.
PATIENT 1. A – total occlusion of the saphenous vein graft (SVG) with a big thrombus; B – introduction of an extraction catheter into SVG and thrombus aspiration (the arrow points to the catheter); C – SVG with restored flow after thrombus aspiration (TIMI 3). SVG is wide, with no visible atherosclerotic stenosis – no indication for balloon angioplasty or stent implantation.

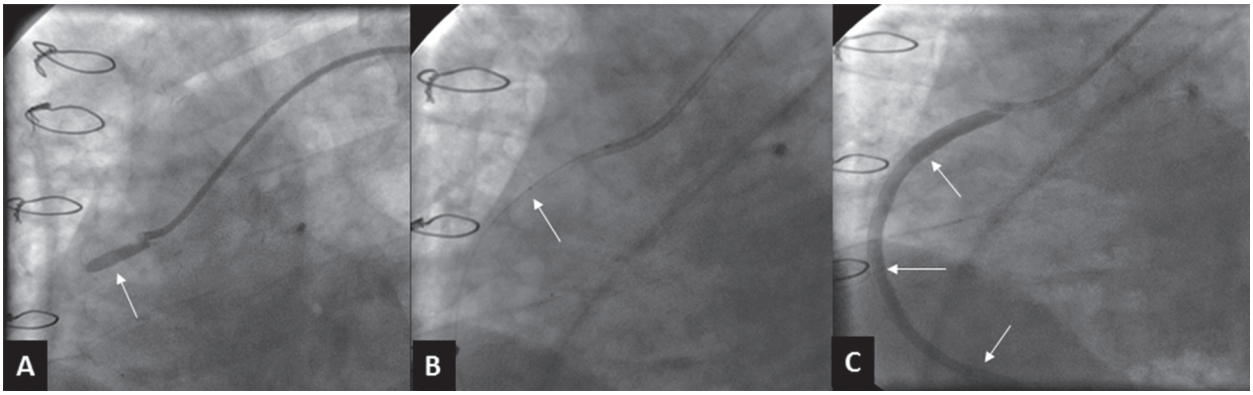
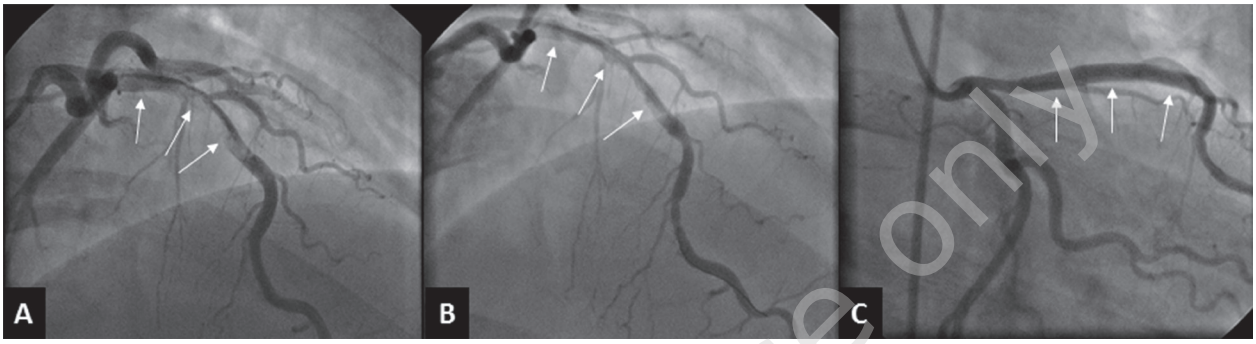


FIGURE 2.
PATIENT 2. A – fresh massive thrombus in proximal and mid LAD; B – after repeated thrombectomy, only a part of the thrombus was evacuated and a decision on stent implantation was taken; C – final result of angioplasty with 2 BMS implanted in LAD and restored coronary flow.



ticagrelor. Next, aspiration thrombectomy was performed (Diver Extraction Catheter, INVATEC). Despite numerous repeated aspirations, only part of the thrombus was evacuated from LAD and 2 bare-metal stents (BMS) – Coflexus 4.5 × 34 mm and 4.0 ×18 mm, 18 atm. were implanted. The patient was discharged 6 days later with normal LVEF (60%). Like in the previous case, enoxaparin at a dose of 1 mg/kg daily was added to DAPT.

DISCUSSION AND CONCLUSIONS

In patients with neoplastic diseases, the leading cause of ACS may be linked to endothelial dysfunction and hypercoagulability due to chemotherapy. On angiography, atherosclerotic lesions may be only slightly advanced in these patients or even invisible in some cases. Therefore, invasive and, later on, pharmacological treatment of ACS should rely on interventional thrombus aspiration as well as intensive, prolonged antiplatelet and antithrombotic therapy, as they might be crucial for short- and long-term clinical success. Some thrombi detected during intervention performed in the acute phase of ACS often prove to be much bigger and thus require individual, non-standard approach. In the current clinical practice, doctors usually prescribe balloon angioplasty and stent implantation. However, given the inflammatory etiology of atherosclerosis and ACS, high-pressure balloon angioplasty and stent implantation – if not necessary – may be particularly harmful for patients with neoplastic disease while on chemotherapy, additionally causing mechanical endothelial damage and dysfunction as well as creating risk of restenosis.

Recent guidelines on ACS treatment and myocardial revascularization do not settle several important issues concerning invasive treatment of patients with ACS and a concomitant neoplastic disease during chemotherapy. Some of the burning questions are: Is routine dual antiplatelet treatment sufficient or should antithrombotic treatment be added? For how long? At what dose? Which stents – bare metal (BMS) or drug-eluting (DES) – should be used? DES are considered more thrombogenic; moreover, it may turn out that some patients require neoplastic surgery. The answer to these questions may be crucial for successful outcome of the patients and is open for a scientific discussion.

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References:

1. Windecker S, Kohl P, Alfonso F et al. 2014 ESC/EACTS Guidelines on myocardial revascularization. The Task Force on Myocardial Revascularization of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J 2014; 35(37): 2497-2503.
2. Steg PG, James SK, Atar D et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J 2012; 33: 2569-2619.

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Andrzej Ciszewski: 60%;
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