

## Lung carcinoma paraneoplastic reactive regional pericarditis mimicking ST elevation myocardial infarct

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### ABSTRACT

ST-elevation myocardial infarction (STEMI) is a cardiac emergency. However, multiple clinical disorders can cause ST-elevation ECG changes, one of which is pericarditis. Regional pericarditis is a less known clinical phenomenon that can mimic STEMI. We report a case of poorly differentiated lung carcinoma associated reactive regional pericarditis mimicking inferior STEMI.

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## 1. Introduction

ST-elevation myocardial infarction (STEMI) is a cardiac emergency. The clinical performance and quality measures for adults with STEMI and non-STEMI requires emergent cardiac catheterization and percutaneous cardiac intervention (PCI), with the recommended first medical contact (FMC)-to-device time to be within 90 minutes [1]. However, there are diverse clinical conditions that can produce ECG changes of ST-elevation, including acute myocardial infarction, Prinzmetal's angina, pulmonary embolism, cardioversion, hyperkalemia, acute pericarditis, early repolarization, left bundle-branch block, left ventricular hypertrophy, and Brugada syndrome [2].

Moreover, secondary cardiac cancer from primary lung cancer or other cancers can induce regional ST elevation and mimic STEMI [3]. This report describes a patient with newly diagnosed poorly differentiated lung carcinoma causing paraneoplastic reactive inferior regional pericarditis who clinically presented as STEMI.

## 2. Case report

A 62-year-old male woke up in the middle of the night with 10/10 substernal chest pain and tightness, non-radiating, which lasted for one hour and was associated with palpitations and shortness of breath, with no aggravating or alleviating factors. His chronic medical problems included essential hypertension, benign prostate hypertrophy, nicotine-dependent tobacco abuse with 42-pack-year, and remote history of bladder cancer which had been cured and currently tumor free. Six weeks ago, he developed a dry cough; 2 weeks ago, he presented to a walk-in clinic where chest x-ray and follow up CT chest with IV

contrast revealed a right upper lobe mass, concerning for primary lung cancer. He endorsed 10 kg unintentional weight loss despite the same oral intake over 2 months. He denied a history of coronary artery disease, exertional chest pain, or shortness of breath. On admission, vital signs demonstrated temperature 99° F, pulse 101 bpm, respiration 19 per minute, BP 117/79 mmHg, SpO<sub>2</sub> 98% on 2 L nasal cannula of oxygen. Physical exam was unremarkable, including regular cardiac rhythm, S1 and S2 are present, no heart murmur or friction rub.

Laboratory studies showed WBC  $13.2 \times 10^3/\mu\text{L}$ , hemoglobin 12.8 g/dL, hematocrit 38.4%, platelet  $246 \times 10^3/\mu\text{L}$ ; potassium 4.4 mEq/L, BUN 13 mg/dL, creatinine 0.88 mg/dL; Calcium 10.7 mg/dL; Magnesium 2.2 mg/dL; Total bilirubin 0.7 mg/dL, AST 15 U/L, ALT 9 U/L, ALP 126 U/L, total protein 7.2 g/dL, albumin 3.7 g/dL; LDH 707 IU/L (reference range, 100–245 IU/L); First set of troponin I, <0.01 ng/dL, 2nd set <0.01 ng/dL 7 hours later, 0.023 ng/dL 24 hours later; Triglycerides 79 mg/dL, total cholesterol 152 mg/dL, HDL 30 mg/dL, LDL 106 mg/dL.

CT chest with IV contrast 2 weeks prior to presentation showed spiculated right upper lobe masses of  $2.4 \times 1.7$  cm and  $2.7 \times 1.7$  cm; superior mediastinum mass of  $5.4 \times 4.1$  cm, large right hilar mass of  $5.5 \times 3.7$  cm with post-obstructive atelectasis or pneumonia; extensive mediastinal lymphadenopathy with narrowing of the superior vena cava likely representing primary lung cancer; small or moderate pericardial effusion; left adrenal nodule suspicious for metastasis. CTA chest on admission showed no evidence of pulmonary embolus; increased pericardial effusion.

On admission, ECG showed sinus rhythm, heart rate 95 bpm, ST elevation at lead II, aVF and III, no



**Figure 1.** Inferior regional pericarditis.

Q wave, with ST depression at lead aVR and V1 (Figure 1). Prior ECG available 5 years ago revealed normal sinus rhythm without ST-T changes (not shown). The patient was diagnosed with presumed acute inferior STEMI, and underwent emergent cardiac catheterization, which revealed middle LAD 30% stenosis, middle LCx 30% stenosis, and distal RCA 30% stenosis, and no percutaneous intervention was warranted.

The initial differential diagnoses for the patient's chest pain and inferior ST-elevation ECG changes included inferior STEMI, unstable angina, coronary artery spasm, regional pericarditis, and pulmonary embolism. Serial evaluation of troponin I, ECGs, and cardiac catheterization findings ruled out acute coronary syndrome. CT angiogram chest ruled out pulmonary embolism.

On hospital day 1, the patient developed positional pleuritic chest pain. Physical exam revealed no pericardial rub. Repeated ECG revealed persistent inferior lead ST elevations, no Q wave (not shown), similar to admission ECG; echocardiogram revealed pericardial effusion without tamponade. Laboratory studies showed WBC  $13.2 \times 10^3/\mu\text{L}$ , CRP 103.7 mg/L, and ESR 102 mm/hr. Given leukocytosis and elevated inflammatory markers of CRP and ESR, as well as echocardiogram evidence of pericardial effusion, the patient was diagnosed with inferior regional pericarditis.

A pericardiocentesis was performed with 680 mL serous fluid drained on hospital day 1. The pericardial catheter was left in place for 48 hours, when the drainage was less than 25 ml/day, the catheter was removed. Pericardial effusion cell count and differential showed WBC 29,855/mm<sup>3</sup>, RBC 473/mm<sup>3</sup>, and neutrophil 83%, lymphocytes 7%, monocyte 10%; pH 7.5, glucose 97 g/dL, triglycerides 25 mg/dL, cholesterol 98 mg/dL, protein 4.9 g/dL, LDH 896 IU/L.

Pericardial fluid culture revealed no bacterial, fungal or mycobacterial growth; Cytopathology showed atypical inflammatory cells and favor reactive, no malignant cells. The patient was treated with colchicine and ibuprofen, and his pleuritic chest pain resolved in 2 days. Patient was continued on colchicine and ibuprofen on discharge. Two weeks later, repeated ECG revealed normalized ST waves (not shown); repeat CT chest without contrast revealed significant improvement in the pericardial effusion.

As for patient's newly diagnosed lung cancer, lung cancer staging revealed brain metastasis by MRI brain, and hepatic and bilateral adrenal metastasis by CT abdomen and pelvis. Bronchoscopy showed complete obstruction right upper lobe bronchus just distal to its origin by submucosal and mucosal infiltration of tumor. Biopsy and surgical pathology revealed poorly differentiated carcinoma.

### 3. Discussion

Classic acute pericarditis ECG presents with widespread ST elevation and PR depression (diffuse pericarditis), and reciprocal ST depression and PR elevation in aVR and/or V1; of note, the degree of ST elevation is typically modest (0.5–1 mm). Pericarditis is diagnosed with at least 2 of the 4 following criteria [4]: 1. Pleuric chest pain; 2. Pericardial rub on physical exam; 3. New widespread ST-elevation or PR depression in aVR on ECG; 4. New or worsening pericardial effusion. Additional supportive findings include elevation of inflammatory markers, i.e., CRP, ESR, and leukocytosis, image evidence of pericardial inflammation by echocardiogram or CT chest.

Regional pericarditis, with regional ST elevation, is a less known clinical phenomenon, which was

initially reported in post-acute myocardial infarction (AMI) [5–7]; literature review revealed that other etiologies of regional pericarditis include post cardiac ablation [8], post-upper abdominal surgery [9], trauma [10], and idiopathic myopericarditis [11]. Moreover, it has been reported that secondary cardiac cancer from primary lung cancer or other cancers can induce regional ST elevation and mimic STEMI [3]. It is a challenge to differentiate regional pericarditis from STEMI, thus cardiac catheterization was performed in most of the regional pericarditis case reports to rule out STEMI.

A large serial autopsy study of 1928 patients with cancer revealed that 8.4% had cardiac metastasis [12]; Another consecutive autopsy study of 154 patients with secondary cardiac tumors showed the pericardium and epicardium were the most common metastatic location, followed by myocardium and endocardium [13]. Therefore, pericardial or cardiac metastatic tumor should be considered whenever a patient with known malignancy develops a pericardial effusion, new heart murmur or rub, ECG changes, or arrhythmia.

For this case, pericardial effusion protein to serum protein ratio was 0.68 (>0.5), pericardial effusion LDH to serum LDH ratio was 1.27 (>0.6), pericardial effusion LDH 896 IU/L (>2/3 of serum LDH upper limit of normal); as per Light's criteria [4], the pericardial effusion was exudative. An exudative pericardial effusion cannot be used to differentiate between lung cancer metastasis versus paraneoplastic reaction; however, the high WBC count with 83% neutrophils and cytology with no malignancy cells in the pericardial effusion, CT chest with no evidence of pericardial or myocardial metastases, and clinical and ECG normalization with anti-inflammatory treatment (colchicine and ibuprofen) were all consistent with a paraneoplastic reactive regional pericarditis. It is less likely this patient had a secondary cardiac tumor, which usually causes persistent ST elevation [14], although we cannot absolutely rule out the possibility of early occult cardiac metastasis, since biopsy was not indicated nor performed.

The chest pain quality and troponin levels [15] are insufficient to distinguish between regional pericarditis and STEMI, however there are some ECG characteristics might be useful to differentiate these two etiologies (Table 1). Notably, this patient had no reciprocal ST depression at lead I and aVL, or Q wave evolution.

In conclusion, we present a case of paraneoplastic reactive inferior regional pericarditis due to poorly differentiated lung cancer, which presents with regional ST elevation and must be differentiated from STEMI and other more common causes of such clinical findings.

**Table 1.** Clinical characteristics for differential of regional pericarditis and STEMI.

	Regional pericarditis	STEMI
Differences	No reciprocal ST depression, except aVR and V1	Reciprocal ST depression, other than aVR and V1 Convex or horizontal ST elevation Q wave evolution
Similarities	Positional or pleuritic chest pain, pericardial friction rub Concave ST elevation PR segment depression	Positional or pleuritic chest pain, pericardial friction rub Concave ST elevation PR segment depression

## Disclosure statement

No potential conflict of interest was reported by the author(s).

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## References

- O'Gara PT, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American college of cardiology foundation/American heart association task force on practice guidelines. *J Am Coll Cardiol.* 2013;61(4): e78–e140.
- Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. *N Engl J Med.* 2003;349(22):2128–2135.
- Suga T, et al. ST segment elevation in secondary cardiac cancer: a case report and review of the literature. *Int J Clin Exp Med.* 2015;8(5):7719–7727.
- Adler Y, et al. 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the task force for the diagnosis and management of pericardial diseases of the European society of cardiology (ESC) endorsed by: the European association for cardio-thoracic surgery (EACTS). *Eur Heart J.* 2015;36(42):2921–2964.
- Oliva PB, Hammill SC, Edwards WD. Electrocardiographic diagnosis of postinfarction regional pericarditis. Ancillary observations regarding the effect of reperfusion on the rapidity and amplitude of T wave inversion after acute myocardial infarction. *Circulation.* 1993;88(3):896–904.
- Oliva PB, Hammill SC. The clinical distinction between regional postinfarction pericarditis and other causes of postinfarction chest pain: ancillary observations regarding the effect of lytic therapy upon the frequency of postinfarction pericarditis, postinfarction angina, and reinfarction. *Clin Cardiol.* 1994;17(9):471–478.
- Dorfman TA, Aqel R. Regional pericarditis: a review of the pericardial manifestations of acute myocardial infarction. *Clin Cardiol.* 2009;32(3):115–120.
- Orme J, Eddin M, Loli A. Regional pericarditis status post cardiac ablation: a case report. *N Am J Med Sci.* 2015;7(1):33–35.

- [9] Alhammouri AT, Omar BA. Regional pericarditis mimicking inferior myocardial infarction following abdominal surgery. *Case Rep Med.* 2014;2014:301976.
- [10] Rechenmacher S, Jurewitz D, Southard J, et al. Barking up the wrong tree: regional pericarditis mimicking STEMI. *Am J Med.* 2013;126(8):679–681.
- [11] Nisbet BC, Breyer M. Acute myopericarditis with focal ECG findings mimicking acute myocardial infarction. *J Emerg Med.* 2010;39(5):e153–8.
- [12] Silvestri F, et al. Metastases of the heart and pericardium. *G Ital Cardiol.* 1997;27(12):1252–1255.
- [13] Lam KY, Dickens P, Chan AC. Tumors of the heart. A 20-year experience with a review of 12,485 consecutive autopsies. *Arch Pathol Lab Med.* 1993;117(10):1027–1031.
- [14] Astorri E, et al. Persistent ST segment elevation in a patient with metastatic involvement of the heart. *Minerva Cardioangiol.* 2001;49(1):81–85.
- [15] Imazio M, et al. Cardiac troponin I in acute pericarditis. *J Am Coll Cardiol.* 2003;42(12):2144–2148.