Correspondence

Tuberculosis and Vitamin D Deficiency

Sirs,

PK Sasidharan et al (Tuberculosis and vitamin D deficiency JAPI 2002;50:554-58) have observed low serum 250 HD in absence of suggestive obvious clinical signs of vitamin D deficiency in patients with tuberculosis. On the basis of their findings and on the role of vitamin D in tuberculous infection as evidenced by in-vitro studies and clinico-biochemical observations made in western environment, they have suggested vitamin D supplementation in patients with tuberculosis. However, assessment of nutritional deficiency in Indian context is extremely difficult in view of wide variations in dietary intake in population groups depending on regional rural-urban and socio-economic stratifications besides age and gender differences. In general, a large segment of the vulnerable population suffer from undernutrition involving multiple nutrient deficiencies. Current emphasis on a few micro-nutrient deficiencies, apparently easier to prevent, tends to overlook this overall dietary deprivation leading to undernutrition, semistarvation and even starvation despite surplus food production and Poverty Alleviation Programmes undertaken so far. In any case, single nutrient deficiency is exceptional and tuberculous patients of Sasidharan et al could possibly have biochemical evidences of other nutrient deficiencies also, some of them having important bearing in immune mechanism related to tuberculous infection.

Vitamin D deficiency clinically manifested as rickets and osteomalacia has long been prevalent in India and its pathogenesis is complex.1 Regrettably, there is lack of awareness of this important problem even amongst the pediatricians and physicians. The other way to look at the role of vitamin D deficiency in tuberculosis is to find out whether tuberculosis occurs more frequently or it is more severe in children with rickets than others in similar environment. Observations in under-seven children admitted to our hospital, mostly from a few Calcutta slums, on this aspect may be of interest in this context. In a series of 192 children with clinical signs of rickets studies during 1968-80, wrist X-ray showed rachitic bone changes which were mild in 51, moderate in 65, severe in 50 and arrested in 26. Pulmonary tuberculosis was detected on routine chest x-ray in 8.3% (hilar adenopathy in 6.2%, primary complex in 2.1%).2 for comparison routine chest x-rays in children with protein-energy malnutrition (PEM), chronic diarrhea and respiratory tract infections were available.3 In a series of 500 children (1957-68) with severe PEM (kwashiorkor, marasmic kwashiorkor and marasmus) 18% showed suggestive pulmonary x-ray signs, the lesions being extensive with considerable fibrosis in 7.8% and conforming to early childhood tuberculosis in 10.2%. In a subsequent series of 200 children with similar disease (1968-74) the incidence came down to 8% and the lesions were early. Similar mild disease was detected in 4% of 123 children with mild-moderate malnutrition (PEM), 7.7% of 39 children with respiratory infections and 6.4% of 47 children with chronic diarrhea. None of these children showed clinical sign of rickets. Serum 250 HD was not estimated. Hence the incidence of pulmonary tuberculosis in rachitic children was more or less similar to what was observed in children suffering from other disease excepting that this was higher in the earlier series of severe PEM in whom the disease was more severe also.

Our clinical observations do not support the synergistic role of vitamin D deficiency in tuberculosis but these do not nullify the elegant studies cited by Sasidharan et al. In the Indian context however, it may be pertinent to take into account other nutritional deficiencies and not that of vitamin D alone in the treatment of tuberculosis, the mainstay in which is at present chemotherapy and as mentioned by Sasidharan et al, nutritional support does not receive enough consideration.

AK Bhattacharyya
Former Professor of Nutritional and Metabolic Diseases, School of Tropical Medicine, Kolkata.
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