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Isolated middle lobe atelectasis: aetiology, pathogenesis, and treatment of the so-called middle lobe syndrome

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ABSTRACT Isolated atelectasis of the middle lobe has been known for many years as the "middle lobe syndrome". Several clinical studies have shown that it may be caused by malignant tumours. A 10-year study of 135 patients with isolated middle lobe atelectasis is presented. Fifty-eight patients (43%) had malignant tumours. Of 38 who had a thoracotomy, lung resection was possible in 25. In 20 patients regional or systemic dissemination of the tumour had been diagnosed before operation. Seventy-seven patients had benign diseases, of which 74 were non-specific infections. Bronchography was performed in 46 of these cases, and all had abnormal findings in the middle lobe, eight revealing definite bronchiectasis. In three cases tuberculosis was found. In 16 cases the benign diagnosis was established at thoracotomy. Only three patients out of 58 with malignant tumours lived more than five years. Atelectasis of the middle lobe is always a sign of potential malignancy especially in patients with a previously normal chest radiograph.

Isolated atelectasis of the middle lobe or "middle lobe syndrome" is a well-recognised entity.[1] Several clinical studies in recent years have demonstrated that the middle lobe syndrome may be caused by malignant neoplasms. To determine the incidence of causes of the middle lobe syndrome, we have collected records for patients admitted with isolated middle lobe atelectasis during the period 1963-73 to the departments of thoracic surgery in Rigshospitalet and Bispebjerg Hospital, Copenhagen, Denmark.

Patients

During the years 1963-73 a total of 135 patients with middle lobe atelectasis were admitted to the two departments. A retrospective analysis of the case records was made, and follow-up examinations were carried out whenever possible.

All the patients showed typical middle lobe atelectasis on plain chest radiographs. The atelectasis was the only abnormal parenchymal finding, but 17 patients also had a slightly increased mediastinal width and a few had slight pleural reaction.

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The figure gives the age distribution and sex ratio. Sixteen patients had an incidental finding of asymptomatic atelectasis of the middle lobe. The remaining patients had varying degrees and combinations of cough, sputum, haemoptysis, fever, and pain.

The diagnostic findings are shown in table 1. Bronchoscopy was performed in 121 patients and bronchography in 63. Almost 43% of the patients had malignant disease, and rather more than 2% tuberculosis, while in the remaining cases the atelectasis was caused by non-specific inflammation.

MALIGNANT DISEASE

Of 58 patients with malignant tumours 53 were examined by bronchoscopy. In 38 the diagnosis was made by biopsy, while in seven patients tumour cells were found in secretions aspirated in the course of bronchoscopy. In eight patients bronchoscopy failed to yield a diagnosis.

Total obstruction was found in the middle lobe bronchus in 16 of 17 patients examined by bronchography. Fifteen patients had slightly increased

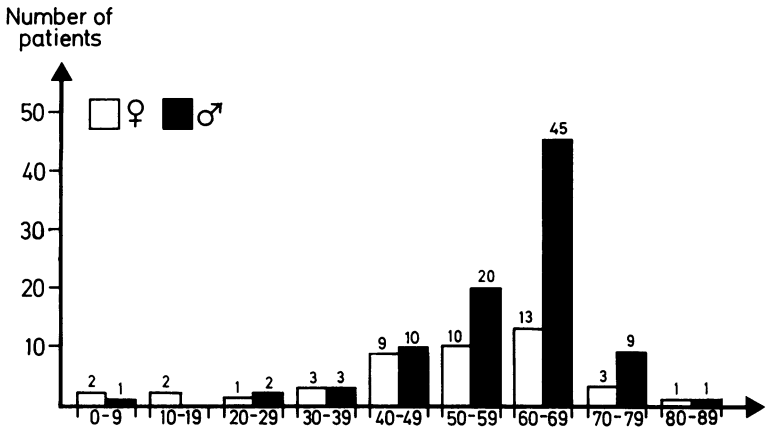

Figure *Age and sex distribution of 135 patients with middle lobe atelectasis.*

mediastinal width.

A total of 38 patients underwent operations, which are listed in table 2.

The diagnosis was made before operation in 29 of the 38 patients, but in nine at operation. These nine patients had no previous symptoms, and bronchography showed total or subtotal obstruction of the middle lobe bronchus while the remaining bronchial tree was normal.

Resection was performed in 25 patients, but in 16 of these lymph node metastases were found in lobar nodes, nodes along the main bronchus, or in the mediastinum. In nine patients the operation was considered radical.

The remaining 13 patients who underwent thoracotomy had large lymph node metastases or invasion of the mediastinum by the tumour or both, so that the operation was terminated without resection.

Twenty patients with malignant disease were not subjected to thoracotomy. In 17 intractable growth of tumour had been disclosed clinically. Eight patients had lymph node metastases in the mediastinum or in the neck or both, five had cerebral and four hepatic metastases.

In three patients the middle lobe atelectasis was at first interpreted as being of infectious origin, but this primary diagnosis had to be revised in a few months when it was realised that the patients had malignant disease, which at that time was beyond surgical treatment.

Only three of the resected patients survived for more than five years. All the others have died of their tumours.

Table 1 *Diagnostic findings in 135 patients with middle lobe atelectasis*

Examination	*Findings*	*Malignant*	*Benign*
Bronchoscopy (n = 121)	Tumour-obstruction	38/53	
	Narrowing		19/68
Bronchography (n = 63)	Total obstruction	16/17	
	Narrowing		37/46
	Ectasia		8/46
	Close bronchial arrangement		19/46
Bronchial secretion (n = 68)	Cytology	7/53	
	Specific culture		2/68

Table 2 *Treatment of 135 patients with middle lobe atelectasis*

Treatment	*Malignant*	*Benign*
Surgical		
Pneumonectomy	16	1
Middle lobectomy	9	10
Bilobectomy		5
Exploratory thoracotomy	13	
Medical		
Palliative treatment	20	
Lung physiotherapy and antibiotics		61

BENIGN DISEASES

Of the 77 patients with benign diseases 68 were examined by bronchoscopy, but only 19 were found to have narrowing of the middle lobe orifice or middle lobe bronchus or both. In one patient biopsy revealed tuberculous granulations around the middle lobe orifice. The bacteria most commonly found in the bronchial secretions were pneumococci and *Pfeiffer's bacillus.* In two patients tubercle bacilli were found in secretions aspirated during bronchoscopy.

Bronchography was performed in 46 patients. The most common finding was irregular narrowing of the middle lobe bronchus and of the segmental bronchi, while only eight patients had definite dilatation of these bronchi. In 19 patients the two segmental bronchi were of irregular calibre and also lying close together, indicating

bronchitis and shrinkage of the middle lobe.

In addition to the middle lobe atelectasis, two patients had a slightly increased mediastinal width.

The treatment of the benign conditions may be seen from table 2. Sixteen patients were operated on. All had middle lobe atelectasis with no response to medical treatment. In five of these bilateral bronchography showed in addition bronchiectasis in the right lower lobe. Lobectomy was carried out in 15 cases, of the middle lobe in 10 and of the middle and lower lobes in five in whom bronchiectasis had been demonstrated before operation. One patient had an emergency pneumonectomy because of profuse haemoptysis. This patient was suffering from tuberculosis, and bronchoscopy revealed fresh haemorrhage from the right main bronchus.

Table 3 shows the pathological findings in the middle lobe of the 16 patients who underwent operation for benign diseases.

Sixty-one patients with middle lobe atelectasis were treated by physiotherapy and antibiotics. In 42 patients the middle lobe atelectasis disappeared between two and 20 weeks. At follow-up all these patients were without symptoms. Chest radiographs were normal in 30 cases while 12 had slight pleural thickening.

In 19 patients with chronic bronchitis and bilateral changes seen at bronchography the middle lobe atelectasis remained unchanged for more than five months. In spite of this, these patients were not operated on, since the aetiology of the disease seemed to be infectious because of bilateral diffuse changes in the lung as well as the middle lobe atelectasis. Several of the patients were followed for many years. In three patients the middle lobe atelectasis was unchanged after between five and 20 years.

Eight of the nine patients who were operated on became symptom-free, but one still had symptoms of bronchitis.

The final aetiology of the middle lobe atelectasis is shown in table 4. A preoperative diagnosis was made in 29 patients (21%), while in 25 cases (19%) the diagnosis was not disclosed until after the operation. Nine of the latter group had a malignant tumour.

Table 3 *Pathological findings in 16 operated cases with benign diseases*

Finding	*Number*
Chronic non-specific infection	14
Lipoid pneumonia	1
Tuberculosis	1
	16

Table 4 *Aetiology in 135 patients with middle lobe atelectasis*

Aetiology	*Number*	*%*
Benign (n=77)		
Non-specific infection	74	57
Tuberculosis	3	
Malignant (n=58)		
Epidermoid carcinoma	38	
Anaplastic carcinoma	15	43
Adenocarcinoma	5	
	135	

Discussion

In 1937 it was demonstrated by Brock *et al*[2] that middle lobe atelectasis in tuberculosis was caused by compression and/or erosion of the middle lobe bronchus by peribronchial tuberculous lymph nodes. In an anatomical study of the bronchial tree Brock[3] further demonstrated that the lymph nodes around the middle lobe bronchus take part in the lymphatic drainage of the right lower lobe.

Zdansky[4] showed that lymph node compression of the middle lobe bronchus might be caused not only by tuberculous lymph nodes, but also by lymph nodes with non-specific infection. In his paper he calls the middle lobe bronchus a "punctum minoris resistentiae". This was confirmed by Graham *et al*[1] who were the first to introduce the term "middle lobe syndrome" for isolated middle lobe atelectasis caused by lymph node compression.

In a convincing paper Culiner[5] discussed the pathogenesis of the middle lobe syndrome. He claimed that the main cause of the frequent occurrence of middle lobe atelectasis was anatomical separation of the middle lobe from the upper as well as lower lobe and a consequent lack of "collateral ventilation".

In 1950 Rubin and Rubin[6] pointed out that a "shrunken middle lobe" might be caused not only by bronchial compression by lymph nodes, but also by benign and malignant tumours or by bronchiectasis.

Several publications in the past decade have confirmed that the aetiology of the middle lobe syndrome is malignant neoplasm in 13–30%.[7-12]

In a study of 77 patients with middle lobe atelectasis Camishion *et al*[9] pointed out that in the absence of bronchial obstruction, the condition is caused by secondary changes from bronchitis, pneumonitis, and the like, while atelectasis

in the presence of bronchial obstruction resulted from specific and non-specific lymphadenitis or malignant tumour.

The present series confirms the high frequency of a malignant aetiology in middle lobe atelectasis. Forty-three per cent of the patients had bronchogenic carcinoma, distributed as 66% epidermoid, 26% anaplastic, and 8% adenocarcinoma.

The clinical diagnosis is based mainly on bronchoscopy, but it must be emphasised that in the present trial bronchoscopy showed nothing directly suggestive of tumour in 25% of the patients (13 patients). Such false negative findings have also been reported by others.[8 9] According to several authors biopsy from the middle lobe orifice is of little value in cases with a normal-looking bronchus.

Bronchography showed total obstruction of the middle lobe bronchus in a high percentage of patients with malignancy in our series. In benign conditions bronchographic findings are variable, including narrowing of the middle lobe and segmental bronchi, and segmental bronchi in close proximity indicating shrinkage of the middle lobe.

The middle lobe syndrome is a radiological syndrome, and the clinician can never exclude carcinoma on the basis of the radiological examination. Fibreoptic bronchoscopy and percutaneous lung biopsy should be used more widely in cases without a definite aetiological diagnosis.

If complete investigations, especially in a patient with previously normal lungs developing middle lobe atelectasis, fail to afford a diagnosis and the atelectasis persists, an exploratory operation should be undertaken without delay. Moreover, if conservative therapy has been started and does not rapidly lead to improvement, there is also an indication for exploratory thoracotomy.

References

1 Graham EA, Burford TH, Mayer JH. Middle lobe syndrome. *Postgrad Med* 1948; **4**:29–34.

2 Brock RC, Cann RJ, Dickinson JR. Tuberculous mediastinal lymphadenitis in childhood: secondary effects on the lungs. *Guy's Hosp Rep* 1937; **87**: 295–9.

3 Brock RC. *The anatomy of the bronchial tree.* London: Oxford University Press, 1947.

4 Zdansky E. Der mittellappen als "punctum minoris resistentiae" der Lunge. *Wien Klin Wochenschr* 1946; **58**:197–204.

5 Culiner MM. The right middle lobe syndrome, a non-obstructive complex. *Dis Chest* 1966; **50**: 57–66.

6 Rubin EH, Rubin M. The shrunken right middle lobe. *Dis Chest* 1950; **18**:127–45.

7 Albo RJ, Grimes OF. The middle lobe syndrome: a clinical study. *Dis Chest* 1966; **50**:509–18.

8 Bross W, Kaniowski T, Rogalski E, Kozuszek W. The so-called middle lobe syndrome. Diagnosis and results of treatment. *Bull Soc Int Chir* 1970; **29**:182–5.

9 Camishion RC, Davies AL, Ballinger WF. Isolated disease of the middle lobe and lingula. *Dis Chest* 1966; **50**:67–71.

10 Šimeček C. Middle lobe syndrome. *Respiration* 1970; **27**:100–3.

11 Geroulanos S, Hahnlosen P, Jenny M, Senning Å. Beitrag zur Malignität des Mittellappensyndroms. *Dtsch Med Wochenschr* 1973; **98**:1095–9.

12 Banyai AL. The middle lobe syndrome and its quasi variants. *Chest* 1974; **65**:135.