An Unusual Complication during Bronchoscopy: Hypotension, Global ST Segment Elevation and Acute Severe Left Ventricular Systolic Dysfunction

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An Unusual ECG pattern during Bronchoscopy

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Abstract:

Bronchoscopy is a procedure that is widely performed and is generally considered to be safe.

Cardiac complications occurring during bronchoscopy are uncommon, and usually occur in

elderly patients with co-existent coronary artery disease, hypertension or severely impaired

pulmonary function with resting hypoxemia. We report an unusual patient who developed

sudden onset restlessness, chest discomfort, hypotension, global ST elevation in multiple ECG

leads with acute severe left ventricular (LV) systolic dysfunction, during a bronchoscopic

transbronchial lymph node biopsy procedure. Differential diagnosis included a massive

myocardial infarction, apical ballooning (Tako-tsubo syndrome) or coronary vasospasm. The

ECG changes resolved spontaneously and a coronary angiogram done later, revealed normal

coronary artery anatomy and normal LV function. The patient made an uneventful recovery. It

is important for physicians to be aware of such unusual complications to be able to appropriately

manage these patients in clinical practice.

Key Words: Bronchoscopy, Cardiac, Myocardial infarction

Introduction:

The morbidity and mortality associated with bronchoscopy procedures is low (0.01-0.02%) and cardiovascular complications are extremely rare. ^{1,2} We report a case who developed sudden onset chest discomfort, hypotension, ST segment elevation in multiple ECG leads and acute severe left ventricular systolic dysfunction during a bronchoscopic transbronchial lymph node biopsy procedure. The ECG changes and cardiac biomarker assay indicated a massive ST elevation myocardial infarction (MI). There was spontaneous resolution of the ECG changes raising the possibility of "acute malignant coronary vasospasm" or apical ballooning (Tako-tsubo syndrome) as the underlying mechanism. A coronary angiogram done 48 hours later, revealed normal coronary artery anatomy and normal LV function without any residual regional wall motion abnormalities. The patient was managed conservatively and made an uneventful recovery.

Case:

The patient was RS,a 61 year old non-diabetic, non-hypertensive ,non-smoker, female who presented with gradually progressive shortness of breath and dry, non-productive cough since the last one year. There was no history of fever, weight loss or hemoptysis. She denied any history of angina or a cardiac event in the past. Routine hemogram and blood chemistry were normal, while a Mantoux test was strongly positive (25 mm induration after 48 hours of ppd injection). The X-ray chest revealed bilateral reticulonodular opacities while CT chest demonstrated interlobular septal thickening, multiple area of bronchiectasis with cystic changes in right middle lobe, multiple enlarged mediastinal lymph nodes and bilateral hilar lymphadenopathy. A

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transbronchial lymph node biopsy was planned to further elucidate the cause of mediastinal

lymphadenopathy.

While performing the transbronchial needle biopsy from the right hilar lymph node region, the

patient developed sudden restlessness, retrosternal chest discomfort and a feeling of uneasiness.

The ECG monitor revealed gross ST elevation and the patient rapidly became diaphoretic,

hypotensive and hypoxemic. The procedure was immediately terminated, and efforts were

instituted to maintain the BP using intravenous fluid infusion and inotropic support. The patient

was transferred to the intensive care unit where a 12 lead ECG revealed gross ST elevation in

inferior as well as anterior precordial leads (Figure 1) suggestive of acute ST elevation MI and a

BP of 70/55 mm Hg.

In view of the ECG changes and obvious likelihood of massive, acute MI, a Cardiology opinion

was sought for consideration of transfer to the cardiac catheterization laboratory and urgent

coronary angiography. An X-ray chest done revealed no evidence of pneumothorax or

pneumomediastinum. At the time of examination by the Cardiology team, the patient was

conscious and oriented and denied any ongoing chest pain. Another 12 lead ECG at this time

interestingly revealed slight reduction in the ST elevation, especially in the inferior leads and

precordial leads V4-V6 (Figure 2).

An urgent bedside echocardiography revealed severe global hypokinesia and an overall left

ventricular (LV) ejection fraction of 30%. Although a peri-procedural MI was still the most

likely possibility, in view of the spontaneous reduction in the ST segment elevation, a likelihood

of "malignant coronary vasopsam" was also now considered. While the cardiac catheterization

laboratory team was on standby, it was decided to perform serial ECG's and observe the

temporal pattern of ST segment evolution before performing an urgent coronary angiogram. As the ST segment elevation gradually resolved, a brief run of rapid ventricular tachycardia was noted (Figure 3) which was managed successfully with a bolus of Amiodarone (IV 150 mg). Serial ECG's performed over the next 30 minutes revealed progressive resolution of the ST segment elevation and normalization of the ST segment. (Figure 4)

Cardiac biomarker assay expectedly revealed elevated Troponin-I (9.2ng/ml) and CKMB (12 ng/ml) which further escalated to 80 ng/ml and 178 ng/ml respectively at 6 hours. The patient made a rapid uneventful recovery over the next 48 hours. A coronary angiogram performed after 48 hours revealed normal coronary arteries (Figure 5). The left ventricular angiogram also revealed normal LV function without any regional wall motion abnormalities. (Figure 6), therefore suggesting possible acute coronary vasospasm as the possible underlying etiology of the global ST elevation.

Discussion:

Cardiac complications like arrhythmias, myocardial ischemia and infarction occurring during bronchoscopic procedures are uncommon, and generally occur in elderly patients with comorbidities such as pre-existent coronary artery disease, hypertension or severe impairment of pulmonary function with resting hypoxemia.^{3,4} Various cardiac arrhythmias ranging from the benign atrial and ventricular premature complexes or supraventricular tachycardias to more malignant ventricular tachycardias, bradyarrhythmias and even cardiac arrest have been reported during bronchoscopy.^{2,4-7} Precipitation of myocardial ischemia and occurrence of acute ST elevation myocardial infarction following bronchoscopy although uncommon has also been reported.^{1,8,9}

Occurrence of cardiac ischemia and arrhythmias, although rare is generally due to increased level of circulating catecholamines, precipitation of true myocardial ischemia, or following administration of anaesthetic drugs especially in patients with underlying hypoxemia and other co-morbidities.

The ECG changes noted in our case, suggested a massive MI, which was corroborated by the elevated cardiac biomarker levels. We initially did plan to perform an urgent coronary angiogram (in view of the ECG findings, hypotension and echocardiographic evidence of LV dysfunction). However due to the spontaneous resolution of the ECG changes (in absence of any fibrinolysis or coronary intervention), the coronary angiogram was deferred and serial ECG's performed which revealed complete normalization of the ST changes. Had the ECG's shown this spontaneous resolution or the patient had continued chest pain, an urgent coronary angiogram would definitely have been performed, since the cardiac catheterization laboratory team was on continuous standby.

The underlying pathogenesis in this case was considered to be either apical ballooning (Takotsubo) syndrome or "acute malignant coronary vasospasm". Apical ballooning (Takotsubo) syndrome is characterized by sudden, severe, transient segmental dysfunction of the LV due to a hyper-adrenergic state usually following intense emotional or physical stress. The clinical presentation simulates that of an acute MI with ST elevation in the ECG and rapid development of acute LV systolic dysfunction. In these patients coronary angiography typically reveals normal coronary anatomy and impaired LV systolic function (apical ballooning), which rapidly resolves with time without any residual impairment. Guerrero et al recently reported the occurrence of Takotsubo syndrome following bronchoscopy. However in contrast to our

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case, their patient did not demonstrate any significant ECG changes or troponin rise, although

coronary angiography confirmed normal coronary anatomy with severe LV dysfunction.

Due to the presence of the completely normal LV function without any regional wall motion

abnormality on the LV angiogram, we hypothesized that "acute malignant coronary vasospasm"

could be the underlying pathophysiology of the global ST elevation in our case. The diffuse

coronary vasospasm may have resulted in global myocardial ischemia, ST elevation and acute

ever LV systolic dysfunction. It is still possible that our patient did have Tako-tsubo syndrome

and the LV function had completely recovered by the time of the coronary angiography.

However the LV was initially globally hypokientic in our patient, while in cases of apical

ballooning syndrome, apical akinesia or dyskinesia with preserved or hyperkinetic contractile

basal LV segments are more typically observed.

Conclusion:

This case highlights the occurrence of an extensive *pseudo myocardial infarction pattern* as a

complication of bronchoscopy, in a patient without underlying coronary artery disease, possibly

secondary to "acute malignant coronary vasospasm." The case highlights the fact that such

potentially life-threatening complications may occur during bronchoscopic procedures. Since it

is not possible to predict the occurrence of such complications, routine ECG monitoring

(especially for elderly/high risk patients) during bronchoscopy is important for early diagnosis

and appropriate management of such potentially rare adverse effects.

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Legends:

Figure 1: Gross ST elevation in inferior as well as anterior precordial leads in the 12 lead ECG

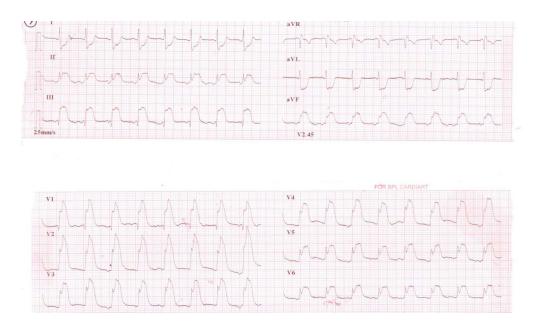
Figure 2: Second 12 lead ECG revealed slight reduction in the ST elevation, especially in the inferior leads and precordial leads V4-V6

Figure 3: A brief run of rapid ventricular tachycardia during the resolution phase of the ST elevation

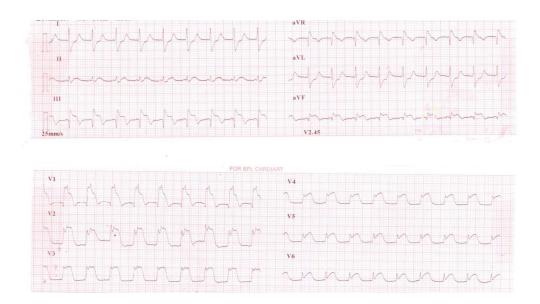
Figure 4: Progressive resolution of the ST segment elevation and normalization of the ST segment

Figure 5: Coronary angiogram performed revealed normal coronary arteries; Left coronary artery (left panel) and Right coronary artery (right panel)

Figure 6: LV angiogram demonstrating normal LV function with no regional wall motion abnormalities (Diastolic frame, left panel) and systolic frame (right panel).



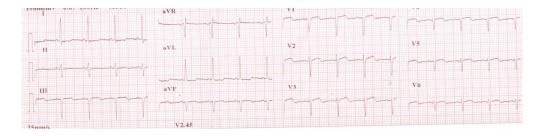
Gross ST elevation in inferior as well as anterior precordial leads in the 12 lead ECG 190x111mm (300 x 300 DPI)



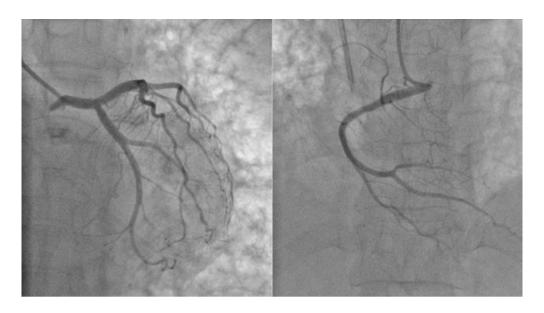
Second 12 lead ECG revealed slight reduction in the ST elevation, especially in the inferior leads and precordial leads V4-V6 $191 \times 104 \, \text{mm}$ (300 x 300 DPI)



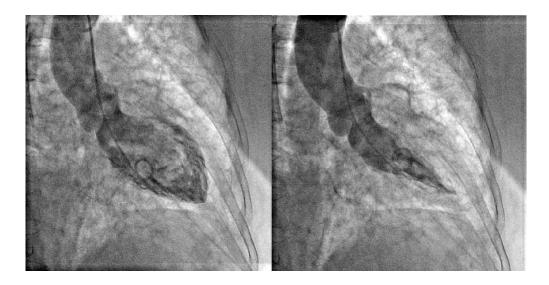
A brief run of rapid ventricular tachycardia during the resolution phase of the ST elevation 185x106mm (300 x 300 DPI)



Progressive resolution of the ST segment elevation and normalization of the ST segment 277x66mm (300 x 300 DPI)



Coronary angiogram performed revealed normal coronary arteries; Left coronary artery (left panel) and Right coronary artery (right panel) $54x30mm \; (300 \; x \; 300 \; DPI)$



LV angiogram demonstrating normal LV function with no regional wall motion abnormalities (Diastolic frame, left panel) and systolic frame (right panel). $280x143mm \; (300 \; x \; 300 \; DPI)$