CASE REPORT

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Changes in ST segment imitating acute coronary syndrome in a patient after pneumonectomy — a case report

Zmiany odcinka ST imitujące ostry zespół wieńcowy u pacjenta po pulmonektomii — opis przypadku

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Abstract

Ischaemic changes associated with the electrocardiographic changes in ST-T are indicators of myocardial ischaemia and acute coronary syndrome. For an effective therapeutic intervention, it is essential to confirm any initial diagnosis. However, many potential causes influencing the changes of ST-T can make differentiation and reaching a final diagnosis problematic. We present the case of a 61 year-old man with multiple comorbidities, and an overdose of digoxin after a pneumonectomy, which contributed to changes in the electrocardiogram which imitated acute coronary syndrome.

Key words: electrocardiography, pneumonectomy, acute coronary syndrome

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Introduction

Since the advent of invasive cardiology, the diagnosis of acute coronary syndrome (ACS) has arisen. In order to make the diagnosis of myocardial infarction, it is necessary to detect an increase and/or a decrease in the levels of cardiac troponin, with at least one being in the 99th percentile of the upper limit of the reference value, plus the presence of at least one of the following criteria: symptoms of ischaemia, new or presumably new changes in an electrocardiogram (ECG) [changes in ST-T, left bundle branch block (LBBB), right bundle branch block (RBBB), appearance of pathological Q], new or presumably new loss of myocardial viability or new segmental dysfunction of contractility confirmed in imaging tests, a procedural flow-limiting complication in an angiography or post-mortem [1]. Despite these characteristic symptoms, and access to ECGs and laboratory measurements of high-sensitivity troponin-T concentrations, the identification of ACS continues to be a diagnostic problem, both in terms of over-diagnosis and under-diagnosis. This problem is multifactorial. During diagnostic procedures, the most important aspect is determining whether the observed changes are primarily of a cardiac nature, or whether they are secondary to other factors.

We present the case of a patient with several comorbidities, in whom a suspicion of ACS was not confirmed.

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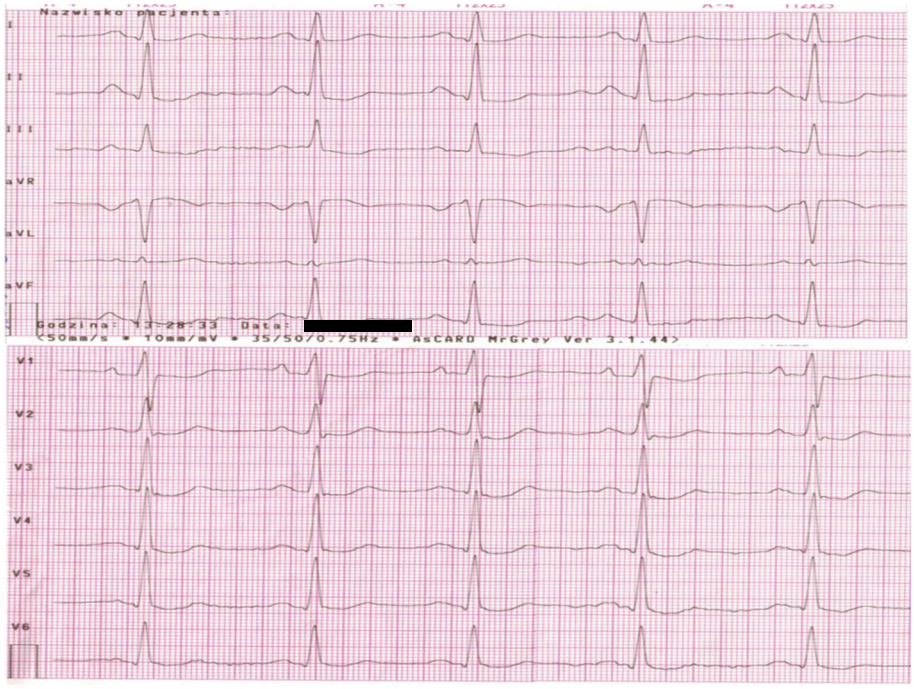

Figure 1. Sinus rhythm around 100/min, P-pulmonale, ST elevation in the aVR lead, ST depression in leads I, III, aVF, V1–V6 and flattened T waves

Case description

A 61 year-old male who had undergone a right-side pneumonectomy due to non-small cell lung cancer in 2005 was moved in a serious condition from the pulmonary ward to the cardiac ward with suspected ACS. The dominant symptom was a progressive shortness of breath, including at rest, of three days' duration. The patient had a history of chronic heart failure with preserved ejection fraction, stage II arterial hypertension as determined by European Society of Hypertension (ESH)/European Society of Cardiology (ESC), dyslipidemia, chronic obstructive pulmonary disease with multiple exacerbations, chronic respiratory failure, chronic anaemia and gastroesophageal reflux. The patient had a history of pulmonary embolism and multiple hospital-acquired pneumonias. Physical examination and auscultation found multiple rales and crackles up to the middle of the scapula above the left lung fields, tachypnoea 40/min, tachycardia 100–120/min, blood pressure (BP) 110/70 mm Hg, and a slightly enlarged liver (up to 2 cm above the costal margin). No peripheral oedema was found.

A resting ECG found a 1 mm ST elevation in the aVR lead, 1–2 mm concave ST depressions in leads I–III, aVF, V1–V6 and a flattened T wave (Figure 1). In laboratory studies, attention was drawn to slightly elevated high-sensitivity troponin T concentrations: 21.49 ng/L at the pulmonary ward and 29 ng/L after admission to the cardiac ward (normal < 14 ng/L). Moreover, upon admission, high concentrations of N-terminal pro-B-type natriuretic peptide (NT-proBNP: 2,297.0 pg/mL), high levels of the inflammatory markers white blood count (WBC: 20.00 × 10^3/μL) and C-reactive protein (CRP: 29 mg/L), and elevated levels of digoxin (2.56 ng/mL) were detected.

In the completed echocardiography examination, only a subcostal projection was obtained, which did not show any impairments in contractility of the non-enlarged left ventricle and the right ventricle, with preserved systolic function of the left ventricle (ejection fraction 53%).

Based upon the full clinical picture, once the patient had been stabilised a physician on the duty decided to conduct a coronary angiography, which did not reveal any sclerotic changes in the coronary arteries (Figure 2). At a further stage of hospitalisation, a continued increase in inflammatory parameters was observed (CRP from 29 to 97.5 mg/L), and hospital-acquired pneumonia was diagnosed. Antibiotic therapy was commenced and the existing treatment was modified to include the standard therapy for heart failure (intravenous loop diuretics and mineralocorticoid receptor antagonists) and digitalis glycosides were discontinued. A clinical improvement was observed, and shortness of breath reduced to class II of New York Heart Association (NYHA). A physical examination found a heart rate of 88/min, BP 114/76 mm Hg, isolated crackles at the base of the left lung, a non-enlarged liver in a palpation

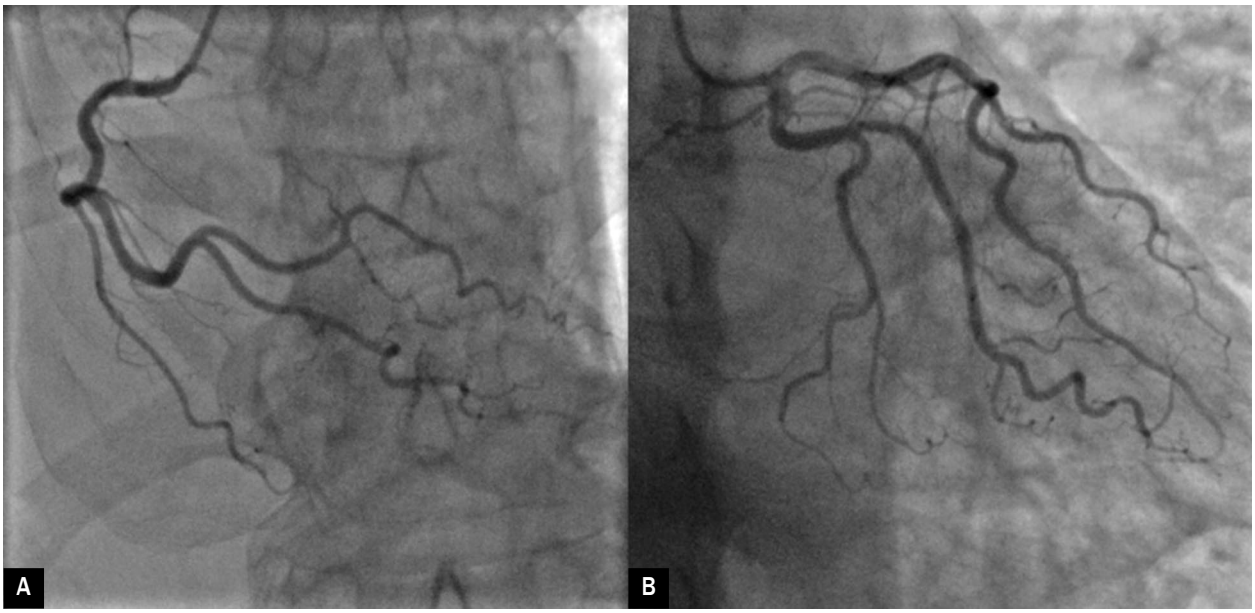

Figure 2A, B. Coronary angiography — not found sclerotic changes in the coronary arteries were found

study, and a drop in CRP to 47.6 mg/L. Over the course of hospitalisation, several ECGs were performed, which did not reveal progressive changes in the ST segment or the T wave. The patient was moved to the Pulmonology Clinic with the aim of further treating his pneumonia.

Discussion

The most notable aspects of this case are the signs and symptoms: slightly elevated concentrations of troponin T with accompanying high concentration of NT-proBNP, and changes in the ST segment in the ECG. On this basis, an initial diagnosis of ACS without ST segment elevation was made on the pulmonary ward. While, the observed changes in the ECG (concave depression of the ST segment) may have been caused by a high concentration of digoxin in the patient's blood plasma [2]. The medication was discontinued. However, at this stage it was not certain whether the drug was responsible for the observed changes as the half-life of digoxin is between 26 and 45 hours [3]. In this case it is recommended to perform a routine transthoracic echocardiogram, which is useful for diagnosing impaired contractility arising from ischaemia by assessing the global and segmental function of the left ventricle, and also allows for the elimination of differential diagnoses [1]. In the described case, the echocardiographic examination did not uncover any segmental dysfunction of contractility.

We think, to boldly patient was qualified for a coronary angiography. In further observations, attention was drawn to increasing markers of inflammation, which consequently

Table 1. Works concerning electrocardiographic changes following lung resection surgeries and changes described

Title	Year of publication	AuthoErs	ECG changes described
Electrocardiographic impacts of lung resection	2013	Chhabra L et al.	ST elevation, ST depression, left- and rightward axial deviation, changes in progression and amplitude of the R wave, changes in P and T waves
Electrocardiographic changes after lung resection: case report and brief review	2015	Abhijit S. Nair et al.	ST elevation in I, II, III, aVF, V4–V6
Acute "pseudoischemic" ECG abnormalities after right pneumonectomy	2017	Vasic N et al.	ST elevation in V2–V6; negative T wave in V3–V6

ECG — electrocardiogram

resulted in a diagnosis of hospital-acquired pneumonia. This appears to be the cause of the exacerbation of the patient's comorbidities, including heart failure, which appears to have led to the observed changes in troponin concentrations [4, 5]. A few days after the discontinuation of digoxin and the normalisation of its concentrations, a follow-up ECG did not reveal any changes compared to earlier records. It should be assumed that the irregularities in the ECG were caused by changes in anatomical conditions in the chest following the pneumonectomy. This possibility is confirmed by a few works discussing the problems surrounding the occurrence of arrhythmias and electrocardiographic changes following lung resection surgery (Table 1).

Conclusions

In presenting this case, we wish to draw attention to the rarely discussed problem of the effects of lung resection surgery on electrocardiographic changes. Awareness of ECG changes and their identification is extremely important. This may not only allow for other acute cardiovascular conditions to be excluded, but also to protect the patient from unnecessary invasive tests and diagnostic procedures.

Conflict(s) of interest

The authors report no conflict of interest.

Streszczenie

Zmiany odcinka ST w zapisie elektrokardiogramu (EKG) przywołują na myśl podejrzenie niedokrwienia mięśnia sercowego i często ostry zespół wieńcowy. W celu prowadzenia prawidłowej strategii terapeutycznej koniecznym jest potwierdzenie wstępnej diagnozy. Jednakże mnogość potencjalnych przyczyn wpływających na zmiany odcinka ST-T może sprawić nie lada problem w różnicowaniu i ustaleniu ostatecznej diagnozy. W niniejszej pracy przedstawiono przypadek 61-letniego pacjenta z wieloma obciążeniami internistycznymi, przedawkowaniem digoksyny oraz po przebytej pulmonektomii, które przyczyniły się do zmian w EKG imitując ostry zespół wieńcowy.

Słowa kluczowe: elektrokardiografia, pulmonektomia, ostry zespół wieńcowy

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