suggest that menopause may play a role in the decrease of tumor size, other growth factors may contribute.

Simeon Abramson, MD
University Hospitals of Cleveland
Cleveland, OH

Correspondence to: Simeon Abramson, MD, Department of Radiology, University Hospitals of Cleveland, 11100 Euclid Ave, Cleveland, OH 44106

REFERENCES


Spirometry in the Diagnosis of Small Airways Obstruction

To the Editor:

In the article titled “Small Airways Obstruction Syndrome” (July 1999), Dr. Stănescu reported on four patients with the functional pattern of decreased vital capacity (VC) and FEV1, normal FEV1/VC ratio and total lung capacity, and increased residual volume (RV). In these patients, Dr. Stănescu alleges, the obstructive pattern would be overlooked on routine spirometry unless RV also was measured.

While a low VC or a low FEV1 may result from either a restrictive or an obstructive defect, the FEV1/VC ratio should be the primary guide to distinguish between the two patterns. Three of the four subjects in Dr. Stănescu's study have FEV1/VC ratios <70%, which would, therefore, place them in the appropriate category of obstructive defect, when FEV1/VC ratio is within normal limits, as shown in Table 1. We also computed in our subjects expected values for the FEV1/VC ratio using two well-known American formulas. According to subjects' ages and heights, the FEV1/VC ratios of our subjects were also within expected limits. Therefore, the FEV1/VC ratio of the patients we presented did not place them in the category of obstructive defect!

The authors also write that they have “discovered this pattern in several subjects who had correctly been diagnosed as obstructive, based on the spirometric assessment alone, allowing a disproportionate decrease in FEF25–75% [forced expiratory flow after 25 to 75% of VC has been expelled] and normal FEV1/VC ratio (confirmed, of course, by the presence of a high RV and RV/TLC ratio).

During a review of our laboratory data, we discovered this pattern in several subjects who had correctly had obstruction diagnosed based on the spirometric assessment alone, which showed a disproportionate decrease in FEF25–75% and a normal FEV1/VC ratio (confirmed, of course, by the presence of a high RV and RV/TLC ratio).

Ravichandran Theerthakarai, MD
St. Joseph's Hospital & Medical Center
Paterson, NJ

M. Anees Khan, MD, FCCP
School of Graduate Medical Education, Seton Hall University
South Orange, NJ

Correspondence to: Ravichandran Theerthakarai, MD, Pulmonary Division, St. Joseph’s Hospital & Medical Center, Paterson, NJ

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To the Editor:

I thank Drs. Theerthakarai and Khan for their interest in my article (July 1999). Alluding to the American Thoracic Society (ATS) statement, the authors write that the FEV1/vital capacity (VC) ratio should be the primary guide to distinguish between a restrictive and an obstructive defect. Of course, the authors are correct. But the problem is how to define the defect, when FEV1/VC ratio is within normal limits, and both FEV1 and VC are decreased, ie, the syndrome we have described.

Drs. Theerthakarai and Khan assert that three of our four patients have FEV1/VC ratios <70%, which would therefore place them in the appropriate category of obstructive defect without the need for residual volume (RV) measurement. I was surprised by this affirmation. According to the ATS statement they quote, “defining a fixed FEV1/VC [vital capacity] ratio as a lower limit of normal [my emphasis] is not recommended in adults because FEV1/VC is indirectly related to age and height.” All our four subjects had percentage of predicted FEV1/VC ratios within normal limits, as shown in Table 1. We also computed in our subjects expected values for the FEV1/VC ratio using two well-known American formulas. According to subjects’ ages and heights, the FEV1/VC ratios of our subjects were also within expected limits. Therefore, the FEV1/VC ratio of the patients we presented did not place them in the category of obstructive defect!

The authors also write that they have “discovered this pattern in several subjects who had correctly diagnosed as obstructive, based on the spirometric assessment alone, showing a disproportionate decrease in FEF25–75% [forced expiratory flow after 25 to 75% of VC has been expelled] and normal FEV1/VC ratio (confirmed, of course, by a high RV and RV/TLC [total lung capacity] ratio)”. One might ask why the authors needed another measurement (FEF25–75%) when the defect was correctly diagnosed by a high RV and RV/TLC and normal FEV1/VC?

The ATS guidelines suggest that, indeed, in the presence of a borderline FEV1/VC, maximal expiratory flow rates may help confirm the presence of airway obstruction. But FEV1/VC ratios in our subjects were not borderline. They were within normal limits. Furthermore, the guidelines emphasize that “when FEV1/VC ratio is within the expected range, abnormalities in flow occurring late in the maximal expiratory flow-volume curve, should not be graded as to severity, and, if mentioned, interpretations of their clinical significance should be guarded.”

Dan Stănescu, MD, PhD
Université Catholique de Louvain
Cliniques Universitaires Saint-Luc,
Brussels, Belgium

Correspondence to: Dan C. Stănescu, MD, PhD, Cliniques Universitaires Saint-Luc, Avenue Hippocrate 10, Bruxelles 1200, Belgium; e-mail: Stanesuc@pneu.ucf.ac.be

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Communications to the Editor
Inflammatory Pseudotumor of the Lung With Pleural Thickening Treated With Corticosteroids

To the Editor:

A 56-year-old man was admitted to Hiroshima University Hospital in 1987 because of radiographic evidence of pulmonary abnormalities, fever, and anorexia. A radiograph of the chest showed bilateral thickening of the pleura in the middle field. A CT scan of the chest disclosed remarkable thickening of the pleura and a tumorous lesion, 2 × 3.5 cm in diameter, with a low density area in the right lung (Fig 1). Because the etiology was unknown except in the lungs, an exploratory thoracotomy was performed. A microscopic examination of the resected specimen disclosed that the tumor was composed of a proliferation of fibroblasts and an infiltration of plasma cells, macrophages, and lymphocytes accompanied by a local accumulation of hyaline collagen layers (Fig 2). Because a low-grade fever and increased levels of C-reactive protein remained after the operation, 20 mg/d prednisolone was administered. After 2 weeks of corticosteroid treatment, the symptoms improved, and the dose of prednisolone was gradually tapered. No recurrence was observed after 10 years of follow-up.

Inflammatory pseudotumors are rare diseases that generally occur in the lung. To our knowledge, there is only one report describing an inflammatory pseudotumor presenting pleural thickening that showed spontaneous regression.1 Ishida et al reported that, intraoperatively, the parietal pleura was involved in three of seven patients with inflammatory pseudotumors. In our patient, although the histologic examination of the resected pleura revealed remarkable fibrosis without evidence of inflammatory pseudotumor involvement, adjacency between the tumor and the pleural thickening suggested a relationship of these lesions. After complete resection of the tumor, the symptoms remained, suggesting an active lesion in the remaining thickening pleura.

The diagnosis of inflammatory pseudotumor is not commonly made before resection, and complete resection leads to an excellent prognosis.3 Unresected or recurred cases in patients were reportedly treated with corticosteroids, which resulted in a decrease in size or complete regression.4,5 Before corticosteroid treatment, our patient was treated with nonsteroidal anti-inflammatory drugs, which were not effective. These observations suggest that corticosteroids may be an option in treating inflammatory pseudotumors.

Shinichi Ishioka, MD, FCCP
Akihiro Maeda, MD, FCCP
Masahiro Yamasaki, MD
Michio Yamakido, MD, FCCP
The Second Department of Internal Medicine
Hiroshima University School of Medicine
Hiroshima, Japan

Correspondence to: Shinichi Ishioka, MD, FCCP, The Second Department of Internal Medicine, Hiroshima University School of Medicine, 1–2–3 Kasumigaoka, Minami-ku, Hiroshima 734-8551, Japan

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