MIDDLE LOBE SYNDROME - PUTTING THE CART BEFORE THE HORSE?


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SUMMARY

Middle lobe syndrome is an uncommon condition. Reports of this condition in West African literature are rare.

We report a 34 year old male patient seen with right-sided chest pain and persistent haemoptysis. Plain chest radiographs showed features of middle lobe consolidation. Several investigations carried out could not elucidate the primary aetiology of the consolidation, except for an elevated erythrocyte sedimentation rate of 100mm/hour. A clinical diagnosis of florid pulmonary tuberculosis was only arrived at during surgery. A bilobectomy was performed. Tuberculosis as the primary aetiology was confirmed by histology. Post-operative anti-tuberculous chemotherapy resulted in cure.

This report highlights the limitations in using the methods detailed in our report below.

Key words: Middle lobe syndrome- lymphadenopathy- middle lobe bronchus- atelectasis.

RESUME

Le syndrome du lobe moyen est une pathologie rare ; il a été rarement rapporté dans la littérature ouest-africaine.

Nous rapportons l’histoire médicale d’un patient de 34 ans de sexe masculin qui présentait une douleur thoracique droite et une hémoptysie persistante. La radiographie du thorax montrait des images de condensation du lobe moyen droit. Les différentes investigations n’ont pu éclaircir l’étiologie de cette condensation radiologique, en dehors d’une élévation de la vitesse de sédimentation (100 mm/h). Le diagnostic clinique de tuberculose pulmonaire évolutive a été l’indication opératoire. Une bilobectomie a été pratiquée et le diagnostic de tuberculose a été confirmé à l’histologie. Ainsi, une chimiothérapie antituberculeuse post-opératoire a permis une guérison. Ce travail met en lumière les limites des méthodes utilisées et détaillées dans le cas clinique ci-dessous décrit.

Case report
A 34 year old male presented with a four month history of cough productive of creamy sputum, occasional haemoptysis and right-sided chest pain. The cough was particularly worse at night and kept him awake. Associated with the above symptoms were night sweats and significant weight loss. He did not recall contact with anybody with chronic cough.

The referring hospital had on two occasions evaluated his sputum for acid fast bacilli using standard World Health Organization (WHO) protocols. All proved negative. Tuberculin skin test with 0.1ml of 0.005% (5 TU) purified protein derivative was also negative, i.e. less than 5mm of induration after 72 hours. He therefore had conventional antibiotic treatment for middle lobar pneumonia. Cardiothoracic opinion was sought when there was neither clinical nor radiological resolution after two courses of parenteral Augmentin™ followed by the oral formulation.

Physical examination revealed clinical evidence of consolidation of the right middle zone anteriorly. The blood profile was normal except for elevated erythrocyte sedimentation rate of 100mm/hr (Westergreen™). Sputum examination revealed Proteus sp, which was treated with cefuroxime, the appropriate antibiotic on sensitivity testing.

Serial plain chest radiographs on postero-anterior view (Fig. 1), revealed an ill-defined opacity abutting the right cardiac border leading to loss of cardiac silhouette (silhouette sign), the lateral view (Fig. 2) further helped in localization of an opacity with rectangular shape overlying the upper cardiac silhouette.

A chest CT scan (Fig. 3) confirmed the plain radiographic findings, and in addition demonstrated right hilar lymphadenopathy. Flexible fibre-optic bronchoscopy showed extrinsic compression of the right middle lobar bronchus. The mucosa was normal. However, cytology of brush biopsy and bronchial washings revealed cells with features of malignancy. At this point the patient was planned for urgent surgery to forestall any further delay.

Fig. 1. Postero-anterior radiograph showing ill-defined density abutting right cardiac border

Fig. 2. Lateral chest radiograph showing rectangular density overlying upper part of cardiac silhouette
Preoperative pulmonary function test revealed a restrictive lung picture with mildly reduced lung volumes (Table 1).

Table 1: Spirometry Results

<table>
<thead>
<tr>
<th>Test Values</th>
<th>Predicted Values</th>
<th>Percentage of Predicted Value</th>
</tr>
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<tbody>
<tr>
<td>FEV1 (L)</td>
<td>3.37</td>
<td>2.34</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>3.97</td>
<td>3.04</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>81%</td>
<td>77%</td>
</tr>
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</table>

He had double-lumen endotracheal intubation and standard right posterolateral thoracotomy. Findings were extensive caseation of the middle lobe with significant involvement of contiguous basilar portions of the upper lobe and a small area of the apical part of the lower. There was also a cuff of enlarged lymph nodes around the right middle lobar bronchus extending into the right hilum. Standard upper and middle bi-lobectomy was performed. Postoperatively, he had anti-tuberculous chemotherapy using the W.H.O. guidelines on Directly Observed Therapy. Short course (DOTS), i.e. 2SHRZ/6HT.

He was discharged home on the 7th post-operative day to continue with the anti-tuberculous therapy. Histopathology of the surgical specimen confirmed florid pulmonary tuberculosis. He remains well after two years of follow-up (Fig. 4).

Discussion

Middle lobe syndrome (MLS) generally refers to atelectasis of the middle lobe of the right lung. It occurs in all age groups and is divided into an obstructive type, with a demonstrable airway occlusion, and a non-obstructive type, with a patent middle lobe bronchus apparent on bronchoscopy.

Middle lobe syndrome is characterized by a wedge-shaped density that extends anteriorly and inferiorly from the hilum of the lung, best visualized on lateral chest radiographs. Certain anatomical characteristics make the middle lobe susceptible to transient obstruction as a result of extrinsic compression, inflammation or oedema. The narrow diameter of the lobar bronchus and acute take-off angle create poor conditions for drainage. Relative anatomical isolation of the middle lobe and poor collateral ventilation decrease the chance of reinflation once atelectasis occurs.

The true incidence is unknown. It is widely under-diagnosed. It is for this reason that we share our experience.
The aetiology is varied and has no consistent clinical definition. It was first described by Graham and co-workers in 1948 in 12 patients who had collapse of the middle lobe from compression of the lobar bronchus as a result of lymphadenopathy of non – tuberculous origin. However, the basic underlying pathology, irrespective of aetiology, is now broadened to include all atelectasis of the middle lobe of the lung, with or without obstruction of the middle lobar bronchus.

Bronchial obstruction can result from extrinsic compression from enlargement of the cuff of lymph nodes which surround the bronchus, as from benign inflammatory conditions, or tumour of neoplastic origin. However, atelectasis in children usually results from a process such as asthma-associated oedema and inflammation from infection and allergic processes. Foreign body aspiration into the middle lobar bronchial orifice can also predispose to collapse of the lobe.

Other rare reported cases are middle lobe syndrome as the pulmonary manifestation of Sjögren syndrome, silicosis, traction deformities of the oesophagus, bronchopulmonary dysplasia and cardiovascular anomalies.

In the case being reported, computerised tomography scan (Fig. 3) clearly demonstrated hilar lymphadenopathy. Tuberculosis was naturally the foremost differential. The patient was investigated for this but all the tests proved negative except for the ESR of 100mm/hour. A negative tuberculin skin test is possible in the setting of immuno-suppression. There was no clinical or laboratory evidence for this; HIV screen (HIV 1 / 2 Tri-Line™, ONE STEPT™, qualitative assay) was negative, and serum proteins were within normal range. Polymerase chain reaction for mycobacterium tuberculosis was not done as it is not routinely available in our laboratories. Its availability may have changed the course of management.

Management of this condition depends on the cause. Conservative measures employed include conventional antibiotic therapy for bacterial infections, glucocorticoid management for aetiologies of allergic origin, mechanical ventilation with nasal continuous positive airway pressure (nCPAP), and fibre optic bronchoscopy with bronchoalveolar lavages. In the presence of bronchiectasis, bronchial stenosis, significant lung destruction from granulomatous infection, failure of lung to re-expand after adequate medical therapy and malignant tumours, early pulmonary resection is indicated. In this reported case, definitive evidence of the aetiology was only arrived at during surgery.

In this case, a pre-operative therapeutic trial of anti-tuberculous chemotherapy would have been the preferred course of management since most cases of middle lobe syndrome result from a benign aetiology, and specifically tuberculosis in most third world countries from where such cases have been reported. This was the original plan. It was however changed in favour of early surgery when the cytology report suggested the presence of malignant cells. The need to be vigilant for tumourigenesis in association with middle lobe syndrome has been stressed. The difficulties inherent in interpretation of cytological details are well recognised. This fact notwithstanding, we chose to err on the side of surgery rather than conservative management, hence the choice of urgent surgery as our primary treatment modality. Though all three lobes were involved in the pathologic process at surgery, the involvement of the lower lobe was thought to be minimal enough to resolve on chemotherapy alone.

Given prior chemotherapy, a less extensive resection may have been afforded or surgery avoided altogether. The patient made uneventful post-operative recovery, resulting in his discharge on the 7th post-operative day. It may therefore seem as if we put the cart before the horse. He remains well two years after follow-up.
Conclusion
The case highlights the difficulty in confirming a suspicion of tuberculosis using the methods detailed above. It also confirms that tuberculosis is an important consideration in the aetiology of the middle lobe syndrome in our sub-region.
Where exhaustive attempts at isolation of the tubercle bacilli are lacking, a prudent trial of chemotherapy constitutes a valid investigative option before the decision for lung resection is made.

References
2. Eid N S.; Eckerle M. Right middle lobe syndrome: Emedicine 2007; Oct. 17
4. Graham EA.; Burford IH.; Mayer JH. Middle lobe syndrome. Postgrad Med 1948; 4: 29-33
8. Tsuichiya M.; Katsuki Y.; Enokibori T.; Ninomiya K.; Fujimura N. Two cases of chronic atelectasis that improved through use of nasal continuous positive pressure: Nihon Kokyuki Gakkai Zasshi 2007; 45(6): 503-7
9. Zhu QS.; Peng LJ. Etiologic analysis of 12 cases of silicosis with right middle lobe atelectasis. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi 2004; 22(1): 70
10. Masahide Kawamura; Yasumichi Arai; Masato Tani. Improvement in right lung atelectasis (Middle Lobe Syndrome) following administration of low-dose Roxithromycin. Respiration 2001; 68: 210-214