Cardiac metastasis of urothelial carcinoma mimicking ST-elevation myocardial infarction

Srdeční metastázy uroteliálního karcinomu napodobující infarkt myokardu s elevací ST

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Summary

Background: Urothelial carcinoma (UC) is the second most common malignancy of the urinary tract. While distant metastases are relatively frequent in advanced UC, cardiac involvement is exceedingly rare and typically asymptomatic. When present, it may mimic acute cardiovascular events, complicating timely diagnosis and management. ***Case:*** We describe the case of an 81-year-old man with a history of metabolic syndrome and stable cardiac disease, who was diagnosed with high-grade urothelial carcinoma of the renal pelvis with pulmonary metastases. Two months after nephroureterectomy and during first-line carboplatin chemotherapy, the patient presented with progressive dyspnoea. ECG showed ST-segment elevations suggestive of acute myocardial infarction, yet urgent coronary angiography and laboratory markers excluded acute coronary syndrome. Echocardiography and cardiac MRI revealed a large myocardial metastasis infiltrating the right ventricle and interventricular septum. Given the advanced stage and poor prognosis, active oncologic treatment was discontinued, and palliative care was initiated. ***Results:*** This case highlights a rare presentation of cardiac metastasis mimicking ST-elevation myocardial infarction in a patient with advanced urothelial carcinoma. Multimodal cardiac imaging was key to establishing the diagnosis. Despite its rarity, myocardial infiltration should be considered in oncologic patients presenting with cardiac symptoms and ECG abnormalities. ***Conclusion:*** Cardiac metastases from urothelial carcinoma are rare but clinically significant, often associated with poor outcomes. ECG changes resembling myocardial infarction warrant thorough evaluation, particularly in patients with known malignancy. Early diagnosis, even in the absence of curative options, may help guide appropriate palliative care.

Key words

urothelial carcinoma – heart neoplasms – neoplasm metastasis – electrocardiography – multimodal imaging – myocardial infarction mimicry

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Souhrn

Východiska: Uroteliální karcinom (UC) je druhým nejčastějším zhoubným nádorem močového traktu. Distanční metastázy jsou u pokročilého onemocnění relativně časté, ale postižení srdce je vzácné a obvykle asymptomatické. Pokud k němu dojde, může imitovat akutní kardiovaskulární příhody, což ztěžuje diagnózu. ***Kazuistika:*** Popisujeme případ 81letého muže s anamnézou metabolického syndromu a stabilního kardiovaskulárního onemocnění, u kterého byl diagnostikován high-grade uroteliální karcinom pánvičky pravé ledviny s plicními metastázami. Dva měsíce po nefrektomii a během 1. linie chemoterapie karboplatinou se pacient dostavil s progresivní dušností. EKG ukazovalo elevace ST úseku, naznačující akutní infarkt myokardu, avšak akutní koronární syndrom byl vyloučen pomocí koronarografie a laboratorních markerů. Echokardiografie a magnetická rezonance srdce odhalily objemnou metastázu infiltrující pravou komoru a mezikomorovou přepážku. Vzhledem k pokročilému stadiu a špatné prognóze byla onkologická terapie ukončena a pacient byl předán do péče paliativního týmu. ***Výsledky:*** Případ ilustruje vzácnou manifestaci srdeční metastázy imitující infarkt myokardu s elevací ST u pacienta s pokročilým UC. Klíčovou roli v diagnostice sehrálo multimodální kardiologické zobrazování. I přes nízkou incidenci je u onkologických pacientů s kardiálními symptomy a změnami na EKG nutné pomýšlet na možnost srdeční infiltrace. ***Závěr:*** Srdeční metastázy u uroteliálního karcinomu jsou vzácné, ale klinicky závažné a často znamenají velmi nepříznivou prognózu. EKG nález napodobující infarkt myokardu vyžaduje pečlivé zhodnocení, zvláště u pacientů s malignitou. Včasné rozpoznání může napomoci adekvátnímu nastavení paliativní péče.

Klíčová slova

uroteliální karcinom – srdeční nádory – metastázy novotvaru – elektrokardiografie – multimodální zobrazování – imitace infarktu myokardu

Introduction

Urothelial carcinoma is the second most common malignancy among urological cancers. Histologically, it originates in the upper urinary tract (including the renal pelvis and ureter) and the lower urinary tract (including the bladder and urethra) [1]. The upper urinary tract is involved in 5–10% of cases, with the renal pelvis being affected much more frequently than the ureter [2]. The peak age for diagnosis in both males and females is in the sixth and seventh decades [2].

Approximately 10% of patients have distant metastases at the time of diagnosis [3]. The most common sites of distant metastasis are lymph nodes, lungs, bones, and liver [3]. Rarely, upper tract urothelial carcinoma can spread to less common sites of metastasis such as the cardiac muscle. Cardiac metastases present rare but serious complications for patients with advanced disease. They are clinically asymptomatic in most cases. Here, we report a rare case of cardiac metastasis that initially manifested as mimicking acute coronary syndrome.

Case report

We present the case of an 81-year-old man with a history of metabolic syndrome, dyslipidemia, and arterial hypertension. He had been under long-term cardiology follow-up due to a previous posterolateral myocardial infarction and paroxysmal atrial fibrillation, which had been successfully treated with electrical cardioversion, resulting in stable sinus rhythm.

The patient was initially admitted to the urology department with macroscopic hematuria. Diagnostic cystoscopy revealed no significant abnormalities apart from bleeding from the anterior commissure of the prostate. Cytological analysis was negative for malignant cells, and the bleeding was attributed to benign prostatic hyperplasia. The patient's symptoms resolved with 5-alpha-reductase inhibitor therapy.

Six months later, he re-presented with recurrent hematuria accompanied by clot formation and unintentional weight loss. A contrast-enhanced CT urography revealed a mass in the lower pole of the right kidney, along with retroperitoneal lymphadenopathy and suspicious pulmonary lesions, suggestive of metastatic disease. He subsequently underwent robot-assisted nephroureterectomy with lymphadenectomy.

Histopathological examination confirmed a diagnosis of high-grade urothelial carcinoma of the renal pelvis (G3), with invasion into the renal parenchyma. The tumour was staged as pT4N1M1, corresponding to stage IV disease with confirmed visceral (pulmonary) metastases. A staging PET/CT scan was performed before the initiation of systemic therapy.

Two months postoperatively, the patient began first-line chemotherapy with carboplatin monotherapy, which was well tolerated initially. However, at a scheduled oncology follow-up after the third cycle, he reported progressive shortness of breath over several days, culminating in dyspnoea at rest.

On examination, he was hemodynamically stable, normotensive, and adequately oxygenated, with no clinical signs of heart failure. An ECG performed in the oncology clinic revealed significant ST-segment elevation and T-wave inversions in leads II, III, aVF, and V2–V6, with Q waves in lead III and additional T-wave inversions in V1; these abnormalities had not been present in an earlier ECG obtained 20 months before (Fig. 1). Based on these findings, the patient was urgently referred to the coronary care unit with a working diagnosis of antero-inferolateral ST-elevation myocardial infarction.

However, urgent coronary angiography did not reveal any acute coronary occlusion or significant stenosis. Initial laboratory investigations showed a high-sensitivity troponin I (hsTnI) level of 18.1 ng/L, which falls within the normal range (0.0–34.2 ng/L) and is well below the myocardial infarction diagnostic cut-off (342 ng/L for men, 156 ng/L for women). Creatine kinase (CK) activity was 0.90 µkat/L (reference range: 0.41–3.24 µkat/L), and N-terminal pro-brain natriuretic peptide fragment (NT-proBNP) was elevated at

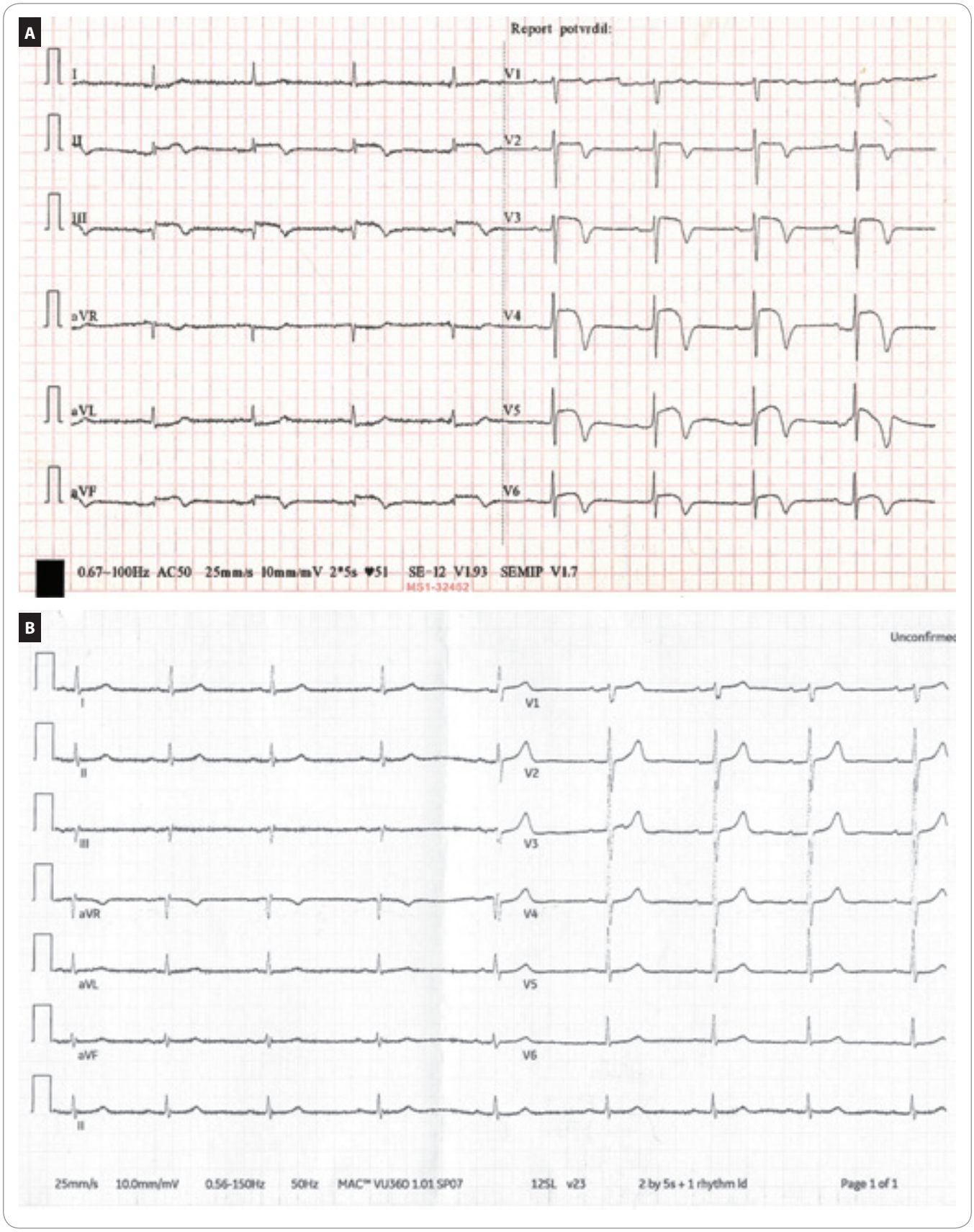

Fig. 1. Electrocardiograms of the patient. (A) ECG of the patient on presentation with dyspnoea (as described in the text), showing ST--elevation, compatible with ST-elevation myocardial infarction; (B) ECG obtained 20 months earlier, showing only sinus bradycardia.

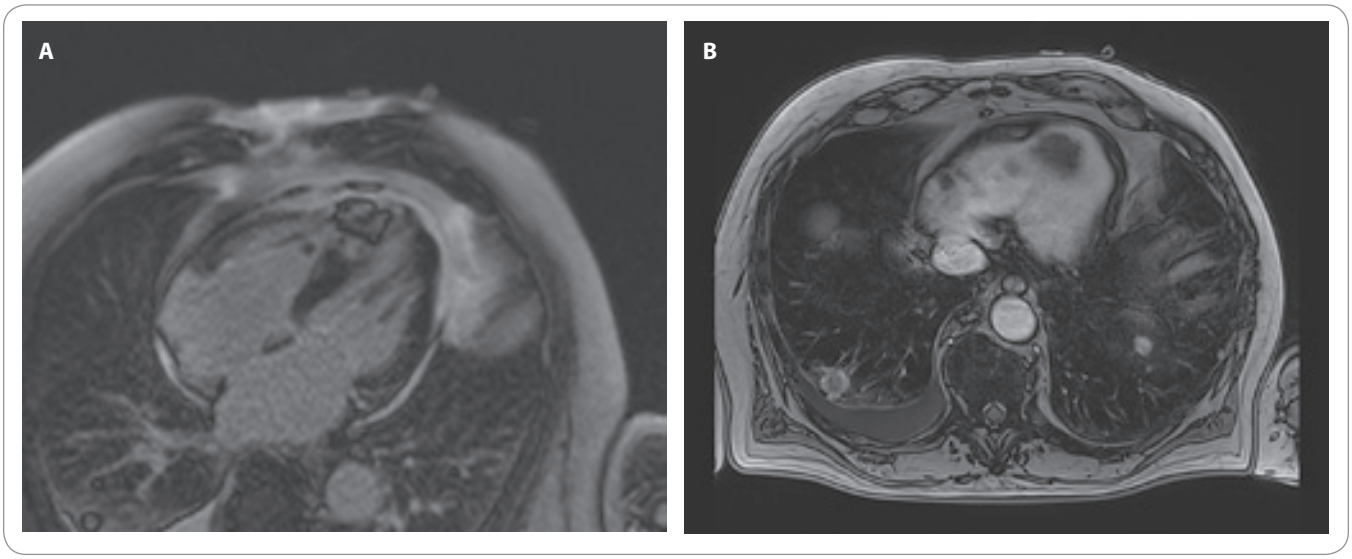

Fig. 2. Selected cardiac MRI images demonstrating the apically located metastasis. (A) Late gadolinium enhancement (LGE) scan showing involvement of the apex; (B) T1-weighted, axial (transverse) 3D GRE Dixon image (also showing lung metastases).

1986.7 ng/L (normal: 35.0–450.0 ng/L). These findings did not support the diagnosis of an acute coronary syndrome.

Transthoracic echocardiography subsequently identified a heterogeneous mass infiltrating the interventricular septum and free wall of the right ventricle, with signs of neovascularization on myocardial contrast echocardiography, raising suspicion for cardiac metastasis. This was confirmed by cardiac MRI, which demonstrated a centrally necrotic myocardial metastasis measuring 40 × 50 mm, protruding into the apex of the right ventricle, infiltrating the interventricular septum, and involving the papillary muscles of the right ventricle (Fig. 2).

Given the advanced disease stage, rapid progression, and limited therapeutic options, a multidisciplinary team decision was made to discontinue active oncologic therapy. The patient was transitioned to symptomatic management, and care was transferred to a specialist palliative care team.

Discussion

Cardiac metastases are generally considered a rare complication of malignancy, yet they are significantly more common than primary cardiac tumours. Postmortem studies have shown that primary cardiac neoplasms occur with a low incidence of 0.001–0.28%, whereas cardiac metastases are more frequently identified, with reported incidences ranging from 2.3% to 18.3% depending on tumour type and study population [4–6]. Certain malignancies, such as lung cancer, melanoma, and breast cancer, exhibit a higher propensity for cardiac involvement.

Among the cardiac structures, the pericardium is most frequently affected by metastases, while the endocardium is least commonly involved. Endocardial metastases tend to localize in the right ventricle and are associated with malignancies that demonstrate intravascular growth patterns, such as renal cell carcinoma and hepatocellular carcinoma [4–9].

A comprehensive study by Bussani et al. in 2007 analyzed 7,289 autopsies of patients with malignant disease and found cardiac metastases in 662 cases (9.1%). Lung adenocarcinoma was the most common primary source (97 cases, 14.6% of all cardiac metastases). However, the highest proportional incidence of cardiac involvement was observed in mesothelioma (48.8% of cases), followed by melanoma (27.8%) and lung adenocarcinoma (21%). Of 307 patients with urothelial carcinoma, cardiac metastases were identified in 12 cases (3.9%), accounting for 1.8% of all metastases to the heart. However, the authors defined cardiac metastasis broadly, including not only involvement of the pericardium, myocardium, and endocardium, but also the great vessels, coronary arteries, and any intracavitary tumour thrombi [4].

While the exact mechanisms behind cardiac metastasis remain incompletely understood, lymphatic spread is believed to play a critical role. The heart's lymphatic system consists of subendocardial, myocardial, and subepicardial networks, with lymph flowing outward toward the mediastinal lymph nodes. Cardiac contractions may exert opposing effects: promoting lymphatic drainage (which may hinder tumour spread) but also facilitating tumour cell dissemination along the epicardium [9].

The clinical manifestations of cardiac metastases are variable and range from asymptomatic incidental findings to life-threatening complications such as acute heart failure or cardiac tamponade. Pericardial involvement may cause pericardial effusion, while myocardial infiltration often results in arrhythmias, most frequently atrial fibrillation or flutter. Less commonly, bradyarrhythmia such as AV block or malignant ventricular arrhythmias may occur. In rare cases, acute myocardial infarction may result from tumour-related coronary artery compression or thrombosis. Metastases involving the ventricles may predispose to embolization, adding to the risk of systemic complications [4–9].

In our patient, electrocardiographic findings mimicked an acute myocardial infarction, with ST-segment elevation. However, this was excluded by normal cardiac enzymes and negative coronary angiography. Similar cases of ST elevation in cardiac metastasis are rarely reported. Although the precise mechanism remains unclear, ST-segment changes may serve as an important diagnostic clue and highlight the need for further evaluation. Nevertheless, the presence of ST elevation should not lead clinicians to prematurely dismiss the possibility of acute coronary syndrome, which remains a common and life-threatening condition.

Cardiac metastasis from urothelial carcinoma typically indicates aggressive disease with very poor prognosis. A series by Hattori et al. described seven cases of cardiac metastases from renal pelvis carcinoma, with uniformly poor survival [10]. These findings underscore the devastating impact of cardiac involvement.

Conclusion

Although rare, cardiac metastases in urothelial carcinoma represent a critical clinical entity associated with rapid deterioration. ECG abnormalities and clinical symptoms may aid in early recognition. Although standardized guidelines are lacking, early symptomatic treatment remains the mainstay of care and may contribute to improved quality of life, even in the absence of disease-modifying therapies.

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