Publications

Dept of Endocrinology Medwin Hospitals

2006 - 2009

Correspondence

Adolescent Overweight and Coronary Heart Disease

TO THE EDITOR: The Centers for Disease Control and Prevention (CDC) recommends using the body-mass index (BMI) for age as a marker in assessing patients between the ages of 2 and 20 years for obesity. In the article by Bibbins-Domingo et al. (Dec. 6 issue), weight alone was used as a marker for obesity in adolescents, which may not reflect the true underlying value. Was this approach justified? Furthermore, the assumption that a high BMI does not directly increase the risk of coronary heart disease (CHD) may be unwarranted, since there is enough evidence to the contrary.

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Medwin Hospitals Hyderabad 500001, India hariendo@rediffmail.com gains were offset by increases in diabetes and obesity. Between 1980 and 2000, BMI (defined as the weight in kilograms divided by the square of the height in meters) increased from 25.5 to 28.2, accounting for approximately 25,900 additional deaths.²

Our model used the large random-effects metaanalysis (with a total of 302,296 subjects) by Bogers et al.³ A five-unit increase in BMI generated a 29% increase in deaths from coronary heart disease, or, crucially, a 16% increase after adjustment for cholesterol level and blood pressure.³ Might Bibbins-Domingo et al. have underestimated the mortality effects?

Trends toward increased mortality from coronary heart disease are already detectable in the U.S. population.⁴ From 1997 through 2002, the mortality rate leveled off among men who were 35 to 44 years of age and actually increased by

Case 16-2008: A Woman with Bone Pain

TO THE EDITOR: The diagnosis of secondary hyperparathyroidism in the case discussed by Demay and colleagues (May 22 issue)1 is doubtful. Primary hyperparathyroidism with coexisting vitamin D deficiency presents with normocalcemia and hypophosphatemia.2 Hence, a normal calcium level with a low phosphorus level is not diagnostic of secondary hyperparathyroidism. The features of bone pain and fatigue, brown tumor, lytic bone lesions, and a single parathyroid adenoma are all suggestive of primary hyperparathyroidism. The markedly elevated level of parathyroid hormone suggests a secondary cause but does not rule out primary hyperparathyroidism3; a markedly decreased level of the calcium-sensing receptor is seen in secondary hyperparathyroidism but not in sporadic adenomas.4 We believe that detection of calcium-receptor protein or technetium-99m-labeled sestamibi imaging would help in establishing the underlying diagnosis.5

There is a higher prevalence of primary hyperparathyroidism than secondary hyperparathyroidism among patients with celiac disease. This condition usually presents with normocalcemic hyperparathyroidism, as in the described case.⁶

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Effect of parental education on child stunting

We have some comments about Richard Semba and colleagues' study on parental education and the risk of child stunting in Indonesia and Bangladesh (Jan 26, p 322).1

First, the observed difference in the prevalence of stunting between Indonesia and Bangladesh, with growth charts from WHO (33-2% and 50-7%, respectively) and the US National Center for Health Statistics (30-7% and 57-5%, respectively) is interesting and needs more explanation. WHO growth charts are recommended to be useful in infancy and region-specific growth charts for later ages.²

Second, birthweight is a major determinant of adult height.3 To draw conclusions in the Bangladeshi population without these data is unjust. Catch-up growth is an important natural defence for short stature and these short individuals reach normal adult height after removal of the offending agent.4

Finally, the graphs comparing parental education with Z score (height for age) showed a negative trend at around 10–11 years of paternal education in both countries. What could be the reason for such an interesting similar observation?

We ded are that we have no conflict of interest.

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Bone Mineral Density and High-Trauma Fractures

To the Editor: We have comments regarding the study of high-trauma fractures and low bone mineral density in older women and men by Ms Mackey and colleagues. First, it appears that the majority of high-trauma fractures described were due to rather minimal trauma, given that 37.7% of patients in the Study of Osteoporotic Fractures (SOF) cohort and 51.1% in the Osteoporotic Fractures in Men Study (MrOS) cohort sustained fracture due to a fall from more than standing height. This could have contributed to the positive association of osteoporosis with high trauma.

It would be helpful to know whether any persons sustained a low-trauma fracture after a high-trauma fracture or vice versa. This would give more information about underlying osteoporosis, rather than the intensity of trauma, as a cause of increased risk of fracture. Also, fracture at any site would increase the predisposition to further fracture by other mechanisms such as immobility or bone loss.² In both the cohorts, the persons who sustained fractures (both high- and low-trauma) reported increased incidence of falls in the past year and fracture since age 50 years, suggesting underlying osteoporosis rather than the trauma as the actual risk factor.

Second, in the SOF cohort, 42% of women with bone mineral density scores indicating osteoporosis and about 58% with bone mineral density in the osteopenia range did not sustain fracture over a mean follow-up period of 9 years. This appears contrary to other studies.^{3,4}

Third, in the SOF cohort, bone mineral density in the osteopenia range was seen in a similar percentage of women among all 3 subgroups (52.8% with high-trauma fracture, 53.7% with low-trauma fracture, and 57.5% with no fracture). This highlights the importance of looking at factors other than bone mineral density in assessment of future fracture risk.⁵

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Effect of Testosterone Therapy versus Other Factors on the Self-reported Sexual Satisfaction of Premenopausal Women TO THE EDITOR: We have some comments regarding the study by

Davis and colleagues (1) on testosterone replacement in premenopausal women. The inclusion criteria specified for the study participants were

an early-morning free testosterone level of 3.8 pmol/L or less (≤1.1 pg/mL). However, the mean free testosterone level at baseline as shown in Table 3 is more than 6 pmol/L (>2 pg/mL) in all the study groups. This seems contrary to the defined inclusion criteria.

Of the 480 symptomatic women who were initially screened, 168 (35%) were ineligible because of free testosterone levels greater than the cutoff value ($\leq 3.8 \text{ pmol/L} [\leq 1.1 \text{ pg/mL}]$). This highlights the fact that androgen levels are not related to sexual dysfunction in women, as reported earlier (2).

Use of oral contraceptive pills result in suppression of free and total testosterone levels and may contribute to decreased libido (3). In the study, the maximum benefit is seen in the group using daily 90-μL doses of transdermal testosterone. Baseline characteristics show that more patients in this group were using oral contraceptives than were those in other groups (34% vs. 2% in the placebo group). Could this be the contributing factor behind the observed benefit of

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daily 90- μ L doses of transdermal testosterone in this group?

2 September 2008 Annals of Internal Medicine Volume 149 • Number 5 361

Vitamin D Therapy and Reduced Mortality in Chronic Kidney Disease

he study by Kovesdy et al1 showed that treatment with activated vitamin D (calcitriol) therapy significantly increased survival in patients with CKD not receiving dialysis. However, baseline data between treated and untreated groups was not comparable regarding the bone and mineral abnormalities. The baseline parathyroid hormone (PTH) level was high in the calcitriol group (152 pg/mL vs 75 pg/mL), and more Correspondence: Dr Kumar, Department of Endocripatients in the calcitriol group were using calcium and nology, Medwin Hospitals, Nampally, Hyderabad, Andhra phosphate binder, indicating that more patients in the Pradesh, India (hariendo@rediffmail.com) calcitriol group had secondary hyperparathyroidism. Thus, a simple explanation of observed benefit with calcitriol therapy is by correcting secondary hyperparathyroidism.

Secondary hyperparathyroidism contributes significantly to progression of the kidney disease and increase in mortality.² Previous studies suggest a decreased progression of CKD with better management of associated 4. Wolf M, Thadhani R. Vitamin D in patients with renal failure: a summary of mineral and bone disorders.3 To suggest a benefit of ac-

tivated vitamin D independent of PTH lowering, the subgroup data regarding calcitriol use in patients with normal PTH levels should be provided. Also, the mean follow-up after starting the calcitriol therapy should be studied to justify its use in reducing mortality. This may highlight the beneficial effects of vitamin D therapy beyond bone health in patients with CKD.4

> Hari Kumar K. V. S., MD Kirtikumar D. Modi, MD, DM Ratan Jha, MD, DM

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Obesity and Hypothyroidism: Symbiotic Coexistence

he study by Fox et al¹ showed that a change in serum thyrotropin (TSH) concentration within the reference range is associated with changes in body weight. However, the authors' focused only on the weight gain seen with the rise in TSH value, but no data were shown regarding the weight loss associated with the fall in TSH value. These data would have helped in further defining the relation between body weight changes in the normal reference range of TSH value.

The authors considered the normal TSH reference range as between 0.5 mIU/L and 5.0 mIU/L in their study population derived from a community sample. However, there are no data regarding the thyroid antibody status of the participants in their study. The debate about the reference range of TSH in a population is increasing, and recent studies have proposed a narrow range of normal TSH levels in a population.2 Furthermore, a progressive rise in TSH concentration was observed in individuals with positive thyroid antibody levels indicating progressive thyroid dysfunction.3 This subclinical hypothyroidism may contribute to the associated weight gain in these patients.4 Hence, the defined normal range in the study population may not be truly representative of the population studied without simultaneous determination of thyroid antibody levels.

An increase in body weight may increase TSH concentration through alterations of hypothalamic-pituitarythyroid axis involving leptin.⁵ The observed rise in TSH concentration in participants with weight gain may be secondary to thyroid axis abnormalities.

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change in TSH concentration throughout the spectrum (encompassing increases and decrease in values) in relation to weight change (including weight gain and loss). As described in the article, the mean change in TSH concentration was 0.06 mIU/L in women and 0.13 mIU/L in men. The lowest quartile of TSH concentration change includes individuals whose TSH value declined over time (range of TSH values in the lowest quartile, -2.45 to -0.34 mIU/L in women and -3.02 to -0.29 mIU/L in men). Women in the lowest quartile of TSH concentration change gained a mean of 0.5 kg, whereas, overall, mean weight change was 1.5 kg in women. Similarly, men in the lowest quartile of TSH change gained a mean of 0.4 kg, whereas, overall, mean weight change was 1.0 kg in men. Therefore, participants whose TSH value declined over time gained less weight compared with the rest of the sample.

Kumar et al raise an important point about thyroid peroxidase antibodies. Unfortunately, these were not measured in our study. However, population estimates suggest that the prevalence of antithyroid peroxidase antibodies is only 12.3% among those free of clinical thyroid disease² and even lower among those with serum TSH concentration in the normal range,³ suggesting that these antibodies are unlikely to account for our observed findings.

Finally, Kumar et al note that the association between body weight change and TSH concentration may be driven by other disturbances in the hypothalamic-pituitarythyroid axis. We agree with this point. For this reason, as indicated in the "Comment" section of our article, we note that our results demonstrate an association but do not establish causality.

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INCREASE IN BONE MASS AFTER VITAMIN D REPLACEMENT: CAN WE IGNORE REGRESSION TO THE MEAN?

To the Editor:

With considerable interest, we read the recently published *Endocrine Practice* article entitled "Increase in Bone Mass After Correction of Vitamin D Insufficiency in Bisphosphonate-Treated Patients" by Geller et al (1). We have the following comments to offer about that study.

- The authors ruled out the phenomenon of regression to the mean as a cause of improvement in bone mineral density (BMD) during the second year of treatment and attributed the improvement to vitamin D replacement alone in 17 responders.
- 2. We believe that regression to the mean cannot be excluded as the reason for improvement in BMD scores, at least in some of these responders, despite concomitant improvement in vitamin D levels. It is well documented that patients who lose maximal BMD during the first year of treatment attributable to the phenomenon of regression to the mean are seen to gain the most during the subsequent year of continued treatment (2).

Definite distinction between the role of vitamin D sufficiency or regression to the mean would be provided by the change in BMD during the third year. If the improvement in BMD seen during the second year continues to advance further during the third year, with maintenance of vitamin D sufficiency, then the positive effect on BMD is more likely to be due to vitamin D replenishment.

The gain in BMD achieved during the second year attributable to the phenomenon of regression to the mean alone is likely to become blunted or even lost during the subsequent year of follow-up. BMD change during the third year of treatment is likely to be a more robust indicator of the benefit of vitamin D administered (2).

- Compliance with vitamin D therapy is likely to be better in patients who had good compliance with bisphosphonate treatment. Greater gain in BMD with improvement in vitamin D level may be the result of better compliance with use of both drugs rather than the effect of vitamin D replacement only.
- Bone mineral density determined by dual-energy xray absorptiometry was repeated within 12 months in most of the cases. This may be too early to note a discernible, clinically significant change in BMD.

Disclosure

The authors have no conflicts of interest to disclose.

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Endocr Pract 2008 Sep; 14(6): 797-8.

PREMICROALBUMINURIA IN POLYCYSTIC OVARY SYNDROME

To the Editor:

We read the article entitled "Premicroalbuminuria in Women With Polycystic Ovary Syndrome: A Metabolic Risk Marker" by Patel et al (1) in the March 2008 issue of Endocrine Practice with considerable interest and have the following comments to offer:

Microalbuminuria is known to be transient and spontaneously reversible in more than half of the affected patients. Hence, the detection of microalbuminuria, on the basis of standard established guidelines, needs confirmation by repeated evaluation at 3 to 6 months (2), which trol groups.

The glucose-insulin ratio or homeostasis model assessment of insulin resistance would have been a better factor than the absolute values of fasting insulin for assessing insulin resistance.

Disclosure

The authors have no conflicts of interest to disclose.

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In Response:

We thank Dr. Muthukrishnan and colleagues for their lot is me insum resistance associated wrant (105 (2).....

All control subjects in our study had regular menses, and none had acne, hirsutism, or any stigmas of PCOS; thus, the comment that a subset of women in the control group might have had PCOS appears not to be likely (3). Rather, we consider it likely that the significantly older age of the control subjects in comparison with the patients who had PCOS contributed to their similarity in baseline variables.

Finally, in a nondiabetic population, there is extremely close correlation between the fasting insulin level and measures such as homeostasis model assessment of insulin

Endocr Pract 2008 Jul; 14(5): 648-9.

level itself (4).

Saudi J Kidney Dis Transpl 2008;19(2):254-255 © 2008 Saudi Center for Organ Transplantation

Saudi Journal of Kidney Diseases and Transplantation

Letter to the Editor

Controversial role of Omega-3 fatty acids in Dyslipidemia

To the Editor:

We have read the article "The effect of Low Dose Omega-3 on Plasma Lipids in Hemodialysis Patients" by Taziki O et al¹ and we have few queries for which clarification is required from the authors:

- 1. The context and design of the study appears inappropriate because chronic kidney disease (CKD) patients have high risk of cardiovascular mortality. As per KDOQI guidelines the primary target should be LDL lowering to less than 100 mg/dl.² All the participants in the study have LDL > 100 mg/dl and the authors have not addressed this in their study exposing the patients to higher risk of cardiovascular events.
- 2. Omega -3 fatty acids alone have debatable efficacy in lowering triglyceride levels and a recent meta analysis did not

 \pm 22, Table 2 – 268 \pm 32).

- 4. The fasting serum total cholesterol should be more than 220 mg/dl as per the inclusion criteria. However, in Table 2 the baseline levels provided for both groups $(102 \pm 32, 229 \pm 31)$ were not in keeping with the inclusion criteria.
- 5. The authors mentioned that in their study, regular fish intake or pharmacological treatment with N-3 PUFA has favorable effect on serum lipids in patients on HD. However, no data regarding the fish intake is provided in the article.

We would like to thank the authors for an interesting paper highlighting the importance of managing dyslipidemia in CKD patients.

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Departments of ¹Endocrinology

a recent meta analysis did not ficant reduction in cardiovasci Saudi J Kidney Dis Transpl 2008 Mar; 19(2):254-255, mortality with the use of dietary or pharma-

Case Reports

Case Report



Ectopic Parathyroid Adenoma - The Hidden Culprit

J Muthukrishnan*, A Verma*, KD Modi*, K Kumaresan***, Sangeeta Jha**

J Assoc Physicians India 2007 Jul; 55: 515-8.

Two cases describing the effects of hypothyroidism on puberty and growth

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Received 13 May 2008; received in revised form 16 May 2008; accepted 19 May 2008

Apathetic thyrotoxicosis – Can color Doppler sonography help?

Color Doppler sonography (CDS) is increasingly being recommended as a first-line investigative tool in differential diagnosis of thyrotoxicosis (1). We report the atypical findings of CDS in a case of apathetic thyrotoxicosis. A 75-yr-old man presented with a 6-month history of anorexia, weight loss of 6-7 kg, lethargy, and extreme was has of limbs of anorexia and recommendation of limbs of side and 18 cm/sec on right side. 99mTc pertechnate scan showed increased uptake of tracer confirming the diagnosis of apathetic thyrotoxicosis. The patient was treated with Carbimazole 30 mg per day in divided doses and after 4 weeks was relieved of his symptoms and gained 3 kg in weight.

Apathetic thyrotoxicosis is a distinct subset of hyperthyroidism often missed due to absence of typical symptoms and signs. The salient features are apathy, depression, weight loss, muscle weakness and wasting. Noteworthy is the absence of hyperkinetic motor activity, tive thyrotoxicosis from Graves' thyrotoxicosis (2). Graves' disease patients typically have markedly increased intraparenchymal vascularity, so called "thyroid inferno" and increased PSV of ITA (3). Based on various studies using Color flow Doppler sonography (CFDS) of thyroid, it is even proposed that CFDS should be the first interesting of thysotoxing grands hourd include all subsets of disease including neonatal/childhood Graves' disease and apathetic thyrotoxicosis. This may be more useful in defining the role of CDS as an alternative to nuclear imaging in the evaluation of thyrotoxicosis.

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Saudi J Kidney Dis Transpl 2009;20(2):278-281 © 2009 Saudi Center for Organ Transplantation

Saudi Journal of Kidney Diseases and Transplantation

Case Report

Conns' Syndrome – Atypical Presentations

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Saudi J Kidney Dis Transpl 2009 Mar; 20(2):278-281.

CASE REPORT

Letrozole as a Booster Therapy in Growth Hormone Deficiency

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Indian Pediatr 2009 Jul; 46(7):625-7.

Case Report

DISTAL RENAL TUBULAR ACIDOSIS DUE TO PRIMARY HYPERPARATHYROIDISM

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Endocr Pract 2008 Dec; 14(9):1133-36.



International Journal of Cardiology xx (2009) xxx-xxx

International Journal of Cardiology

www.elsevier.com/locate/ijcard

Letter to the Editor

Peripartum cardiomyopathy in type II autoimmune polyendocrine syndrome

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Int J Cardiol 2009 Mar 19. Epub

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Clinical Brief

Central Hypothyroidism

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Indian J Pediatr 2009 Nov 20. Epub ahead of Print.

Nerve, muscle or bone disease? Look before you leap

Muthukrishnan J, Harikumar K V S, Sangeeta J, Singh M K, Modi K

Singapore Med J 2009 Aug; 50(8): e293-4.

Type 1 Diabetes Mellitus and Polycystic Ovary Syndrome

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India

Inherited Renal Tubular defects with Hypokalemia

Pregnancy predisposes to Rhabdomyolysis due to hypokalemia

Saudi J Kid Dis Transpl (In Press)

Right Sided Syndrome – Congenital absence of Right Kidney & Testis

Saudi J Kid Dis Transpl (In Press)

Improved Glycemic Control in Pregnancy with CSII

Int J Diab Dev Ctries (In Press)

Long term Alendronate in Polyostotic Fibrous Dysplasia

Am J Orthopedics (In Press)

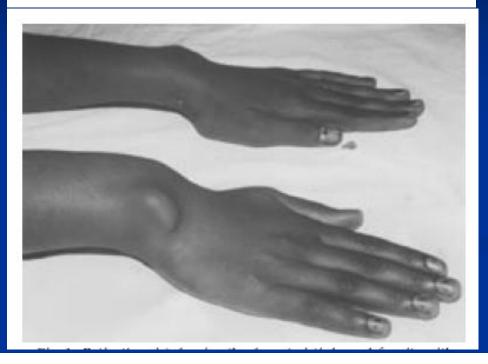
Precocious Puberty & Pineal Cyst

Indian Pediatr (In Press)

Clinical Images

Pictorial CME

Madelung's Deformity



J Muthukrishnan*, A Verma*, KD Modi**

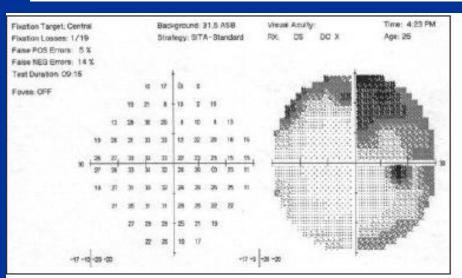
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J Assoc Physicians India 2006 Nov; 54: 867.

Visual Vignette

Submitted by

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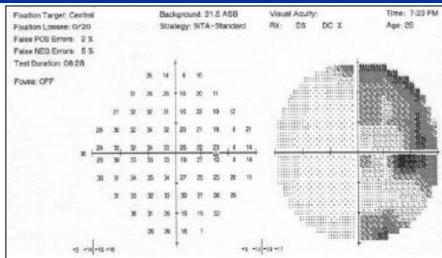


Fig. 1

Fig. 2

Visual Vignette

Submitted by

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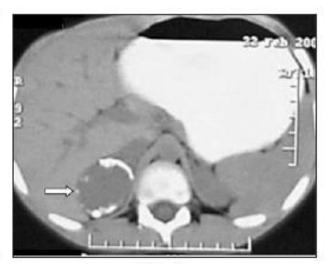


Fig. 2

Endocr Pract 2009 May; 15(3): 278.

IMAGES IN PEDIATRIC ENDOCRINOLOGY

Short Child with Interesting Hand Radiograph



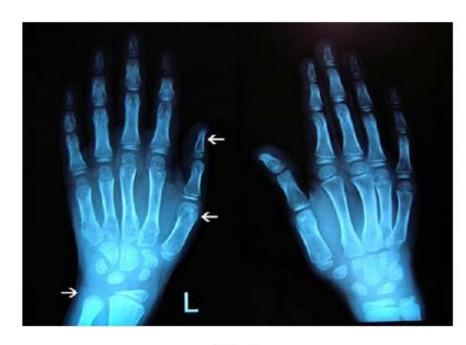


Fig. 1

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Visual Vignette

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Fig. 2



Fig. 3

Visual Vignette

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MEDMIN HOSPITALS HYDERAGAD

Fig. 1

Fig. 2

Endocr Pract 2008 Sep; 14(6): 799.

0021-972X/07/\$15.00/0 Printed in U.S.A.

Etiology of Early-Onset Type 2 Diabetes in Indians: Islet Autoimmunity and Mutations in Hepatocyte Nuclear Factor 1α and Mitochondrial Gene

Ravi P. Sahu, Ajay Aggarwal, Ghazala Zaidi, Ajay Shah, Kirti Modi, Srikanth Kongara, Suraksha Aggarwal, Sudha Talwar, Su Chu, Vijayalakshmi Bhatia, and Eesh Bhatia

J Clin Endocrinol Metab 2007 Jul; 92(7): 2462-7.



Efficacy of Teriparatide in Increasing Bone Mineral Density in Postmenopausal Women with Osteoporosis – An Indian Experience

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J Assoc Physicians India 2008 Jun; 56: 418-24.

International Journal of Gynecology and Obstetrics (2008) 102, 152-155







CLINICAL ARTICLE

Evaluation of thyrotoxicosis during pregnancy with color flow Doppler sonography

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Symptomatic Primary Hyperparathyroidism: A Retrospective Analysis of Fifty One Cases from a Single Centre

J Muthukrishnan*, Sangeeta Jha*, KD Modi*, R Jha**, J Kumar**, A Verma*, KVS Harikumar*, Kiran Patro**, B Srinivas**, K Kumaresan***, Rayudu Ramasubba+

J Assoc Physicians India 2008 Jul; 56: 503-7.

Hypothyroidism and obesity

Cause or Effect?

Abhyuday Verma, MD, Muthukrishnan Jayaraman, MD, Hari K. V. S. Kumar, MD, Kirtikumar D. Modi, MD, DM.

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CORRELATION BETWEEN BONE MARKERS AND BONE MINERAL DENSITY IN POSTMENOPAUSAL WOMEN WITH OSTEOPOROSIS

K. V. S. Hari Kumar, MD, Jayaraman Muthukrishnan, MD, Abhyuday Verma, MD, and Kirtikumar D. Modi, MD, DM

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Original Article

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ROLE OF THYROID DOPPLER IN DIFFERENTIAL DIAGNOSIS OF THYROTOXICOSIS

K. V. S. Hari Kumar, MD,¹ Vamsikrishna Pasupuleti, MBBS,² Muthukrishnan Jayaraman, MD,¹ Verma Abhyuday, MD,¹ Ramasubba Rayudu B, MD,² and Kirtikumar D. Modi, MD, DM¹

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Association between thyroid hormones, insulin resistance, and metabolic syndrome

Hari K. Kumar, MBBS, MD, Raj K. Yadav, MBBS, MD, Jayaram Prajapati, MBBS, Challa VK. Reddy, MSc, PhD, Manchala Raghunath, MSc, PhD, Kirtikumar D. Modi, MD, DM.

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Obesity and Thyrotropinemia

K.V.S. Hari Kumar, A. Verma, J. Muthukrishnan and K.D. Modi

Department of Endocrinology, MEDWIN Hospitals, Hyderabad, AP, India

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DIABETES TECHNOLOGY & THERAPEUTICS Volume 11, Number 12, 2009 © Mary Ann Liebert, Inc. DOI: 10.1089/dia.2009.0070

Ileal Interposition with Sleeve Gastrectomy for Control of Type 2 Diabetes

K.V.S. Hari Kumar, M.D., Surendra Ugale, M.S., Neeraj Gupta, M.B.B.S., Vishwas Naik, M.S., Pawan Kumar, M.D., P. Bhaskar, M.D., and K.D. Modi, M.D., D.M.

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Acquired perforating dermatoses in patients with diabetic kidney disease on hemodialysis

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Hemodialysis Int 2009 Sep 16 - Epub ahead of print

Role of Color Doppler Sonography in Graves' Disease

West Indian Med J (In Press)

Clinical Profile of Distal Renal Tubular Acidosis – A Single Centre Experience

Saudi J Kidney Dis Transpl (In Press)

Review Article

REVIEW ARTICLE

Glycemic control in patients of chronic kidney disease

K. V. S. Hari Kumar, K. D. Modi, Ratan Jha¹

Departments of Endocrinology and ¹Nephrology, Medwin Hospitals, Hyderabad, Andhra Pradesh, India

Int J Diab Dev Ctries 2007 Dec; 27(4): 99-103.

Verma Abhyuday et al



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HbA1c and Average Blood glucose

Verma Abhyuday, J. Muthukrishnan, KVS Harikumar, Modi K.D

Department of Endocrinology, Medwin Hospitals, Hyderabad, India

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Abstracts in International Meetings

ENDO Abstracts

Abstract #1017

ROLE OF COLOR DOPPLER SONOGRAPHY IN GRAVES' DISEASE

Hari Kumar KVS, MD, Vamsikrishna P, Verma A, Muthukrishnan J, Ramasubba Rayudu B, KD Modi

Abstract #209

ILEAL SWITCH WITH SLEEVE GASTRECTOMY FOR CONTROL OF TYPE 2 DIABETES

Hari Kumar KVS, MD, Suren Ugale, MS, KD Modi, MD, DM, N Gupta, MS, V Naik, MS

AACE Abstracts

ABSTRACT #205

LOW URINE PH PREDISPOSES TO URIC ACID NEPHROLITHIASIS IN TYPE 2 DIABETES

Hari Kumar Kvs, MD, KD Modi, MD, DM, Ratan Jha, MD, DM

Abstract #1008

ASSOCIATION BETWEEN THYROID HOR-MONES, INSULIN RESISTANCE AND METABOLIC SYNDROME

Hari Kumar KVS, MD, KD Modi, MD, DM, CVK Reddy, PhD, M Raghunath, PhD

APPES Abstracts

P03-03 Bone and Calcium Disorders

Long Term Alendronate in Polyostotic Fibrous Dysplasia

Muthukrishnan Jayaraman, Harikumar K.V.S., Abhyuday Verma, Kirtikumar Modi

Endocrinology, Medwin Hospital, Hyderabad, India

P03-04 Bone and Calcium Disorders

Clinical Profile of Distal Renal Tubular Acidosis in Children: A Single Centre Experience

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