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Acute heart failure after kidney tumor embolization. A case report

Ostra niewydolność serca po embolizacji guza nerki. Opis przypadku

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ABSTRACT

Renal cell carcinoma (RCC) is the most common primary malignant renal tumor. The classic triad of symptoms includes palpable abdominal mass, hematuria and flank pain. However, some of the patients diagnosed with RCC first present with paraneoplastic symptoms occurring as a consequence of secretion of various substances from the tumor. We present a case of a woman diagnosed with large kidney tumor, who developed acute heart failure soon after being treated with percutaneous embolization. We discuss toxic myocarditis as one of the possible causes for the acute hemodynamic compromise and the fatal outcome of the patient. Our case report highlights the complexity of this pathology and the importance of awareness of the various possible life-threatening complications.

Key words: renal cell carcinoma, percutaneous embolization, acute heart failure, toxic myocarditis

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STRESZCZENIE

Rak nerkowo-komórkowy jest najczęstszym pierwotnym nowotworem złośliwym nerki. Klasyczna triada objawów z nim związanych obejmuje guz w jamie brzusznej, krwiomocz i ból w okolicy lędźwiowej. Jednak niektórzy pacjenci z rakiem nerkowo-komórkowym najpierw mają objawy paraneoplastyczne wywoływane przez substancje produkowane przez guz. W pracy przedstawiono przypadek kobiety, u której zdiagnozowano duży guz nerki i u której bezpośrednio po jego embolizacji rozwinęły się objawy ostrej niewydolności serca. Jako jedną z możliwych przyczyn ostrych zaburzeń hemodynamicznych i zgonu pacjenta rozważono toksyczne zapalenie mięśnia sercowego. Przedstawiony opis przypadku podkreśla złożoność patologii, jak również potrzebę świadomości różnych możliwych zagrażających życiu powikłań związanych z jego leczeniem.

Słowa kluczowe: rak nerkowo-komórkowy, przezskórna embolizacja, ostra niewydolność serca, toksyczne zapalenie mięśnia sercowego

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Introduction

Renal cell carcinoma (RCC) is the most common primary tumor of the kidney. Approximately 30% of patients with this neoplasm present with one of the following symptoms — hematuria, palpable abdominal mass and flank pain. However, in about 20% the first presentation of the malignancy is paraneoplastic symptom [1, 2]. The paraneoplastic syndrome consists of various systemic manifestations. It is a result of release of various tumor-associated humoral substances that are produced by the tumor itself or by other tissues via immune-modulation mechanisms [3]. Herein, we present a case of a 40-year-old female patient who developed acute heart failure after percutaneous embolization of kidney tumor and discuss the potential etiologies of the hemodynamic decompensation.

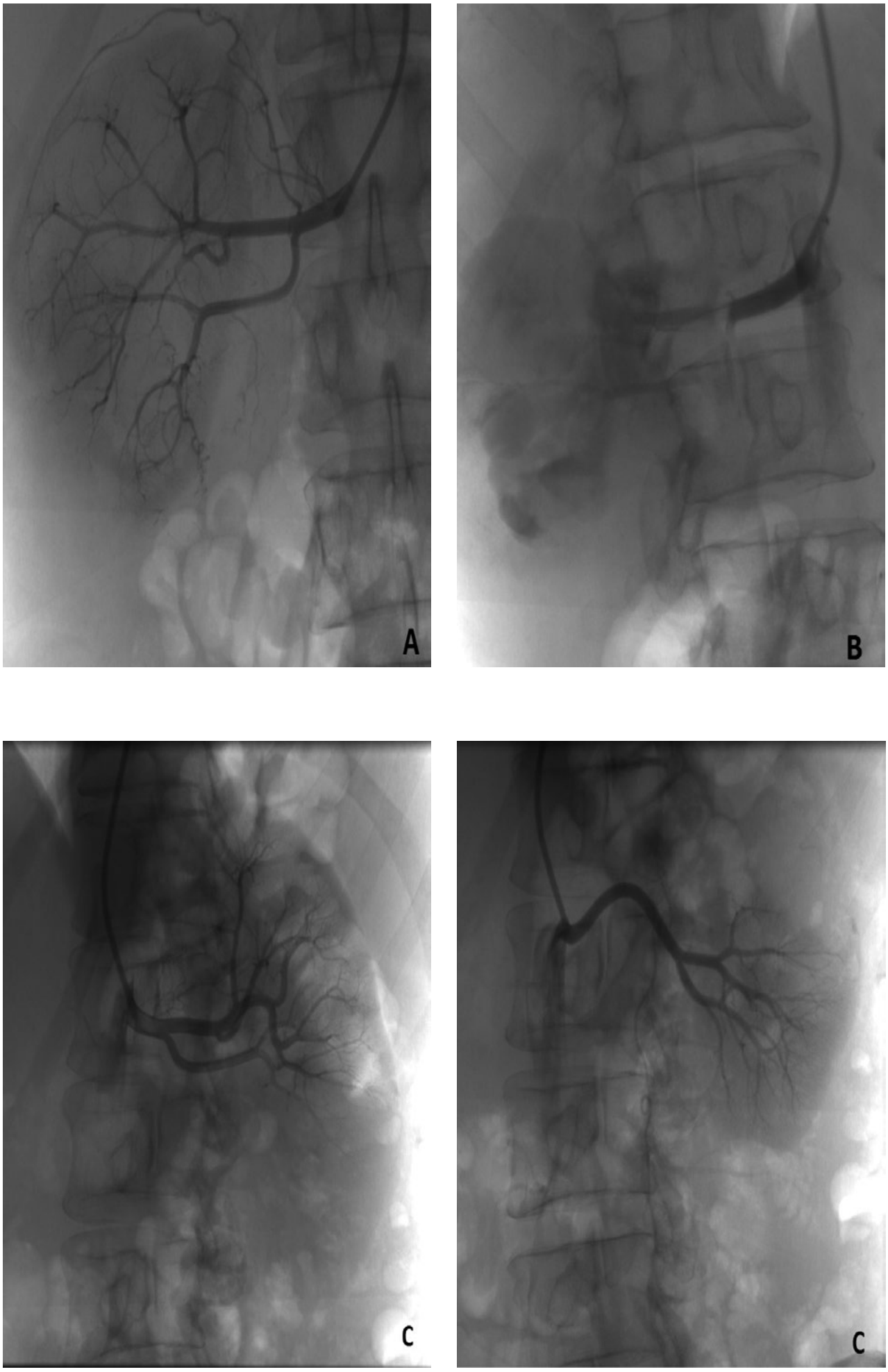

Figure 1. Renal angiography. **A.** Right renal artery before the embolization; **B.** Blood flow after embolization of right renal artery with 1000 mcm and 1500 mcm microspheres; **C.** Left renal artery and accessory inferior renal polar artery with normal blood flow

Case report

A 40-year-old Caucasian female patient was referred to our department for percutaneous embolization of large kidney tumor. Computed tomography (CT) scan, performed two weeks before the admission, revealed large tumor originating from the right kidney with infiltration of the liver and inferior vena cava. Histology result from tru-cut biopsy was consistent with a clear cell renal cell carcinoma. On admission the patient's vital signs included heart rate of 70 bpm, arterial blood pressure of 130/75 mmHg, body temperature of 37.1°C, respiratory rate of 15 breaths per min. Her airway, lung, and heart examinations were unremarkable. The initial electrocardiogram showed normal sinus rhythm at 75 bpm; laboratory results were with no clinically significant abnormalities. Echocardiography before the intervention showed normal systolic function, ejection fraction (EF) of 55%, and normal kinetics of the left ventricle with mild hypertrophy.

On the second day after the admission the patient underwent selective embolization of the right renal artery. We used 1000 mcm and 1500 mcm microspheres to achieve complete blood flow stasis (Figure 1). The procedure went uneventfully without any complications. After the intervention the woman complained of nausea, vomiting and abdominal pain, which was considered to be part of post-embolization syndrome. She was treated with opioid analgesics, antibiotics, crystalloid fluid infusions and stayed hemodinamically stable.

However, within the next 24 hours there was sudden deterioration of the patient's condition. She complained of severe dyspnea and developed

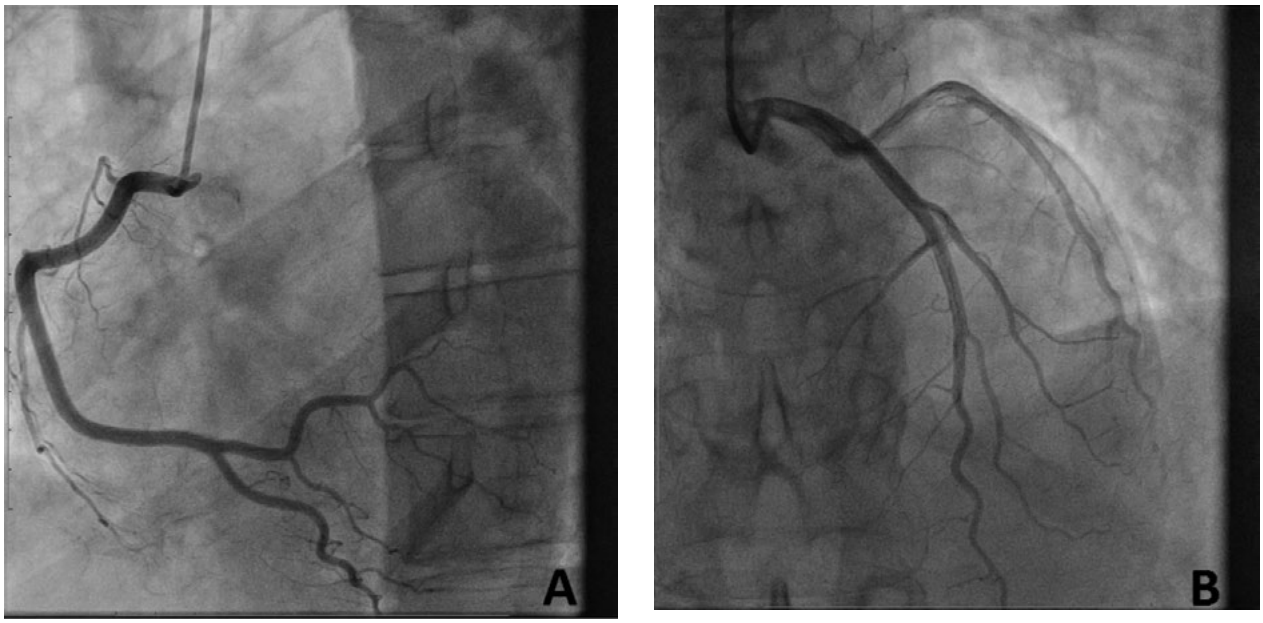

Figure 2. Coronary angiography revealing coronary vessels with no significant stenosis. **A.** Left anterior oblique view of the right coronary artery; **B.** Right anterior oblique view of the left anterior descending (LAD) and left circumflex (LCx) artery

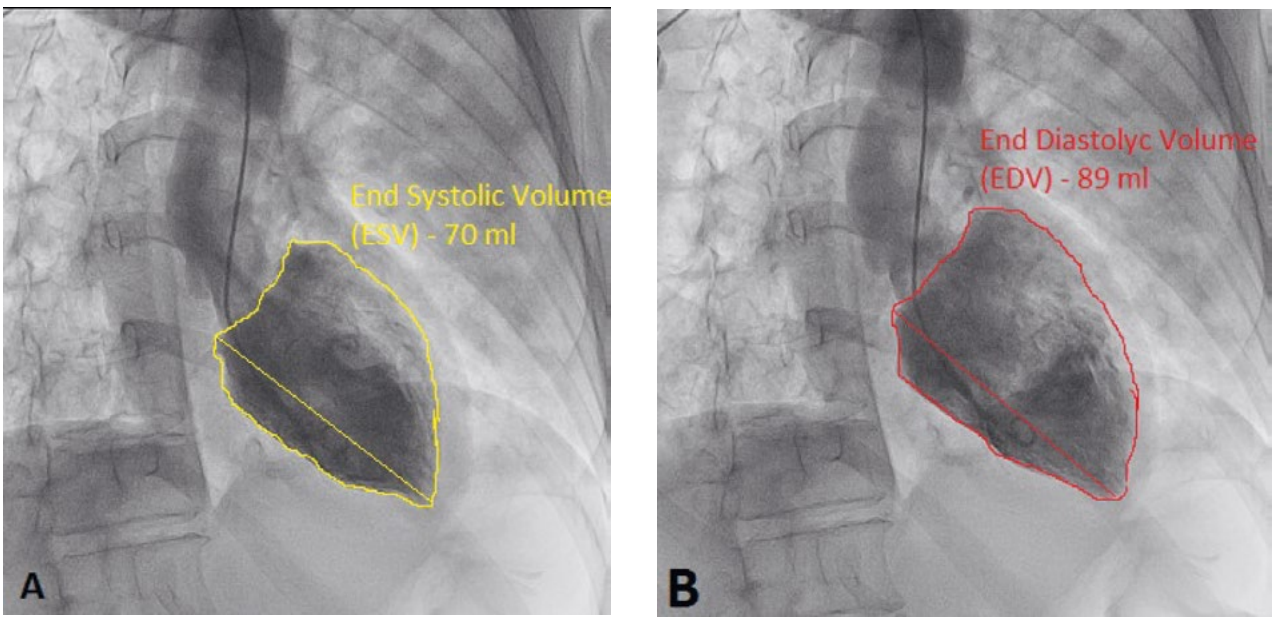

Figure 3. Left ventricle angiography. **A.** Systole; **B.** Diastole

pulmonary edema. Echocardiography revealed left ventricular systolic dysfunction with EF 20% and diffusely hypokinetic left ventricle. The laboratory results showed significant leukocytosis with white blood cell count of 52 G/l, increased creatinine, blood urea nitrogen and liver enzymes. However, there was adequate urine output (60 ml/h). The cardiac biomarkers also showed significant increase — creatine phosphokinase (CPK) 4542 (reference range: 20–180) U/L; MB fraction 143 (reference range < 25) U/L; high sensitive Troponin T 0,522 (reference range: < 0.014) ng/ml.

Urgently, the patient was brought to the catheterization lab and underwent invasive coronary angiography. The study did not reveal any significant coronary stenosis (Figure 2). Left ventricle angiography confirmed the severe left ventricular systolic dysfunction (Figure 3). For further evaluation of the patient's pathology pulmonary artery angiography was performed. There was no evidence of pulmonary hypertension or embolism. During the invasive procedure the patient decompensated with arterial blood pressure of 90/60 mmHg. Intra-aortic balloon pump was inserted in 1:2 mode. Soon afterwards the patient was intubated with subsequent mechanical ventilation support. Despite the therapy with diuretics, antibiotics and catecholamine infusion, there was no improvement in patient's condition and she died on the second day after the embolization.

Discussion

There are several cases reported of cardiovascular complications in patients who underwent percutaneous embolization of different types of tumors. Reports of rhythm and rate disturbances, as well as hypertensive crisis are described in the literature [4, 5]. Furthermore, there are several cases described of patients who developed acute heart failure soon after percutaneous embolization or ablation [6].

One of the probable mechanisms of the acute hemodynamic compromise after such invasive procedure is stress-induced cardiomyopathy [7]. Also known as Takotsubo syndrome it occurs as a result of increased sympathetic tone and excessive release of catecholamines — epinephrine, norepinephrine and dopamine. Women and especially middle aged are considered to be the most commonly affected [8]. This syndrome is associated with reversible systolic dysfunction, segmental contractile disturbance and electrocardiographic changes and it can develop in patients without any primary heart disease [9, 10].

Another possible cause for the acute hemodynamic decompensation after tumor embolization is the development of fulminant toxic myocarditis as a consequence of the tumor lysis syndrome. We believe that this is the most probable cause for the myocardial injury in our patient. The lysis of cancer cells after embolization leads to excessive release of intracellular proteins, nucleic acids, uric acid and electrolytes into the circulation [11]. What is more, it is possible that the renal cell carcinoma played a direct causative role via paraneoplastic systemic effects. It has been reported that clear cell renal carcinoma tumor cells secrete various proteins that serve as mediators for paraneoplastic symptoms — parathyroid hormone-related protein, erythropoietin, interleukin 6, vascular endothelial growth factor and others [12]. All these substances are associated with systemic manifestations such as cachexia, fever, polycythemia, and hypercalcemia and they may have been part of the pathogenesis of fulminant toxic myocarditis.

Conclusion

Nowadays, embolization of kidney tumors is a procedure performed preoperatively or a standard therapeutic option in patients contraindicated for surgery. However, interventions on kidney neoplasms can lead to various secondary complications. Such patients are challenging cases and their treatment demand experience. We have described a rare case of a woman with acute heart failure due to fulminant toxic myocarditis in the context of angiographic embolization of a large clear cell renal cell tumor. Knowledge of this association accentuates the importance of awareness of the different cardiovascular complications and the necessity of continuous monitoring of this kind of patients.

Disclosures

None

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