Concealed Metastatic Lung Carcinoma Presenting as Acute Coronary Syndrome With Progressive Conduction Abnormalities

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A 70-year-old diabetic woman with recent onset of cough was admitted to the emergency room for acute chest pain with evidence of T-wave abnormalities in the V2 through V6, L1, and aVL leads (Figure 1A). Her coronary arteries were angiographically normal, and her troponin I was elevated at 0.3 μg/L. Two-dimensional echocardiography revealed a hypertrophic left ventricle without kinetic abnormalities. Three weeks later, the patient was readmitted for chest pain and a complete left bundle-branch block on 12-lead ECG (Figure 1B) with a peak troponin I of 0.429 μg/L. To exclude a myocarditis, a cardiac magnetic resonance (CMR) was performed. On early scout images, diffuse multiple nodules with irregular epicardial borders were evident (Figure 2A–2C), which were also present on CMR T1 cine balanced images, indicating an irregular tissue composition (Movies I and II in the online-only Data Supplement). A moderate pericardial effusion was also detected. On T2-weighted images, the nodules presented a signal intensity higher than skeletal muscle (Figure 3A). First-pass contrast sequences showed inhomogeneous perfusion at the level of intramyocardial nodules (Movie III in the online-only Data Supplement). On late gadolinium CMR imaging, an inhomogeneous and reduced contrast uptake was noted, diffusely involving the ventricular myocardium besides the nodules (Figure 3B). These features were in keeping with an abnormal myocardial tissue composition, not resulting from myocardial edema, thus ruling out myocarditis. During the same examination, a right pulmonary mass was also detected and better defined on computed tomography (Figure 4). A transbronchial biopsy was then performed, revealing a lung carcinoma (Figure 5). One week later, the patient developed a complete atrioventricular block (Figure 1C), and she died 3 days later. Postmortem definitely ruled out coronary artery disease and provided a diagnosis of lung adenosquamous cell carcinoma with multiorgan metastases and pericardial effusion. Multiple firm white masses were already visible on external examination of the heart. On serial transverse sections, corresponding to equivalent CMR postcontrast sequences images, the nodules consisted of neoplastic tissue proliferation with variable amounts of myocardial necrosis (Figure 6). At higher magnification, the metastatic tumor was composed of irregularly shaped glands with marked cytological atypia infiltrating the myocardium and spreading to the epicardium (Figure 7).

Although primary cardiac tumors are extremely uncommon, secondary tumors are not. In a recent postmortem survey of 7289 malignant neoplasms, an incidence of 9.1% of heart metastases was found. The highest rate of heart metastasis was reported in pleural mesothelioma (48.4%), melanoma (27.8%), lung adenocarcinoma (21%), undifferentiated carcinoma (19.5%), lung squamous cell carcinoma (18.2%), and breast carcinoma (15.5%).

To the best of our knowledge, this is the first case describing a concealed lung carcinoma presenting like acute coronary syndrome as a result of the diffuse infiltration of the myocardium, as detected in vivo by CMR and then confirmed by postmortem pathology examination. Moreover, the extension of the neoplastic infiltration to the basal septum, as detected by CMR, explains the progressive conduction system involvement, eventually leading to complete atrioventricular block.

The multiparametric tissue characterization provided by CMR helps differential diagnosis with more common causes of troponin release such as myocardial infarction or myocarditis.

Disclosures

None.

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References

Figure 1. ECG dynamic abnormalities. The admission 12-lead ECG (A) shows ST- and T-wave abnormalities compatible with acute coronary syndrome. Three weeks later, a complete left-bundle branch block appears (B), evolving into a complete atrioventricular block 1 week later (C).

Figure 2. Cardiac magnetic resonance scout images. The scout localizer images in vertical long-axis views (A and B) and horizontal long-axis view (C) show an inhomogeneous myocardial tissue composition with multiple intramural nodules. Note also the pericardial effusion.
Figure 3. Cardiac magnetic resonance myocardial tissue characterization. A T2-weighted with fat saturation sequence in the long-axis view shows multiple hyperintense nodules (A), with an inhomogeneous uptake of gadolinium agent on postcontrast delayed T1 inversion recovery in the same section (B). Note the multiple nodules involving the full interventricular septum on both sequences.

Figure 4. Contrast computed tomography. A right-sided lung mass (maximum diameter, 4 cm) near the pulmonary hilum is detected.

Figure 5. Transbronchial biopsy. Histology and immunohistochemistry reveal diffuse infiltration of irregularly shaped glands with cytological atypia (A and inset, hematoxylin-eosin stain); marked cytoplasmic (B and C) and nuclear (D) immunoreactivity for 34β12, CK7 and TTF1 are demonstrated.
Figure 6. Cardiac magnetic resonance–histopathology correlation on the heart specimen. The inhomogeneous uptake of gadolinium agent on postcontrast delayed T1 inversion recovery in the basal (A) and apical (B) short-axis view correspond to macroscopic whitish firm metastatic nodules on transverse sections at postmortem (C and D). In full-thickness histological sections corresponding to the boxed areas in C and D, the nodules consist of neoplastic epithelial tissue proliferation (E and F, hematoxylin-eosin stain).

Figure 7. Histology of the neoplastic nodules. At higher magnification, histology of myocardial (A and B) and epicardial (C and D) metastasis shows multiple foci of carcinomatous infiltration with both glandular and solid sheet. Note also the presence of fibrinous pericarditis (arrow) and intravenous thrombosis (asterisk) (hematoxylin-eosin stain).
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