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Severe acute myocardial ınfarction and peripheral thrombosis in patient with bladder cancer: A case report

Mesane kanserli hastada ciddi akut miyokard enfarktüsü ve periferik toromboz: Olgu sunumu

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Abstract

Cancer-associated thrombosis worsens the lives of patients substantially. Venous manifestations of cancer-associated thrombosis include deep vein thrombosis and pulmonary embolism. Arterial events include stroke and myocardial infarction. In this patient, myocardial infarction and cardiogenic shock are associated with diffuse coronary thrombosis together with peripheral thrombosis. He had surgery because of bladder carcinoma. Severe hypercoagulable condition probably facilitated by cancer itself and surgery caused multivessel coronary and peripheral intense thrombus burden. Intracoronary 10 mcg/kg tirofiban bolus and 15 mg tissue plasminogen activator (tPA) were administered respectively before revascularization and thrombectomy operation was performed. Complete revascularization was achieved.

Keywords: Bladder cancer, Myocardial infarction, Peripheral ischemia, Thrombosis

Öz

Kansere bağlı tromboz, hastaların yaşamlarını önemli ölçüde etkiler. Kansere bağlı trombozun venöz olayları, derin ven trombozu (DVT) ve pulmoner embolinin (PE) yanı sıra visseral veya splanknik ven trombozunu içeren venöz tromboembolizmdir (VTE). Arteriyel olaylar ise inme ve miyokard enfarktüsünü içerir. Bu hastada, miyokard enfarktüsü ve kardiyojenik şok, periferik trombozla birlikte yaygın koroner tromboz ile ilişkilidir. Hastada kardiyovasküler risk faktörlerinden sigara ve hipertansiyon, ayrıca öyküsünde mesane karsinomu nedeniyle cerrahi mevcuttu. Muhtemelen kanserin kendisi ve cerrahisi tarafından kolaylaştırılan ciddi hiperkoagülan durum çok damar koroner ve periferal yoğun trombüs yüküne neden oldu. Revaskülarizasyon ve trombektomi ameliyatı yapılmadan önce intrakoroner 10 mcg / kg tirofiban bolus ve 15 mg doku plasminojen aktivatörü (tPA) sırasıyla uygulandı. Tam revaskülarizasyon elde edildi.

Anahtar kelimeler: Mesane kanseri, Miyokard enfarktüsü, Periferik iskemi, Tromboz

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Introduction

The relationship between cancer and thrombosis is defined obviously [1]. Thromboembolism including venous and arterial events is one of most common cause of death in cancer patients [2,3]. The underlying mechanisms are not exactly understood [4]. In this report, we present a case with extensive multivessel coronary thrombotic occlusion concomitant with acute peripheral thrombotic occlusion in a patient with bladder carcinoma and discussed the potential underlying mechanism.

Case Presentation

A 68 years old man admitted to our hospital with the complaint of chest pain and new onset severe left leg pain. He had smoking and hypertension as cardiovascular risk factors. He had an operation because of bladder cancer and prostate hypertrophy one week ago. No adjuvant therapy had been required. In immunohystochemical analysis of cancer tissue p53 (+) Ki67 (+) papillary urothelial carcinoma had been detected. In emergency room blood pressure was 75/35mmHg, heart rate 80bpm, and O2 saturation was 87%. In electrocardiogram 5mm ST segment elevation in leads II, III, aVF and V4-V5-V6 and ST segment depression in leads V1-V2, aVL was detected. Bilateral rales in the inferior zones of lungs was heard by auscultation. Bilateral femoral pulses were palpable but left popliteal and dorsalis pedis pulses were nonpalpable. In laboratory parameters, wbc 27000/µl, hb 12 g/dl, plt 196000/µl, troponin

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>50000ng/ml, CK-MB 162u/l, creatinine 1.34 mg/dl, urea 52 mg/dl were detected. After 600 mg clopidogrel loading dose administration, coronary angiography was performed. Coronary angiogram showed 80% proximal left anterior descending artery (LAD) lesion and mid LAD occluded with intense thrombus burden and 100% occlusion of well-developed circumflex-obtuse marginal branch (CX-OM) with intense thrombus burden as well (figure1A, 1B). A floppy guidewire was advanced to CX-OM lesion and provided TIMI 3 flow and intracoronary 10 mcg/kg tirofiban bolus and 15 mg tPA were administered respectively after showing thrombus existence angiographically. Shortly after coronary reperfusion, blood pressure increased to 110/70mmHg. Bare metal stent (2.75x24 mm) implanted into CX-OM lesion. Then LAD lesion was advanced by floppy guidewire and 2.0x20 mm balloon was inflated. And then 4.0x20 mm bare metal stent was implanted. TIMI 3 flow is provided for both lesions (figure 2A). After that right coronary artery (RCA) was cannulated by JR4 catheter and %100 occlusion was seen on proximal RCA (figure 1C). A floppy guidewire was advanced to RCA and thrombectomy was performed. After thrombectomy bare metal stent (3,0x32 mm) was implanted and postdilatation with 3,5x10 NC balloon was performed successfully. TIMI 3 flow was provided (figure 2B). Vital values of patient improved. The patient was taken to coronary care unit (CCU) and tirofiban infusion administered but the left leg pain of patient proceeded.

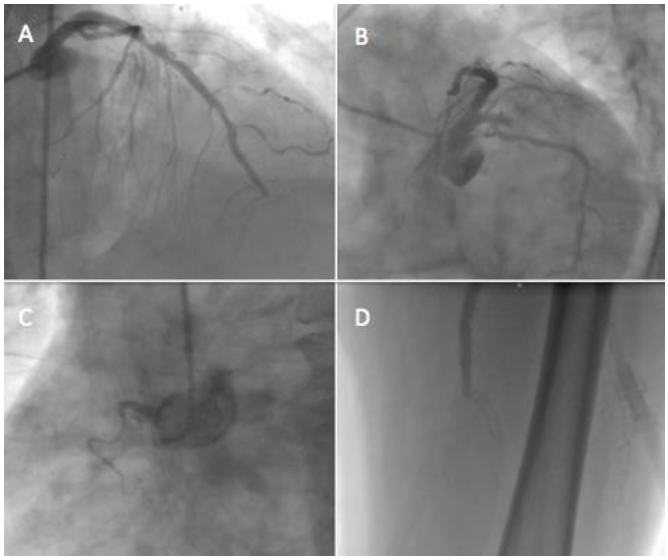

Figure 1: Coronary angiography showed (A) Critically proximal left anterior descending artery (LAD) lesion and mid LAD thrombotic occlusion. (B) Proximal Circumflex coronary artery (Cx) thrombotic occlusion. (C) Right coronary artery (RCA) proximal thrombotic occlusion. (D) Thrombotic occlusion in left superficial femoral artery.

A peripheral angiogram (PAG) showed chronic total occlusion in right superficial femoral artery and a 100% occlusion with intense thrombus burden in left superficial femoral artery and left arteria profunda femoralis (figure 1D). The patient was consulted with cardiovascular surgeons. Surgical thrombectomy was preferred because of the necessary peripheral interventional materials. Thrombectomy operation was planned and performed successfully. No complication was observed after operation. His coagulation profile showed normal results. The patient discharged with fully relieved symptoms.

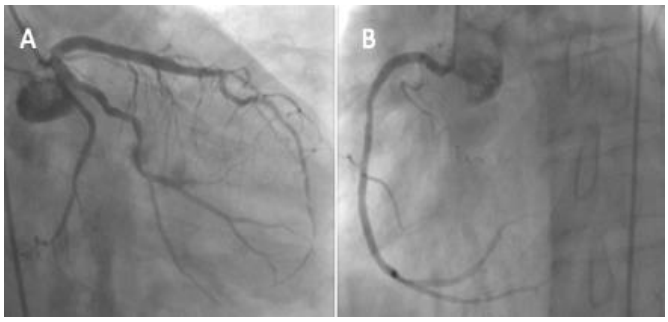

Figure 2: Post-revascularization (A) Left anterior descending artery (LAD) and Circumflex coronary artery (Cx) image. (B) Right coronary artery (RCA) image.

Discussion

Cancer and its treatments such as chemotherapy, immunotherapy, hormone therapy, radiotherapy and surgery are often complicated by thromboembolism and cardiovascular events [5]. In patients with cancer, venous thromboembolism including deep venous thrombosis and pulmonary embolism, arterial thromboembolism including stroke, myocardial infarction and peripheral thrombosis are also more common [4]. The etiology of prothrombotic status cannot explain one particular mechanism, but it clarifies presumably multifactorial [4,6]. The expression of tissue factor (TF) by procoagulant tumor or stromal cells is especially blaming factor. In this pathway, TF expressed by tumor can directly prompt factor X; TF released by monocytes or macrophages can activate factor VII [2]. Another mechanism of hypercoagulability is inflammatory response such as cytokine release, acute phase reaction induced by tumor cell interactions with endothelial cells and macrophages. Tumor necrosis factor (TNF) and interleukin-I procoagulant role was proved in some studies [7]. Some solid tumors, such as renal cell carcinoma, can stimulate coagulation cascade due to vessel wall injury [1]. Out of these factors, cancer treatments can also enhance tendency of thrombosis even if they improve cancer free survival [7]. Cardiovascular events such as heart failure, myocardial infarction and stroke are undesired result of treatment [5].

In our case severe hypercoagulable condition probably facilitated by cancer itself and surgery. Although the relationship between cancer and thrombosis was clearly shown, the relationship between the type of cancer and thrombosis is conflicting. An analysis conducted by Nalluri et al [8], shown that solid tumors are more prone to develop thrombosis. Pancreas, brain, prostate cancers more than other types of tumors are known to be at higher risk of the development of thrombosis [9]. Although a study reported that bladder cancers are relatively small proportion of cancers associated with thrombosis, contrary to this data Chew and colleagues reported higher risk of deep vein thrombosis in patients with bladder cancer [10,11]. In another study conducted by Sandhu et al [12], reported that higher risk of thrombosis in bladder cancer especially if the patient is older and male. Villemur et al [13] reported occluded with thrombus bypass graft in three patient with cancer including one is bladder, and concluded that bypass grafts are more prone to thrombosis in patients with cancer. In another case Kanemaru et al, reported a male with bladder cancer with carotid stent thrombosis despite regular use of antiplatelet therapy [14]. In our

case, patient was a 68 years old male and was operated one week ago. Besides being male patients of advanced age, it is known that the history of operation is another factor for increasing the risk of thrombosis development. However, in our case femoral artery thrombosis as well as coronary arteries signifies a systematic tendency to develop thrombosis as well as local factors. Referring to our case we conclude that bladder cancer could be more associated with thrombosis as well as prostate, pancreas and brain cancer. Patients with bladder cancer especially who have atherosclerotic risk factors such as advanced age, male gender, diabetes mellitus, it should not be forgotten to develop acute coronary syndrome or systemic thrombosis. These patients should be detailed questioned about acute coronary syndrome or cardiovascular disease and physical examination and electrocardiograms should be investigated.

In patients with cancer thromboembolic complications secondary to the hypercoagulable clinical condition increase both morbidity and mortality. We should be aware of thromboembolic situations and we should also consider using antithrombotic prophylaxis for these patients.

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