


On the Role of Chest CT Scanning in a TB Outbreak Investigation

To the Editor:

A recent article in CHEST (May 2010) by Lee et al1 reported the use of high-resolution chest CT (HRCT) scanning during a TB outbreak investigation. They found that nine of 18 (50%) cases diagnosed with active TB would have been missed without the use of HRCT scanning and concluded that inclusion of HRCT scanning seems helpful to reliably identify cases with active TB during an outbreak investigation. Routine use of HRCT scanning will add cost, increase radiation exposure, and undermine confidence in existing screening tools where HRCT scanning is unavailable, but the most important reason for caution is the likely absence of clinical relevance.

Patients with asymptomatic “HRCT confirmed active TB” present a case-definition dilemma, because the natural history of Mycobacterium tuberculosis infection demonstrates that transient phenomena, such as parenchymal consolidation and/or hilar adenopathy (even M tuberculosis excretion), occur quite frequently after recent primary infection.2,3 Only a small percentage of these asymptomatic “patients” progress to active disease on long-term follow-up.3 Therefore, it is not unexpected that HRCT scanning identifies a subset of recently exposed individuals with visible parenchymal and/or lymph node involvement despite being asymptomatic and having a normal chest radiograph. However, the clinical relevance of these findings remains questionable—whether it represents transient phenomena or is truly indicative of active disease, and whether treatment with combination therapy is warranted.

None of the patients with a normal chest radiograph and lesions suggestive of active TB on HRCT scan were sputum smear or culture positive for M tuberculosis, indicating uncertain diagnosis and/or low organism load. The United States Public Health Service Tuberculosis Prophylaxis preventive therapy trial conducted in the 1950s demonstrated that isoniazid monotherapy prevented progression to symptomatic disease in child TB contacts, despite the presence of radiologic signs suggestive of recent primary infection and/or minimal disease.4 This provides the rationale for symptom-based screening approaches in children.5 In certain high-risk groups the use of sensitive screening tools may well be warranted, but because subclinical transient phenomena may be detected and treated with increased regularity the routine use of HRCT scanning to screen asymptomatic TB contacts for active disease requires rigorous scrutiny. Given the current evidence, cost, and potential risks involved, there is no role for HRCT scanning as a routine screening test during TB outbreak investigations.

Ben J. Marais, MD, PhD
Tygerberg, South Africa

Elevation of IL-6 Solely Is Not Sufficient to Infer Systemic Inflammation

To the Editor:

In an excellent study in CHEST (July 2010), Sabit et al1 showed that a 2-h hypoxic challenge in patients with mild COPD who are clinically stable results in an elevation of serum IL-6 and some coagulation markers, such as prothrombin activation fragments 1 + 2 and thrombin-antithrombin complex. The authors concluded that a strong association exists between hypoxia, coagulation activation, and systemic inflammation and that patients with COPD may be at increased risk of VTE during air travel.

However, elevation of IL-6 solely cannot be used to infer the presence of systemic inflammation due to the pleiotropic action of IL-6 involving a variety of systems and diseases. IL-6 has been mostly studied in the context of the acute inflammatory response, although growing evidence shows IL-6 also plays a vital role in the pathogenesis of aging and chronic disease.5 In addition, IL-6 is also a “myokine,” a cytokine produced from muscle, and is elevated in response to exercise.5 Previous studies have demonstrated IL-6 levels can increase up to 100-fold during exercise, in a duration- and intensity-dependent manner.6,7

In the study by Sabit et al,1 subjects in the group with hypoxia experienced a hypoxic challenge with a mean oxygen saturation decline from 94% ± 2% to 90% ± 3%. When the oxygen concentration in the arterial blood fell, the chemoreceptors were stimulated and the ventilation increased, as shown by a rising respiratory rate (RR) or tidal volume.8 The muscle must be loaded with additional respiratory work, possibly contributing to a surge of IL-6. Although ventilation was not measured, the findings of higher RR and heart rate (HR) in the subjects receiving the hypoxic challenge were also in part compatible with our concern. (In the group with hypoxia, the RR increased from 14 ± 1 per min to 16 ± 3 per min, and the HR increased from 86 ± 6 per min to 94 ± 4 per min; in the control group, the RR increased from 12 ± 2 per min to 13 ± 2 per min, and the HR increased from 80 ± 6 per min to 82 ± 5 per min.)

REFERENCES


Affiliations: Department of Paediatrics and Child Health, Faculty of Health Sciences, Stellenbosch University.

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Correspondence to: Ben J. Marais, MD, PhD, Department of Paediatrics and Child Health, Faculty of Health Sciences, Stellenbosch University. E-mail: bjmarias@sun.ac.za

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