Correspondence

Growth Hormone Therapy in Turner’s Syndrome: What is the Gain?

Sir,

Recombinant human growth hormone (rhGH) was approved by United State Food and Drug Administration in 1985 for growth hormone (GH) deficiency.1 Subsequently rhGH was approved for conditions without GH deficiency – Turner’s syndrome (TS), Prader-Willi syndrome, chronic renal insufficiency and small for gestational age children.2 Initial studies show that girls with Turner’s syndrome gained 7.2 cms over a period of 5.7 years of GH treatment.2 However a recently concluded study by Ross et al showed the average height gain after 7.2 years of treatment was only 5 cms.3 The point to be noted is that in a developing country like India whether state sponsored rhGH treatment will be cost effective. Currently state run institutions like Armed Forces, Central Government Health Scheme and Railways to name a few are providing rhGH treatment free of cost to TS girls. The dose of GH in TS is 0.375 mg/kg body weight/week. On an average a 30 kg girl would require 45 mg of rhGH per month which will cost about Rs 60,000 per month and a whooping Rs 60,000 per month and a whooping ₹50 lakhs for seven years! It would be a provocative thought if the state could take care of education, job prospects and health insurance of these girls rather than spending such a huge amount of money for a gain of 5 cm of height.

References

The Levels of Oxidants and Antioxidants Status in Chronic Obstructive Pulmonary Diseases (COPD) with Relation to Oral Vitamin E Supplementation

Sir,

The present work was planned to study thiol proteins as well as oxidant/antioxidant balance during inflammation in COPD. Study was also aimed at investigating the effect of vitamin E supplementation on oxidative stress parameters. Chronic obstructive pulmonary disease (COPD) a chronic slowly progressive disorder is characterized by airflow obstruction.

Oxidative stress has been recognized as a central feature of smoke induced chronic obstructive pulmonary disease (COPD) imbalance between oxidant and antioxidant enzymes is also an established fact in these patients. The oxidative stress is believed to play a vital role in the pathogenesis of COPD being responsible for a series of events including recruitment of neutrophils and macrophages increased mucus secretion and vascular permeability airway inflammation, bronchospasm and inhibition of protease inhibitors.1 The oxidative stress and protease antiprotease imbalance promote alveolar damage and chronic airway inflammatory which are pathophysiologic hallmarks of COPD.2 Smoking known to be the primary risk factor for COPD can also cause severe oxidant damage.3

While oxidative stress has been well documented in COPD. There have been only a few studies on the therapeutic role of antioxidants and none using vitamin E. In this study we compared the oxidation product (MDA) and antioxidant superoxide dismutase (SOD) level and Total plasma sulphydryl level in COPD and healthy non-smokers. Further we tested the effect of vitamin E Supplementation at 400 IU per day for 12 weeks on MDA (malondialdehyde), SOD (superoxide dismutase) and total plasma sulphydryl levels.

20 patients with stable COPD were included in the study. Their base line clinical examination. Malondialdehyde (MDA) alpha tocopherol and erythrocyte superoxide dismutase (SOD) level were measured. 30 healthy non-smokers who were matched for age and sex served as controls. All the above parameters were repeated after 12 weeks of supplementation with 400 IU of vitamin E daily.

The mean malondialdehyde levels in the patients at base line were higher than Control, Plasma alpha-tocopherol levels, SOD levels and protein sulphydryl were lower in the patients compared to controls. Exogenous vitamin E (400 IU per day) Supplementation did not bring about any significant change in plasma Erythrocyte Superoxide Dismutase and vitamin E were not significant either on day 1 or after 12 weeks of vitamin E Supplementation. But slight increase in the plasma protein sulphydryl level was seen. The present study shows that initially the plasma lipid peroxide (MDA) levels were high and antioxidant (alpha- tocopherol, SOD and protein sulphydryl) were low in patients with COPD. Exogenous supplementation with vitamin E does not have significant effect on the alpha tocopherol and SOD levels though it brings down the levels of MDA showing attenuation of further damage.

Conclusion

Our results suggest the presence of oxidative stress and the augmentation of antioxidant defenses as shown by slight increase in plasma sulphhydryl level. However, inclusion of larger number of patients and supplementation with vitamin E for longer periods with higher doses may throw more light on free radical injury and protective effects of antioxidants.

References
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