Mediastinal tuberculous lymphadenitis with anthracosis as a cause of vocal cord paralysis

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ÖZET

Vokal kord paralizisi sebebi olarak mediastinal tüberküloz lenfadenit ve antrakozis birlikteliği


Anahtar Kelimeler: Ses kısıklığı, vokal kord paralizisi, mediastinal tüberküloz lenfadenit, pulmoner antrakozis.

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409 Tüberküloz ve Toraks Dergisi 2007; 55(4): 409-413
Anthracosis, a form of pneumoconiosis, is most commonly seen in coal workers (1). Air pollution, biomass smoke and cigarette smoke are also known as the other environmental factors for anthracosis (2-4). Anthracosis often causes intrapulmonary lymphadenopathy but it rarely causes mediastinal or axillary lymphadenopathy (5,6). Dark anthracotic pigmentation in the bronchial mucosa has been regarded as a bronchoscopic finding of pneumoconiosis or evidence of heavy atmospheric soot. Anthracotic pigmentation with bronchial narrowing or obliteration, surrounded by calcified or noncalcified lymph nodes is typical finding of anthracofibrosis. There is a potential relationship between bronchial anthracofibrosis and tuberculosis. Tuberculous lymphadenopathy of superior mediastinum presentation with hoarseness is very rare. The paper reports a case of tuberculous mediastinal lymphadenitis with anthracosis causing vocal cord paralysis. A 66-year-old woman was admitted to our clinic with the symptoms of dry cough, hoarseness, malaise, anorexia, night sweats and with the multiple mediastinal lymphadenopathy. Fiberoptic bronchoscopy revealed left vocal cord paralysis, bronchial mucosal inflammation and multiple anthracotic plaques. Bronchial lavage and mucosal biopsy were negative for malignancy and tuberculosis. The thoracotomy was performed and a mediastinal lymph node showing caseating granulomatous inflammation with anthracosis and parenchymal anthracosis were detected. The diagnosis of anthracosis and mediastinal tuberculous lymphadenitis was made and the patients put on antituberculous treatment. But she was unfortunately died in the second month of the treatment because of the abdominal complication of gastric adenocarcinoma operation.

Key Words: Hoarseness, vocal cord paralysis, mediastinal tuberculous lymphadenitis, pulmonary anthracosis.
platelet: 493,000/mm³, ESR: 60 mm/hour, total protein: 8.4 mg/dL, albumin: 5.2 mg/dL and all other biochemical parameters were in normal limits. The Mauntoux test was strongly positive for tuberculosis (20 mm). Her chest X-ray revealed left hilar calcification consistent with old tuberculosis (Figure 1). Computed tomography (CT) of the neck was normal, but thorax CT revealed a few millimetric pulmonary nodules and multiple lymphadenopathy which were localized pretracheal, prevascular and hilar regions (Figure 2). Fiberoptic bronchoscopy revealed left vocal cord paralysis, and diffuse bronchial inflammatory appearance accompanied by anthracotic plaques localized on the multiple lobar orifices. Bronchial lavage was negative for acid fast bacilli and negative for malignancy. The histopathological examination of transbronchial lung biopsy revealed interstitial fibrosis and anthracosis, but no granuloma formation or malignancy. Thereafter, the Löwenstein-Jensen culture of bronchial lavage remained negative. As definite diagnosis could not be reached with these procedures, thoracotomy was performed as a diagnostic procedure. Right paratracheal lymphadenopathy and a 2 mm intraparenchymal nodule observed during thoracotomy were extracted. Histopathological examination of these two materials revealed a fibrotic nodule surrounded by parenchymal tissue with anthracotic pigmentation and a lymph node showing caseating granulomatous inflammation with anthracosis (Figure 3). In the view of these findings the diagnosis of pulmonary anthracosis and mediastinal tuberculous lymphadenitis was made and patient was put on antituberculous treatment namely, isoniazide, rifampicin, pyrizinamide, and ethambutol along with corticosteroids administered in tapering doses. The patient was readmitted to emergency room with the symptoms suggesting acute abdomen such as stomach ache, nausea and vomiting on the second month of the treatment. Histopathological examination of endoscopic biopsy revealed antral gastric adenocarcinoma. She was unfortunately died after a short time from the diagnosis due to postoperative complication of total gastrectomy.

DISCUSSION

Anthracosis is mostly seen in coal workers due to accumulation of coal dust in lung, but it can also occur due to exposure to air pollution and biomass or cigarette smoke (1-5). Anthracosis often causes intrapulmonary lymphadenopathy but rarely mediastinal mass or lymphadenopathy (6). Beside this, intrathoracic lymph nodes are often anthracotic in elderly persons (11). In the course of erosion into the bronchus carbon particles in the lymph node may penetrate through the bronchial wall and the bronchial mucosa may be colored. On the other hand, neither accumulation of anthracotic pigment from air pollutants nor smoke induces focal bronchial abnormality, because carbon is inert and elicits little or no fibrosis. Intrabronchial perforation of tuberculous lymphadenitis is the other well-known cause of anthracotic pigmentation in the bronchial mucosa (12,13). In the study of Chung et al., the patients showing only anthracotic pigmentation on bronchial mucosa without
bronchial stenosis were excluded, and the characteristic features of the patients with anthracofibrosis were described as follows (8):

1. Preponderance of older female patients,
2. No association with pneumoconiosis or smoking,
3. Chief complaint of cough and dyspnea without constitutional symptoms,
4. Segmental or lobar consolidation on simple chest radiographs,
5. Abnormalities of bronchial airways with peribronchial cuffs of soft tissue or surrounding lymph nodes on chest CT,
6. Most frequent involvement of the right middle lobe bronchus,
7. Active tuberculous infection demonstrated in >60% of the patients.

In our case, she had no history of occupational dust exposure, but air pollution is rampant and biomass usage is highly prevalent in Isparta, a rural town in Lakes region of Turkey where she used to live. In bronchoscopic examination we did not find any sign of endobronchial tuberculosis or anthracofibrosis. The pathological findings revealed paranchymal anthracosis but there was no evidence indicating malignancy or tuberculosis. So, it was assumed that bronchoscopic findings might be due to air pollution or biomass usage.

The next step in the differential diagnosis of mediastinal lymphadenopathies is mediastinoscopy. Instead of mediastinoscopy, thoracotomy was performed for excisional biopsy because mediastinoscopy can not be performed in our center. The histopathological examination of biopsies showed tuberculous lymphadenitis with anthracosis.

Tuberculous lymphadenitis is one of the most common forms of extrapulmonary tuberculosis and the first lymphoid tissues encountered during spread are presumably hilar and mediastinal lymph nodes (14). However, mediastinal lymph nodes are rarely reported at the site of tuberculous lymphadenitis and it is also very rare in adults (14).

Clinical conditions are quite variable depending on the affected structures in patients with mediastinal tuberculosis. Hoarseness resulting from involvement of RLN, dysphagia due to compression of esophagus, and typical manifestations of superior vena cava syndrome resulting from involvement of superior vena cava can be seen (9,10,14-17).

In developed countries mediastinal lymphadenopathy with vocal cord dysfunction is generally related to malignant process (15). Tuberculosis is rarely reported in the etiopathogenesis of hoarseness, although it is frequently seen in the developing countries like ours (9). Entrapment in scar tissue, traction by fibrosis, or compression with enlarged lymphadenopathy lodged in the prevascular or aortopulmonary window should be discussed in the pathology of paralysis of RLN. Fowler and Hetzel suggested that direct spread of infection from perforated lymph node abscess damage the RLN rather than mechanical factors alone (15). Functional damage of
RLN caused by direct spread of the infection from caseous necrosis in the mediastinum is irreversible, but it is complete reversible with antituberculous treatment if it has been resulted from compression (10). Although it is suggested that compression of enlarged lymph nodes is the cause of RLN involvement, we couldn’t evaluate any response to the antituberculous therapy because of the early death of our patient.

As the detection of acid fast basilli in microscopy and culture of the material other than sputum is very low; the diagnosis of mediastinal tuberculous lymphadenitis is quite difficult. The Mantoux test is found positive in 90% of patients with tuberculous lymphadenitis, as in our case. About 30% of chest X-rays show abnormality consistent with sequelae of involvement of right paratracheal and tracheobronchial lymph nodes are frequent (14). After injection of contrast medium, central low attenuation and peripheral rim enhancement of nodes larger than 2 cm in diameter is highly suggestive for active tuberculous lymphadenitis (18-20). Similar to the anthracofibrotic patients of Chung et al., multiple mediastinal lymphadenopathies were detected in our case without low attenuation and rim enhancement (8). The radiological absence of low attenuation and rim enhancement may be due to the deposition of the coal dust in lymph nodes.

In conclusion, we decided to present this case report because;

1. Hoarseness is an unusual presentation in the tuberculous lymphadenitis,
2. In contrast to anthracofibrosis, bronchial anthracosis and tuberculous lymphadenitis were found coincidentally,
3. Both tuberculosis and anthracosis was found in the same lymph node, and
4. Typical radiological image of active tuberculous lymphadenitis was not detected.

REFERENCES