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Case report

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# Hypertensive Pulmonary Edema, COVID Pneumonia, Old Myocardial Infarction, and Graded Q-Waves Resolution in a Resected Urothelial Carcinoma in Elderly; Risk and Management

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## Abstract

**Rationale:** Hypertensive pulmonary edema is a lethal consequence of hypertensive emergency. It is considered one of the most serious complaints in the emergency room for hypertensive patients. There is an intense relationship between COVID-19 infection and coronary artery disease. The fatality of COVID-19 pneumonia may contribute to the associated pulmonary edema. The management of acute myocardial infarction (AMI) in cancer patients has significant challenges. Pathological Q waves usually indicate current or prior myocardial infarction.

**Patient concerns:** Elderly married male retired officer, heavy smoker Egyptian patient was admitted to the intensive care unit with hypertensive pulmonary edema, COVID-19 pneumonia, and old myocardial infarction in a resected left urothelial carcinoma.

**Diagnosis:** Hypertensive pulmonary edema, COVID pneumonia, old myocardial infarction, and graded Q-waves resolution in a resected urothelial carcinoma in the elderly.

**Interventions:** Electrocardiography, oxygenation, biopsy, CT scan, and echocardiography.

**Outcomes:** A dramatic clinical and electrocardiographic improvement happened.

**Lessons:** The urothelial carcinoma may be contributing to acute myocardial infarction. The graded Q-waves resolution in the ECG over the years of follow-up supports that the Q-waves may be transient. Stabilization and non-metastasis of urothelial carcinoma over four years may be a good sign. The associated and idiopathic sporadic RBBB beats are interesting. Elderly, male sex, heavy smoker, hypertensive, pulmonary edema, COVID-19 pneumonia, old myocardial infarction, urothelial carcinoma, and marked elevated Pro B-type natriuretic peptide are the current constellation risk factors.

**Keywords:** Hypertensive crises; urothelial carcinoma; malignancy; hypertensive pulmonary edema; ischemic heart disease; COVID-19 pneumonia; Q-Waves

**Abbreviations:** ACS: Acute Coronary Syndrome; AMI: Acute Myocardial Infarction; CAS: Coronary Artery Spasm; CBC: Complete Blood Count; COVID-19: Coronavirus Disease 2019; CTO: Chronic Total Occlusion; ECG: Electrocardiography; ICU: Intensive Care Unit; IHD: Ischemic Heart Disease; LAD: Left Axis Deviation; NSR: Normal Sinus Rhythm; MI: Myocardial Infarction; O2: Oxygen; PCI: Percutaneous Coronary Intervention; PVC: Premature Ventricular Complex; RBBB: Right Bundle Branch Block; VR: Ventricular Rate



## Introduction

Urothelial carcinoma (UC) is a urinary tract carcinoma, mainly your bladder and kidneys. UC is the second most common urological malignancy in developed countries. They can be localized in the bladder, urethra pyelocaliceal cavities, and ureter. Bladder cancer (BC) represents 90–95% while upper tract urothelial carcinomas (UTUC) only 5–10% of UCs. Smoking and aristolochic acid are the main risk factors for upper tract urothelial carcinomas (UTUC) [1]. The spectrum of urothelial cancer at presentation includes non-muscle invasive, muscle-invasive, and metastatic disease [2]. Diagnosis of UTUC may be incidental or symptom related. Flank pain can occur in 20–32% of cases. Computed tomography (CT) urography has the highest diagnostic accuracy of the available imaging techniques [1]. Kidney-sparing management should be offered as a primary treatment option for patients with low-risk UTUC. Platinum-based chemotherapy after radical nephroureterectomy and adjuvant nivolumab for patients resistant to chemotherapy are options in a high-risk UTUC [3].

Hypertensive crises with acute elevations in blood pressure that are associated with end-organ damage such as myocardial infarction, cerebrovascular accident, pulmonary edema, or renal failure are defined as a hypertensive emergency [4]. Hypertensive crises represent the most immediate danger to those afflicted and the most dramatic proof of the lifesaving potential of antihypertensive therapy [5]. Hypertensive crises are present when markedly elevated blood pressure or > 180/120 mmHg are common issues in the emergency department [6,7]. Prompt diagnosis, based primarily on signs and symptoms is essential. Appropriately aggressive therapy will often result in a satisfactory outcome [7]. There are multiple potential mechanisms leading to pulmonary edema in severe Coronavirus Disease (COVID-19) patients. The abnormal humoral metabolism and pulmonary edema contribute to the severity of symptoms and fatality of COVID-19 patients. Focusing on decreasing the formation of body fluid in the lung or promoting the absorption of body fluid can contribute to a decrease in lung damage and decreased mortality in COVID-19 patients [8]. Myocardial infarction (MI) is a common cardiovascular disease and a worldwide leading cause of death.

The etiology of MI is complicated and not completely understood. Lifestyle factors, environmental factors, psychosocial factors, and genetic factors are some implicated risk factors. Identifying the risk of MI is highly significant for secondary prevention [9]. Cancer and cardiovascular disease are the major causes of morbidity and mortality. There is a shared risk factor between cancer and cardiovascular disease. The management of acute myocardial infarction (AMI) in cancer patients poses significant challenges, especially due to comorbidities [10]. Ischemic symptom improvement is the primary indication for chronic total occlusion (CTO) percutaneous coronary intervention (PCI) or CTO-PCI [11]. Two published randomized, controlled clinical trials [12,13] and several observational studies showed symptomatic improvement after post-CTO-PCI [14]. The Q-wave represents the normal left-to-right depolarization of the interventricular septum. Pathological Q

waves usually indicate current or prior myocardial infarction [15]. In asymptomatic patients, the presence of abnormal Q waves on an electrocardiogram indicates a high probability of ischemic heart disease. The development of Q waves during STEMI increases the prognostic risk [16].

## Case Presentation

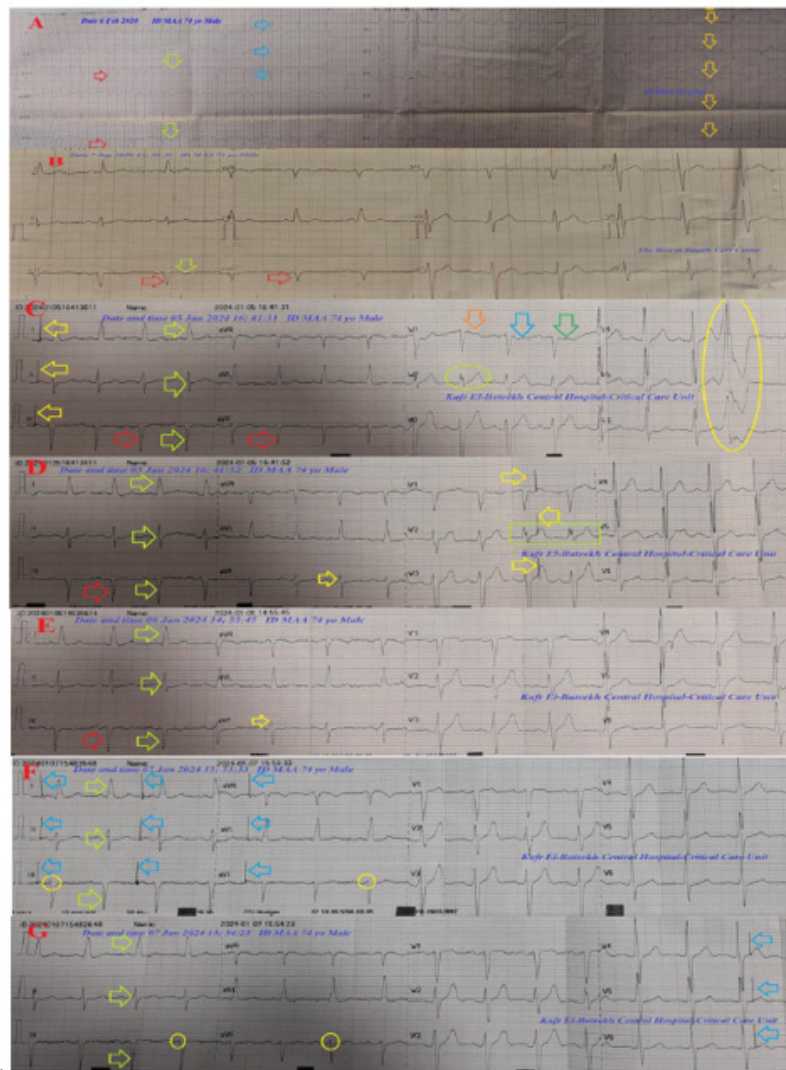
A 74-year-old, retired Officer, married Egyptian male heavy smoker patient was presented to the intensive care unit (ICU) with tachypnea, dyspnea, palpitations, and angina chest pain. Generalized body aches, sore throat, cough, fatigue, loss of appetite, and loss of smell were associated symptoms. The patient started to complain of fever 3 days ago. He has recent direct contact with a confirmed case of COVID-19 pneumonia 8 days ago. The patient gave a history of acute myocardial infarction (AMI) with chronic total occlusion (CTO) 4 years ago (Figure 1A). He managed acutely in the ICU with thrombolytic therapy and other traditional anti-ischemic measures. Later, the patient underwent percutaneous coronary intervention (PCI) dilation with ballooning placement. The ECG after PCI showed evidence of old inferior MI (Figure 1B). The patient gave a history of resected left renal tumor 3 years ago. Informed consent was taken. Upon general physical examination, generally, the patient looked cyanosed, irritable, dyspneic, tachypneic, and distressed with a regular rapid pulse rate of VR; 105 bpm, blood pressure (BP) of 230/150 mmHg, respiratory rate of 27 bpm, a temperature of 36°C, and pulse oximeter of oxygen (O<sub>2</sub>) saturation of 85%.

Coarse generalized chest crepitation was heard on chest auscultations. Currently, the patient was admitted to the ICU for acute hypertensive pulmonary edema with old. Initially, the patient was treated with O<sub>2</sub> inhalation by O<sub>2</sub> system line (100%, by nasal cannula, 10L/min) one sublingual isosorbide dinitrate tablet (5 mg), 4 furosemide IV amp (40 mg, then TDS), sublingual captopril tablet (25 mg), and continue nitroglycerine IVI (10 mg/50 ml solvent, 5 ug/min and titrated according to BP) were given. The patient was maintained and treated with cefotaxime; (1000 mg IV TID), azithromycin tablets (500 mg, OD), and hydrocortisone sodium succinate (100 mg IV BID). After controlling the BP, SC enoxaparin 80 mg, BID), aspirin tablet (75 mg, OD), clopidogrel tablets (75 mg, OD), captopril tablets (25 mg; BID), diltiazem tablets (60 mg, OD), and atorvastatin tablets (40 mg, OD) were added. The patient was daily monitored for temperature, pulse, blood pressure, ECG, and O<sub>2</sub> saturation. The current ECG tracing done on the presentation shows sinus tachycardia with evidence of old inferior MI, spike artifacts, Wavy triple sign in V1 lead (Yasser's sign), and sporadic monomorphic premature ventricular complex (PVC) (Figure 1C).

The fourth ECG tracing done within 21 seconds of the above ECG tracing showing sinus tachycardia with pathological Q waves in the lead III, with no pathological Q waves in the aVF lead, spike artifacts, two beats of RBBB in the V2 lead, and physiological LAD (Figure 1D). The fifth ECG tracing taken within 24 hours of the above ECG tracing showing NSR with pathological Q waves in the lead III, with no pathological Q waves in the aVF lead, and physiological LAD (Figure 1E). The sixth ECG tracing taken within 24 hours of the above ECG tracing showing NSR with no pathological Q waves

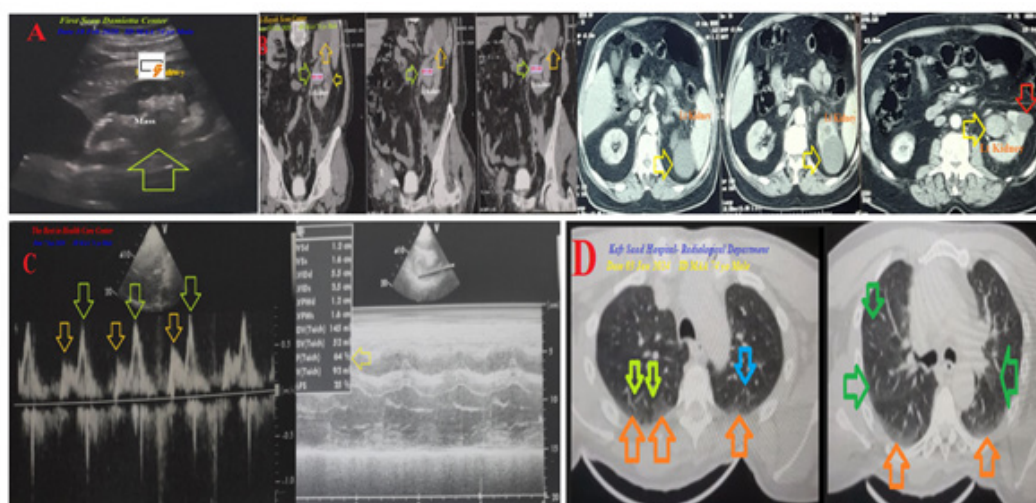
in inferior leads (III and aVF), spike artifacts, and physiological LAD (Figure 1F). The seventh ECG tracing taken within 50 seconds of the above ECG tracing showing NSR with no pathological Q waves in inferior leads (III and aVF), and physiological LAD (Figure 1G). The abdominal ultrasound done four years ago showed a left renal

mass, prostatic enlargement, and chronic cystitis (Figure 2A). The abdominal and pelvic CT scan with contrast done four years ago and showed a left renal mass, dilated left renal pelvis, and calyces, and left cortical renal cysts (Figure 2B).



**Figure 1:** Serial ECG tracings; A. tracing done four years ago and showed sinus tachycardia (of VR 100) with pathological Q waves and T-wave inversion in the inferior leads (III and aVF; red and lime arrows), extensive anterior horizontal ST-segment depression in the V1-6 leads (orange arrows), and physiological left axis deviation (LAD; light blue arrows). B. tracing taken within 7 months of the above ECG tracing, after the ICU treatment of acute MI, and PCI dilation with ballooning placement showed normal sinus rhythm (NSR; of VR; 72) with pathological Q waves (red arrows) and T-wave inversion in the III lead (lime arrows), normalization of the above horizontal ST-segment depression in the V1-6 leads, and physiological LAD. C. tracing done on the current presentation shows sinus tachycardia (of VR of 102) with pathological Q waves in the inferior leads (III and aVF; red and lime arrows), sporadic monomorphic PVCs in V4-6 leads (yellow circle), spike artifacts (yellow arrows), Wavy triple sign in V1 lead (Yasser's sign; orange, light blue, and green arrows), single beat of right bundle branch block (RBBB) in V2 lead, and physiological LAD (light blue arrows). D. tracing done within 21 seconds of the above ECG tracing shows sinus tachycardia (of VR of 100) with pathological Q waves in the lead III (red arrow), with no pathological Q waves in aVF lead (yellow arrow) spike artifacts (yellow arrows), two beats of RBBB in V2 lead (lime rectangle), and physiological LAD (lime arrows). E. tracing taken within 24 hours of the above ECG tracing shows NSR (of VR 83) with pathological Q waves in lead III (red arrow), with no pathological Q waves in aVF lead (yellow arrow) and physiological LAD (lime arrows). F. tracing taken within 24 hours of the above ECG tracing shows NSR (of VR 87) with no pathological Q waves in inferior leads (III and aVF; yellow circle), spike artifacts (light blue arrows), and physiological LAD (lime arrows). G. tracing taken within 50 seconds of the above ECG tracing shows NSR (of VR 90) with no pathological Q waves in inferior leads (III and aVF; yellow circle), and physiological LAD (lime arrows).





**Figure 2:** A. Section of abdominal ultrasound done four years ago showed left renal mass (lime arrow). B. Abdominal and pelvic CT scan with contrast done four years ago showed left renal mass (golden arrows), dilated left renal pelvis and calyces (lime arrows), and left cortical renal cysts (red arrow). C. Echocardiography done after PCI showed a diastolic dysfunction (lime and orange arrows), mild tricuspid regurgitation, dilated left atrium, mild concentric LVH, and a good LV systolic function of an EF of 64% (yellow arrow). D. The current Chest CT scan showed bilateral ground glass consolidation (orange arrows), hallow sign (lime arrows), reversed halo sign (light blue arrow), and bilateral haziness (green arrows).

The previous echocardiography was done after PCI showed a diastolic dysfunction, mild mitral regurgitation, mild aortic regurgitation, mild tricuspid regurgitation, dilated left atrium, mild concentric LVH, and a good LV systolic function grade I of an EF of 64% (Figure 2C). The current echocardiography shows a diastolic dysfunction grade III, mild tricuspid regurgitation, dilated left atrium, and regional wall motion abnormalities (RWMA) with anterior, anteroseptal, and apical hypokinesia of an EF of 58%. The chest CT showed bilateral ground glass consolidation, hallow sign, reversed halo sign, and bilateral haziness (Figure 2D). The renal biopsy of the left nephrectomy specimen showed non-invasive grade II superficial papillary Urothelial carcinoma limited to the lamina propria, no muscle invasion with old Bilharzial infestation. The initial complete blood count (CBC); Hb was 12.4 g/dl, RBCs;  $4.74 \times 10^3/\text{mm}^3$ , WBCs;  $9.4 \times 10^3/\text{mm}^3$  (Neutrophils; 83.7 %, Lymphocytes: 12.3%, Monocytes; 4.0%, Eosinophils; 0% and Basophils 0%), and Platelets;  $131 \times 10^3/\text{mm}^3$ . CRP was (6.0g/dl). SGPT was (26U/L) and SGOT was (42 U/L).

The serum albumen was (4.0g/dl). Serum creatinine was (1.3mg/dl) and blood urea was (30 mg/dl). RBS was (113 mg/dl). Pro B-type natriuretic peptide was (2741.0pg/mL). Plasma sodium was (130mmol/L). Serum chloride was (107.0mmol/L). Serum potassium was (3.9mmol/L). Ionized calcium was (0.99mmol/L). The troponin test was negative. ABG (PH: 7.51, PaCO<sub>2</sub>: 33, HCO<sub>3</sub><sup>-</sup>: 28.6, SaO<sub>2</sub>: 97.6, PaO<sub>2</sub>: 90) showed primary respiratory alkalosis, with secondary metabolic alkalosis. Hypertensive pulmonary edema, COVID pneumonia, old myocardial infarction, and graded Q-waves resolution in a resected urothelial carcinoma in the elderly was the most probable diagnosis. Within 3 days of the above

management, the patient finally showed nearly dramatic clinical and mostly electrocardiographic improvement. The patient was discharged after clinical stabilizations and continued on aspirin tablets (75 mg, OD), clopidogrel tablets (75 mg, OD), captopril tablets (25 mg; BID), diltiazem tablets (60 mg, OD), furosemide tablets (40 mg OD), oral nitroglycerine capsule (2.5 mg, twice daily), and atorvastatin tablets (20 mg, OD). Further recommended cardiac, chest, urological, and oncological follow-up was advised.

## Discussion

### Overview

- An elderly retired officer married male, heavy smoker Egyptian patient was admitted to the ICU with hypertensive pulmonary edema, COVID-19 pneumonia, and old myocardial infarction in a resected left urothelial carcinoma.
- The primary objective for my case study was the presence of hypertensive pulmonary edema, COVID-19 pneumonia, and old myocardial infarction in a resected left urothelial carcinoma in an elderly male heavy smoker patient in the ICU.
- The secondary objective for my case study was the question of; how you would manage this case in the ICU?
- Interestingly, the presence of a positive history of contact with a confirmed COVID-19 case, bilateral ground-glass consolidation, and some laboratory COVID-19 suspicion on top of clinical COVID-19 presentation with fever, dry cough, generalized body aches, anorexia, and loss of smell will strengthen the higher suspicion of COVID-19 diagnosis.

- e) The renal biopsy of the left nephrectomy specimen supports the diagnosis of non-invasive grade II superficial papillary urothelial carcinoma.
- f) The acute myocardial infarction in parallel to the present urothelial carcinoma causation. The other risk factors such as heavy cigarette smoking and elderly are considered.
- g) The renal biopsy evidence of bilharzial infestation with heavy cigarette smoking [1] are suggested risk factors for urothelial carcinoma.
- h) An elderly, male, heavy smoker, hypertensive, pulmonary edema, COVID-19 pneumonia, and old myocardial infarction past urothelial carcinoma are current risk factors.
- i) The graded Q-waves resolution in the ECG was noted over the years of follow-up.
- j) Stabilization and non-metastasis of urothelial carcinoma over about four years was observed.
- k) The current mild laboratory hypocalcemia, ECG evidence of Wavy triple sign in V1 lead (Yasser's sign), and ABG suggesting respiratory alkalosis is mostly due to tachypnea during the hypertensive pulmonary edema.
- l) Marked elevated Pro-B-type natriuretic peptide contributes to both hypertensive pulmonary edema and COVID-19 pneumonia. The other differential causes such as heart failure are considered.
- m) The sporadic monomorphic PVCs are insignificant.
- n) The associated and sporadic RBBB beats are interesting. However, there is no known mechanism.
- o) Acute heart failure was the most probable differential diagnosis for the current case study. However, the findings of the clinical examination are against it.
- p) I can't compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison.
- q) The only limitation of the current study was the unavailability of a new coronary angiography.

## Conclusion and Recommendations

- a) The urothelial carcinoma may be contributing to acute myocardial infarction.
- b) The graded Q-waves resolution in the ECG over the years of follow-up supports that the Q-waves may be transient.
- c) Stabilization and non-metastasis of urothelial carcinoma over four years may be a good sign.
- d) The associated and idiopathic sporadic RBBB beats are interesting.
- e) Elderly, male sex, heavy smoker, hypertensive, pulmonary edema, COVID-19 pneumonia, old myocardial infarction,

urothelial carcinoma, and marked elevated Pro B-type natriuretic peptide are the current constellation risk factors.

## Conflicts of Interest

There are no conflicts of interest.

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