Agenesis of Pulmonary Artery

To the Editor:

We enjoyed reading the report by Dr. Kochiadakis and co-workers (June 2002).1 We would like to point out, however, that we have reported on a similar case of a 51-year-old woman with pulmonary hypertension and agenesis of the right pulmonary artery.2 Her pulmonary artery pressure was 68/24 mm Hg, as measured by right heart catheterization. The diagnosis of agenesis was confirmed by conventional pulmonary angiography as well as by magnetic resonance angiography, which also showed a common origin for both the common carotid arteries and an aberrant right subclavian artery, originating in the aortic arch distal and proceeding to the left subclavian artery. Left heart catheterization revealed communicating vessels between the left circumflex coronary artery and the right pulmonary circulation (Fig 1). Similar to the report by Kochiadakis et al.1 our patient did not have any evidence of myocardial ischemia due to these abnormal vessels, and surgical intervention was not undertaken.

Although this report is not cited in MEDLINE, it was submitted and published in the case report section of the Chest 2000 International Meeting.

George Mixides, MD
Imran Niami, MD, FCCP
Baylor College of Medicine
Houston, TX

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New Diagnostic Tool for Tumor Angiogenesis

To the Editor:

In a recent issue of CHEST, Ohta and colleagues (May 2002)1 showed excellent results in which the expression of vascular endothelial growth factor (VEGF) protein in BAL fluid was higher in patients with lung cancer than in those with noncancerous diseases, suggesting that VEGF concentrations in the airways may reflect responsive tumor angiogenic status on the host side. Following the accepted concept that the growth of solid tumors is dependent on their ability to elicit the development of new blood vessels into the tumor mass, there has been an increasing focus on targeting tumor vasculature. The angiogenic potential of the endothelial cell is carefully balanced between positive and negative regulation. Tumors have the potential to up-regulate or down-regulate these controls, producing an environment in which new blood formation occurs, thereby supporting tumor growth. Angiogenic factors include VEGF, fibroblast growth factor (FGF)-α and FGF-β, endothelial growth factor, tumor necrosis factor-α, and integrins. On the other hand, angiostatic factors include angiotatin, endostatin, transforming growth factor-β, tissue inhibitors of metalloproteinases, 2-methoxyestradiol, and squalamine.2,3 Indeed, angiogenic agents offer a potential for the treatment of cancers, but emerging data suggest that there may be limits to their use as monotherapy in patients with advanced malignancies. Therefore, the further accumulation of other markers of angiogenesis or other angiogenic molecules in malignant tumors, including lung cancers, are needed for future novel strategies.

 Peroxisome proliferator-activated receptors (PPARs) are members of the nuclear hormone receptor superfAMILY of ligand-activated transcription factors, which include vitamin D3, retinoid, and thyroid hormone receptors. Among PPARs, PPAR-γ has been well-recognized to correlate with inflammation and tumorigenesis. In fact, agonists of PPAR-γ suppress experimental arthritis and proliferation of cancer cell lines.6,7 We examined8–10 the expression of PPAR-γ in two different types of cancer tissues, lung cancer and renal cell carcinoma. Because PPAR-γ is expressed in endothelial cells,11 we compared the immunoreactive expression of PPAR-γ protein in endothelial cells as well as cancer cells between endogenous and normal tissues on a scale of 0 to 4, according to the grading methodology previously described.12 As a result, the means (± SD) immunohistologic scores of PPAR-γ in the endothelial cells of normal lung tissues and normal kidney tissues were 0.5 ± 0.3 and 0.6 ± 0.4, respectively. However, the immunohistologic scores of endothelial cells in lung cancer tissues and renal cell carcinoma tissues were 1.8 ± 0.9 and 3.1 ± 1.1, respectively (unpublished data), indicating that the extent and intensity of immunoreactive PPAR-γ in endothelial cells in these cancer tissues was significantly greater than that in the corresponding normal tissues.

In conclusion, we would like to recommend PPAR-γ as an angiogenic factor, and its ligands as angiostatic factors in malignant tumors.

Ken-ichiro Inoue, MD
Yutaka Kawahito, MD, PhD
Hajime Sano, MD, PhD
Hirohisa Takano, MD, PhD
Toshikazu Yoshikawa, MD, PhD
Kyoto Prefectural University of Medicine
Kyoto, Japan
To the Editor:

For treating latent tuberculosis infection (LTBI), we agree with Dr. Medinger’s advice (May 2002)1 to select treatment candidates conservatively and to monitor them closely for liver injury and other potential adverse effects. In August 2001, the American Thoracic Society and the Centers for Disease Control and Prevention (CDC) published revised guidelines2 that were based on investigations of cases, including the case reported by Dr. Medinger, of severe liver injury subsequent to the administration of rifampin and pyrazinamide for the treatment of LTBI. Included in the revisions are restrictive treatment-candidate selection criteria and an admonition for more frequent observations with which to detect adverse effects.

In order to estimate the incidence rate of liver injury in patients receiving rifampin and pyrazinamide therapy for the treatment of LTBI and to assess potential risk factors (eg, patient age, as suggested by Dr. Medinger), the CDC currently is investigating cohorts of patients who received this regimen and associated cases of liver injury. The CDC encourages providers to report severe liver injury (ie, that leading to hospital admission or death) associated with rifampin and pyrazinamide therapy for the treatment of LTBI by notifying their local or state tuberculosis control program or by calling the Division of Tuberculosis Elimination, CDC, at 404-639-8442.

Kenneth G. Castro, MD  
John A. Jereb, MD  
Centers for Disease Control and Prevention  
Atlanta, GA

Venkataramu Rao Kopple, MD, PhD, FCCP  
Virginia Department of Public Health  
Richmond, VA

David L. Cohn, MD  
Denver Public Health  
Denver, CO

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Erratum

In the February 2002 article, “A Cross-Cultural Comparison of Critical Care Delivery: Japan and the United States” (CHEST 2002; 121:539–548) by Sirio et al, the legends for Figures 1 and 2 are reversed.