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Case Report

Cor-pulmonale: a rare presentation in a case of middle lobe syndrome

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ABSTRACT

Brock's syndrome or middle lobe syndrome (MLS) is chronic or recurrent collapse of right middle lobe due to causes which may be obstructive or non obstructive. The pathogenesis is not completely understood. An expert committee of the world health organization defined cor pulmonale as hypertrophy of the right ventricle resulting from diseases affecting the function and/or structure of the lungs." Cor pulmonale is a common heart disease and a leading cause of disability and death. We are reporting this association in a 65y old female who presented to the emergency with acute exacerbation of COPD with SpO_2=64% room air. Chest X-ray and HRCT thorax showed features suggesting MLS and ECG shows features suggesting P pulmonale /right atrial enlargement. BNP too was elevated. Patient was resuscitated and put on mechanical ventilation after ABG showed respiratory acidosis. Appropriate treatment with bronchodilators and antibiotics was given.

Keywords: MLS, Cor pulmonale, Chest imaging, Case report

INTRODUCTION

The eyes cannot see what the mind does not know "is a common teaching every physician hears from his teachers and it is the high index of suspicion based on clinical history that helps in connecting dots between what is published in the texts and the missing gaps.

MLS is a rare encounter in clinical practice and was 1st identified clinically by Graham et al.[2] Brock was 1st to describe the recurrent collapse of right middle lobe due to enlarged tuberculous lymph nodes and thus syndrome is widely known as "Brock's syndrome".[3] Recurrent/fixed right middle lobe opacification is almost pathognomonic for MLS; however, lingular involvement is common due to similar anatomic and physiological burdens.

Females are more commonly affected than men and these women present later in age than men.[4,5] There are two distinct pathologies that lead to recurrent middle lobe collapse/ bronchiectasis: non obstructive and obstructive. The pathology of non-obstructive MLS is poorly understood and is probably due to transient hypoventilation in the setting of chronic inflammatory and infectious changes. The obstructive type of MLS can be due to intraluminal or extraluminal obstruction.

The most common etiology of the obstructive type is often subject to mediation by peribronchial lymphadenopathy secondary to granulomatous diseases, endemic fungal infections, and various mycobacterial species.[6] In developing nations like India tuberculosis is a major cause for such compression.

The fibro-bronchiectatic changes of the right middle lobe over the years can cause COPD and Cor-pulmonale which leads of morbidity and mortality of such patients.

The clinical symptoms associated with MLS includes chronic cough, hemoptysis, dyspnea, and features consistent with recurrent, difficult to treat pneumonia.

The D/Ds of MLS are COPD, ciliary disorders, cystic fibrosis, bronchiectasis, aspergillosis, asthma etc.

CASE REPORT

A 65-year-old female was referred to emergency facility, department of medicine from a PHC of Bihar with complaint of shortness of breath for the last 3 days which was present even at rest but increased on moderate activity, non-seasonal, more on lying down (Figure 1). She also had non-productive dry cough which was more in the early hours of the morning and diffused chest pain on right side of chest. Loss of appetite, malaise lethargy was present for last 2-3 months but had no significant loss of weight, no hemoptysis/fever was present. She had history of tuberculosis 20 years back for which she did't take complete course of ATT. No h/o type 2 DM, hypertension/thyroid disorder was present.

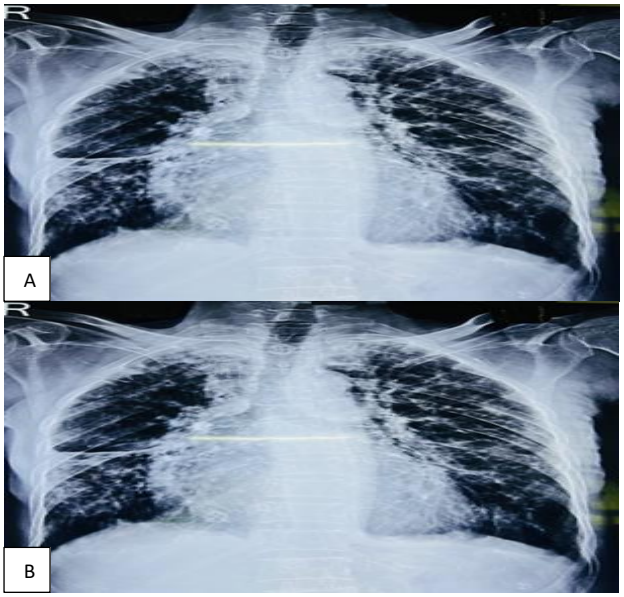

Figure 1 (A and B): Chest radiograph (posteroanterior view) showed fibrotic scarring with associated bronchiectatic changes diffusely in both lungs and an ill-defined opacity in right middle zone.

General physical examination revealed 65 years female of lean built in alert, conscious, co-op, agitated state, oriented to time, place, person and situation with GCS being E4V5M6. She was in acute respiratory distress with SpO_2 being=64% on room air. Her pulse was 84 bpm, regular, fair volume, normal character, no arterial wall stiffening, no radio-radial or radio-femoral delay and palpated in all peripheral arteries. Her BP 90/60 mmHg in supine position at level of heart. There was no pallor, cyanosis, clubbing, edema, icterus/lymphadenopathy.

Examination of the respiratory system revealed diminished breath sound over right middle lung zones, reduced vocal resonance in the right middle to lower zones and B/L crepts. CNS, CVS and per abdomen examination revealed no significant abnormalities.

Hemogram showed WBC=11.01, Hb=14.7, RBC=5.60, platelets=136, CRP=6.8, S. CR=1.39, SGPT=310, SGOT=268 and viral markers negative. The chest X-ray showed fibrotic scarring with associated bronchiectatic changes diffusely in both the lungs and an ill-defined opacity in right middle lobe, bronchovascular markings appear prominent on both sides with bilateral perihilar haziness; findings s/o post infective sequelae. The HRCT of thorax showed diffuse fibro-bronchiectatic and fibro-calcific changes with mild interstitial thickening in bilateral lung parenchyma and near complete fibro-bronchiectactic collapse of medial and lateral segment of right middle lobe, associated with few calcified mediastinal and right hilar lymph nodes; features were suggestive of sequelae to previous infection (Figure 4 A and B). The ECG 12 leads showed p pulmonale and right atrial enlargement (Figure 2). BNP was raised. USG was normal other than having a nonsignificant simple cortical cyst in right lower pole of kidney measuring 3×3.6 cm.

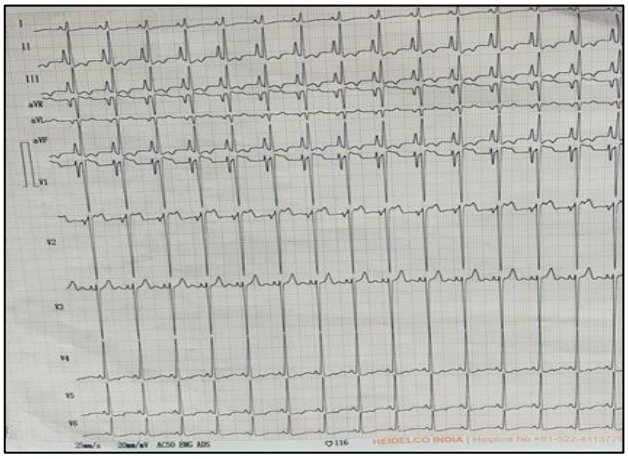

Figure 2: ECG 12 leads showed p pulmonale and right atrial enlargement.

The ABG showed respiratory acidodis for which the patient was intubated and put on mechanical ventilation along with bronchodilator as well as corticosteroids (Figure 3).

Figure 3: Patient mechanically ventilated.

Diagnosis of MLS, made on basis of clinical presentation supplemented by findings on HRCT thorax (Figure 4 C and D). Etiology of MLS-TB as documented from history. ECG findings-diagnosis of cor pulmonale.

The patient was treated with intravenous antiobiotics, nebulization with bronchodilators, low dose diuretics and was intubated and mechanically ventilated for respiratory acidosis. Patient improved on regime within 5-7 days.

Table 1: ABG analysis of patient.

Results			Crit Low	Reference Low	Reference High	Crit High
ABG on 23/4-acidosis resolved, pH=7.48, pCO2=40, HCO3=29.8.						
Measured (37º C)						
pH	↓7.48	mmHg	--	7.35	7.45	--
pCO_2	40	mmHg	--	32	48	--
pO_2	↑419	Mmol/L	--	83	108	--
Na^+	138	Mmol/L	--	136	145	--
K^+	3.8	Mmol/L	--	3.5	5.1	--
Cl	102	Mmol/L	--	98	108	--
Ca^{++}	↓0.83	Mmol/L	--	1.15	1.33	--
Hct	43	%	--	36	53	--
Glu	↑124	Mg/dl	--	74	101	--
Lac	↑2.7	Mmol/L	--	1.0	1.8	--
CO-oximetry						
tHb	14.0	g/dL	--	11.5	17.5	--
O_2Hb	96.2	%	--	94.0	98.0	--
COHb	0.0	%	--	0.0	3.0	--
MetHb	0.5	%	--	0.0	1.5	--
Hsb	↑3.2	%	--	0.0	2.9	--
SO_2	96.8	%	--	94.0	98.0	--
Derived						
TCO_2	31.0	Mmol/L	--	--	--	--
BEecf	6.3	Mmol/L	--	--	--	--
tHb(C)	14.6	g/dL	--	--	--	--
BE(B)	5.8	Mmol/L	--	--	--	--
Ca^{++} (7.4)	0.88	Mmol/L	--	--	--	--
AG	10	Mmol/L	--	--	--	--
PɎF ratio	Incalc	mmHg	--	--	--	--
pAO_2	Incalc	mmHg	--	--	--	--
CaO_2	20.0	mL/dL	--	--	--	--
O_2 cap	20.7	mL/dL	--	--	--	--
O_2 ot	20.0	mL/dL	--	--	--	--
sO_2 (c)	100.0	%	--	--	--	--
HCO_3 (c)	29.8	Mmol/L	--	--	--	--
HCO_3 std	29.4	Mmol/L	--	--	--	--
A-aDO_2	Incalc	mmHg	--	--	--	--
paO_2lpAO_2	Incalc	-	--	--	--	--
Rl	Incalc	-	--	--	--	--
CcO_2	Incalc	mL/dL	--	--	--	--
ABG on 21/4-respiratory acidosis, pH=7.07, pCO_2=117, HCO_3=33.						
Measured (37º C)						
pH	↓7.07	mmHg	--	7.35	7.45	--
pCO_2	↑117	mmHg	--	32	48	--
pO_2	↓55	Mmol/L	--	83	108	--
Na^+	↓130	Mmol/L	--	136	145	--
K^+	↑5.4	Mmol/L	--	3.5	5.1	--
Cl	↓88	Mmol/L	--	98	108	--
Ca^{++}	↓0.61	Mmol/L	--	1.15	1.33	--
Hct	↓ 21	%	--	36	53	--
Glu	85	Mg/dl	--	74	101	--
Lac	↑2.3	Mmol/L	--	1.0	1.8	--

Continued.

Results			Crit Low	Reference Low	Reference High	Crit Low
ABG on 21/4-respiratory acidosis, pH=7.07, pCO_2=117, HCO_3=33						
CO-oximetry						
tHb	↓ 6.7	g/dL	--	11.5	17.5	--
O_2Hb	↓84.7	%	--	94.0	98.0	--
COHb	1.4	%	--	0.0	3.0	--
MetHb	0.0	%	--	0.0	1.5	--
Hsb	↑13.9	%	--	0.0	2.9	--
SO_2	↓85.9	%	--	94.0	98.0	--
Derived						
TCO_2	37.5	Mmol/L	--	--	--	--
BEecf	3.8	Mmol/L	--	--	--	--
tHb(C)	7.1	g/dL	--	--	--	--
BE(B)	3.1	Mmol/L	--	--	--	--
Ca^{++} (7.4)	0.53	Mmol/L	--	--	--	--
AG	14	Mmol/L	--	--	--	--
PɣF ratio	Incalc	mmHg	--	--	--	--
pAO_2	Incalc	mmHg	--	--	--	--
CaO_2	8.1	mL/dL	--	--	--	--
O_2 cap	9.4	mL/dL	--	--	--	--
O_2 ot	8.1	mL/dL	--	--	--	--
sO_2 (c)	73.5	%	--	--	--	--
HCO_3 (c)	33.9	Mmol/L	--	--	--	--
HCO_3 std	27.2	Mmol/L	--	--	--	--
A-aDO_2	Incalc	mmHg	--	--	--	--
paO_2lpAO_2	Incalc	-	--	--	--	--
Rl	Incalc	-	--	--	--	--
CcO_2	Incalc	mL/dL	--	--	--	--

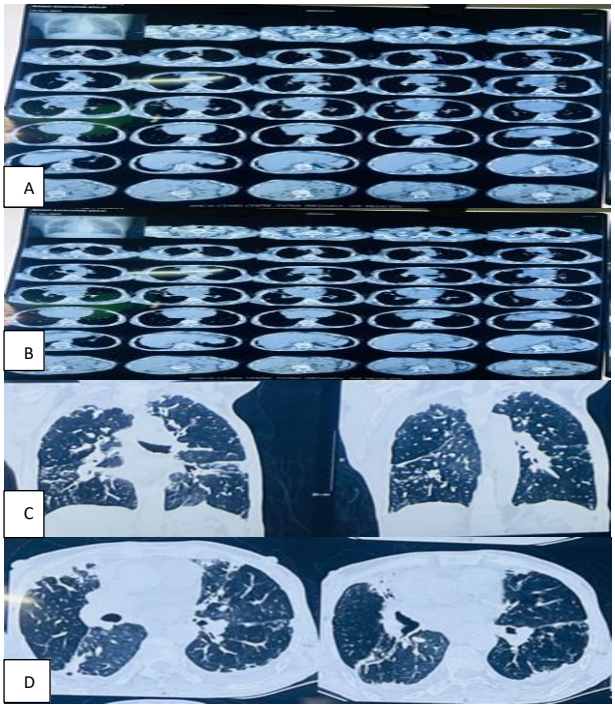

Figure 4 (A-D): HRCT thorax-diffuse fibro-bronchiectatic and fibro-calcific changes with mild interstitial thickening in bilateral lung parenchyma. Near complete fibro-bronchiectactic collapse of medial and lateral segment of right middle lobe.

DISCUSSION

MLS is classified into obstructive and non-obstructive types. Obstruction can further be due to intraluminal or extraluminal cause and is thought to be a feature characteristic of MLS.[7] Primary tumours account for 24% while tuberculosis is responsible for 8-10%.[8] In an earlier study from Poland, MLS was observed in 25% of the elderly females with tuberculosis.[9]

It is hypothesised that middle lobe has a greater susceptibility to collapse as its bronchus is rather narrow where it originates. The long length as well as the angular takeoff of this bronchus makes it liable to be compressed due to enlarged lymph nodes. In addition, poor collateral ventilation as a consequence of the anatomical separation of the middle lobe from the right upper and lower lobes by fissures along with inadequate clearance of the impacted mucous can also contribute to the propensity to collapse in isolation. In our patient fibrocalcific changes lead to external compression of the bronchus leading to MLS. Imaging is important to diagnose MLS-the chest radiograph showing obliteration of the right cardiac border (silhouette sign) due to the proximity of the medial segment of the middle lobe with the right atrium or fibro calcification changes in the right middle lobe of lung. The presence of trapezoidal or broad triangular opacity with base towards the hilum, contiguous with the right cardiac border on CT thorax confirms the diagnosis

of MLS. With the disease process set in, fibro calcific changes of the lungs can lead to COPD with the involvement of bronchial tree in MLS. If the disease process causes increased pulmonary vascular resistance, it can lead to right ventricular dilation and dysfunction resulting in cor-pulmonale as seen in our patient

CONCLUSION

In conclusion, though the pathogensis of MLS is not fully understood it still intrigues a physician on how an entity can have a devastating impact on the health of a patient. A high index of suspicion would be required to establish the diagnosis. Early recognition and appropriate therapy can possibly prevent complications of this disease and prevent diseases like cor pulmonale in long run.

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