The development of Adult Respiratory Distress Syndrome (ARDS) in miliary tuberculosis is extremly rare [1, 12], but its appearance after initiation of appropriate antibiotics is intriguing [12, 13]. *Onwubalili* et al. [13] reported two such cases. In addition, in their review of the literature they found several cases of patients who died unexpectedly and without obvious cause during treatment of tuberculosis. None of these patients had received corticosteroids, and clinical worsening developed early after initiation of antituberculous therapy.

To our knowledge, our case is the first one to report secondary development of extensive pleural effusion after initiation of treatment for disseminated tuberculosis.

Though the explanation for worsening during treatment remains unclear, in most cases this phenomenon occurs when tuberculosis is disseminated [5, 8, 13]. Dissemination leads to massive release of mycobacterial products inducing a production of high levels of inflammatory mediators after monocyte activation. Lipoarabinomannan from *M. tuberculosis* has been shown to induce the production of tumor necrosis factor from human macrophages [14], and it was speculated that lipoarabinomannan or other mycobacterial products may act *in vivo* like the lipopolysaccharide from gram-negative bacilli [15]. Introduction of lytic antibacterial therapy may sometimes initiate this process [12, 13]. Inflammatory mediators as well as mycobacterial components probably have a central role in pathogenesis of ARDS, pancytopenia [16] and in the paradoxical worsening of tuberculosis during therapy including secondary pleural effusion. Clinicians should be aware that initial worsening or development of new tuberculous lesions after introduction of specific treatment usually does not reflect treatment failure and that associated corticosteroid therapy may improve the outcome.

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Book Review

H. Koprowski, H. Meada (eds.)
The Role of Nitric Oxide in Physiology and Pathophysiology
90 pages, 21 figures
Springer, Berlin–Heidelberg–New York–London–Paris–Tokyo–Hong Kong, 1995
Price: DM 139,– DM

This book contains eight contributions presented at a workshop in Philadelphia in 1993. Each contribution of this thin 90-page book is written by specialists in this area and gives a comprehensive overview of the special physiological and pathophysiological aspects of nitric oxide, e.g. "S-Nitrosothiols and the Bioregulatory Actions of Nitrogen Oxides through Reactions with Thiol Groups;" "A New Nitric Oxide Savenger, Imidazolineoxyl N-Oxide Derivative, and Its Effects in Pathophysiology and Microbiology;" "The Role of Nitric Oxide in the Pathogenesis of Virus-Induced Encephalopathies." The reference list at the end of the concise contributions provides a quick orientation in field and many of the separate papers have excellent figures. On the whole, the book could be of interest to insiders in research on nitric oxide, since renown scientists present exceptional but detailed information on this subject. However, it seems less suitable for the reader who, from the title, expects a quick survey of the physiological and pathophysiological significance of NO.

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