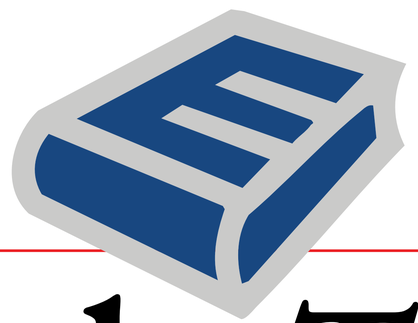

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Diabetes Mellitus and Carbohydrate Metabolism – DiabetesManager

Editors: Kathleen Dungan, MD, MPH and Ketan Dhatariya, MBBS, MSc, MD, MS, FRCP, PhD

PATHOGENESIS OF DIABETES

DIABETES DIAGNOSIS, ASSESSMENT, AND MONITORING

DIABETES DIAGNOSIS, ASSESSMENT, AND MONITORING

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Skin Manifestations of Diabetes Mellitus

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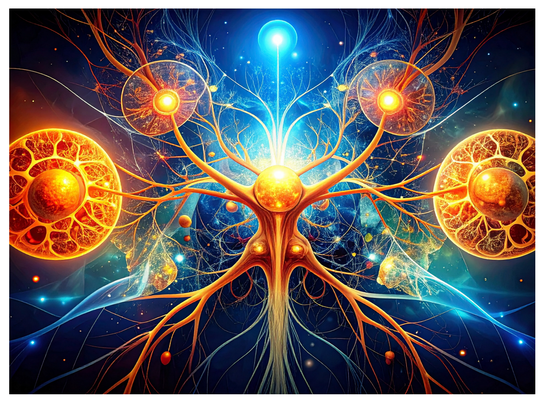

← Back to Diabetes Mellitus and Carbohydrate Metabolism---DiabetesManager Section

ABSTRACT

Diabetes mellitus is a common and debilitating disease that affects a variety of organs including the skin. Between thirty and seventy percent of patients with diabetes mellitus, both type 1 and type 2, will present with a cutaneous complication of diabetes mellitus at some point during their lifetime. A variety of dermatologic manifestations have been linked with diabetes mellitus; these conditions varyin severity and can be benign, deforming, and even life-threatening. Such skin changes can offer insight into patients' glycemic control and may be the first sign of metabolic derangement in undiagnosed patients with diabetes. Recognition and management of these conditions is important in maximizing the quality of life and in avoiding serious adverse effects in patients with diabetes mellitus.

INTRODUCTION

The changes associated with diabetes mellitus can affect multiple organ systems. Between thirty and seventy percent of patients with diabetes mellitus, both type 1 and type 2, will present with a cutaneous complication of diabetes mellitus at some point during their lifetime (1). Dermatologic manifestations of diabetes mellitus have various health implications ranging from those that are aesthetically concerning to those that may be life-threatening. Awareness of cutaneous manifestations of diabetes mellitus can provide insight into the present or prior metabolic status of patients. The recognition of such findings may aid in the diagnosis of diabetes or may be followed as a marker of glycemic control. The text that follows describes the relationship between diabetes mellitus and the skin, more specifically: (1) skin manifestations strongly associated with diabetes, (2) non-specific dermatologic signs and symptoms associated with diabetes, (3) dermatologic diseases associated with diabetes, (4) common skin infections in diabetes, and (5) cutaneous changes associated with diabetes medications.

SKIN MANIFESTATIONS STRONGLY ASSOCIATED WITH DIABETES MELLITUS

Acanthosis Nigricans

EPIDEMIOLOGY

Acanthosis nigricans (AN) is a classic dermatologic manifestation of diabetes mellitus that affects men and women of all ages. AN is more common in type 2 diabetes mellitus (2) and is more prevalent in those with darker skin color. AN occurs more frequently in African Americans, Hispanics, and Native Americans (3). AN is observed in a variety of endocrinopathies associated with resistance to insulin such as acromegaly, Cushing syndrome, obesity, polycystic ovarian syndrome, and thyroid dysfunction. Unrelated to insulin resistance, AN can also be associated with malignancies such as gastric adenocarcinomas and genitourinary cancers, as well as with autoimmune disorders, various medications, and familial disorders (4-4F).

PRESENTATION

AN presents chronically as multiple poorly demarcated plaques with grey to dark brown hyperpigmentation and a thickened velvety to verrucous texture (figure 1). Classically, AN has a symmetrical distribution and is located in intertriginous or flexural surfaces such as the back of the neck, axilla, elbows, palmar hands (also known as "tripe palms"), inframammary creases, umbilicus, or groin. Affected areas are asymptomatic; however, extensive involvement may cause discomfort or fetor. Microscopy shows hyperkeratosis and epidermal papillomatosis with acanthosis. The changes in skin pigmentation are primarily a consequence of hyperkeratosis, not changes in melanin. AN can present prior to the clinical diagnosis of diabetes; the presence of AN should prompt evaluation for diabetes mellitus and for other signs of insulin resistance.

Figure 1. Acanthosis nigricans. From Wikipedia.

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Our single purpose is to help improve the clinical practice of endocrinology around the world through online access to information that is continuously updated. We target the website chapters and content to practicing endocrinologists, fellows, and trainees, and intend that it should be available to anyone around the world without cost.

Physicians caring for their patients often need immediate access to information on disease manifestations, testing procedures, diagnostic criteria, and the very latest information on therapy. With a moment of leisure, the provider may also wish to read in detail about the disease physiology, differential diagnosis, and genetic diagnosis, if available. Traditionally this material came from printed textbooks. Numerous excellent books are available, and they will continue to be cornerstones in physician education. But most printed texts are outdated, expensive, and often unavailable. Currently, nearly every well-trained physician has access to an internet-connected computer or device and increasingly he/she looks to the Web for patient care information. However, the Web is also inundated with medical information that is unreliable or biased. Thus, if providers are to depend on a Web-site, it must demonstrate the highest standards of integrity and reliability by having highly respected editors and authors, independence of operation, and thorough review of all published material.

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Division of Metabolism, Endocrinology, and Nutrition
University of Washington Medical Center

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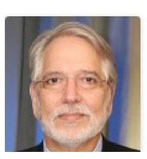

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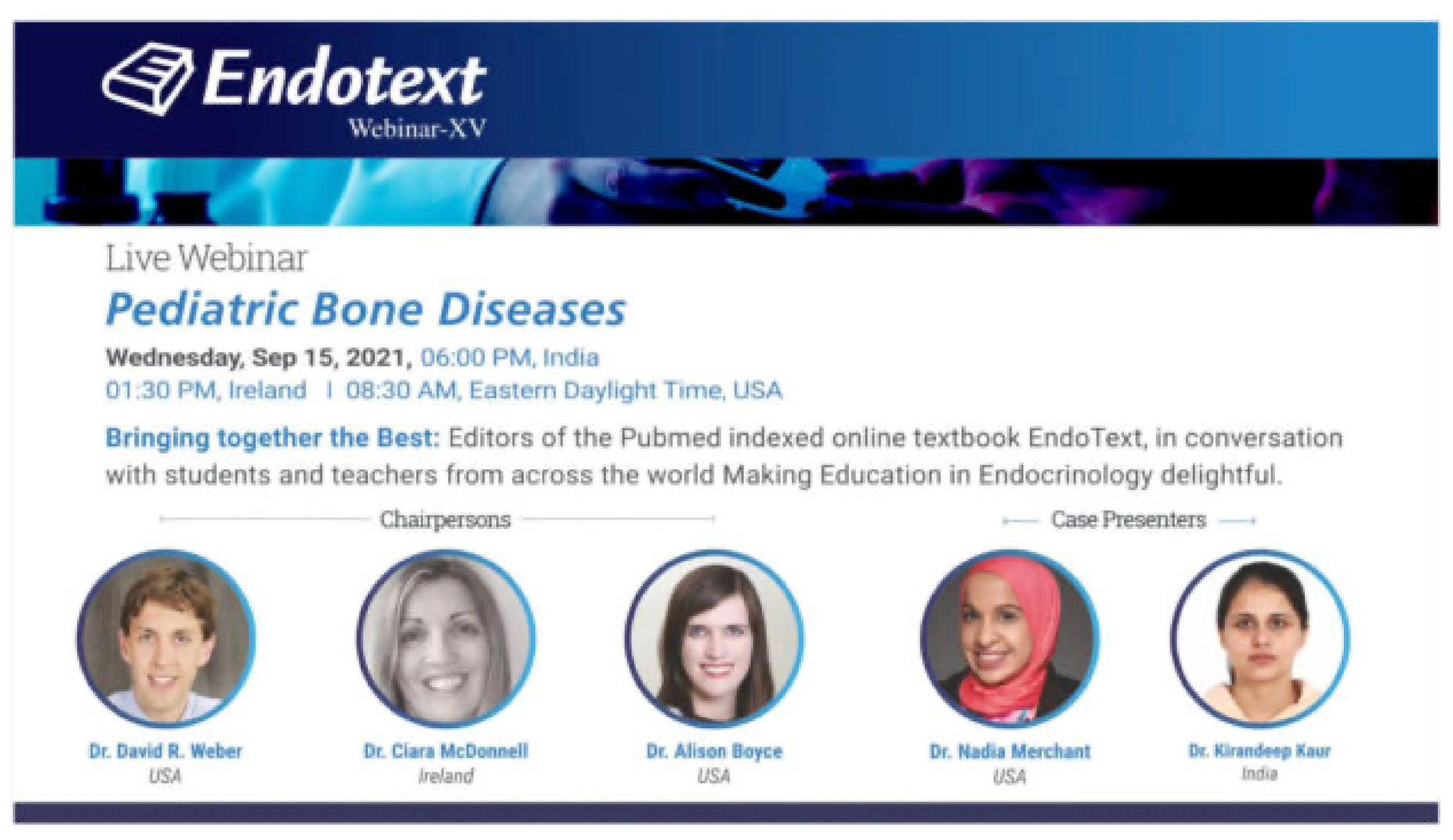

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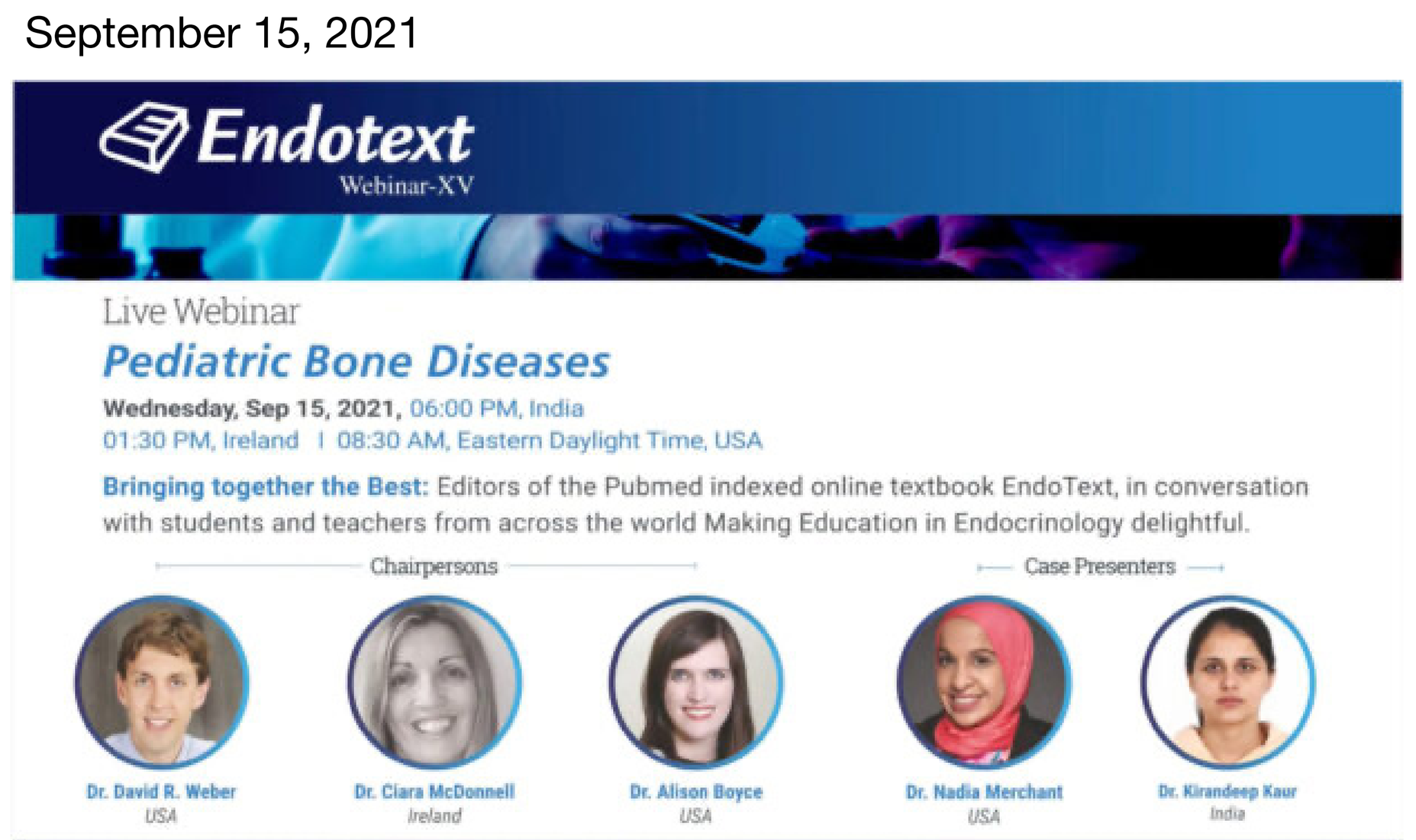

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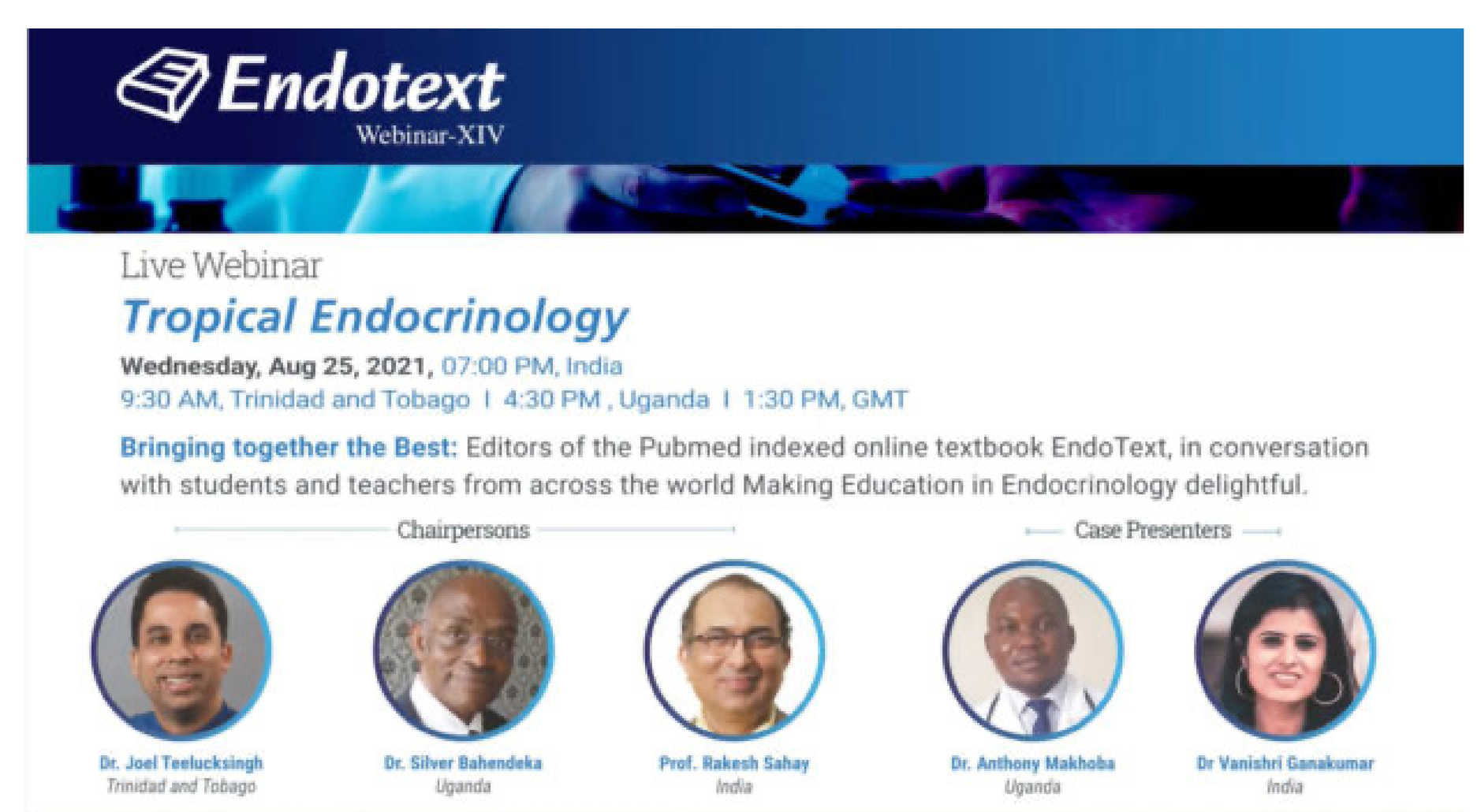

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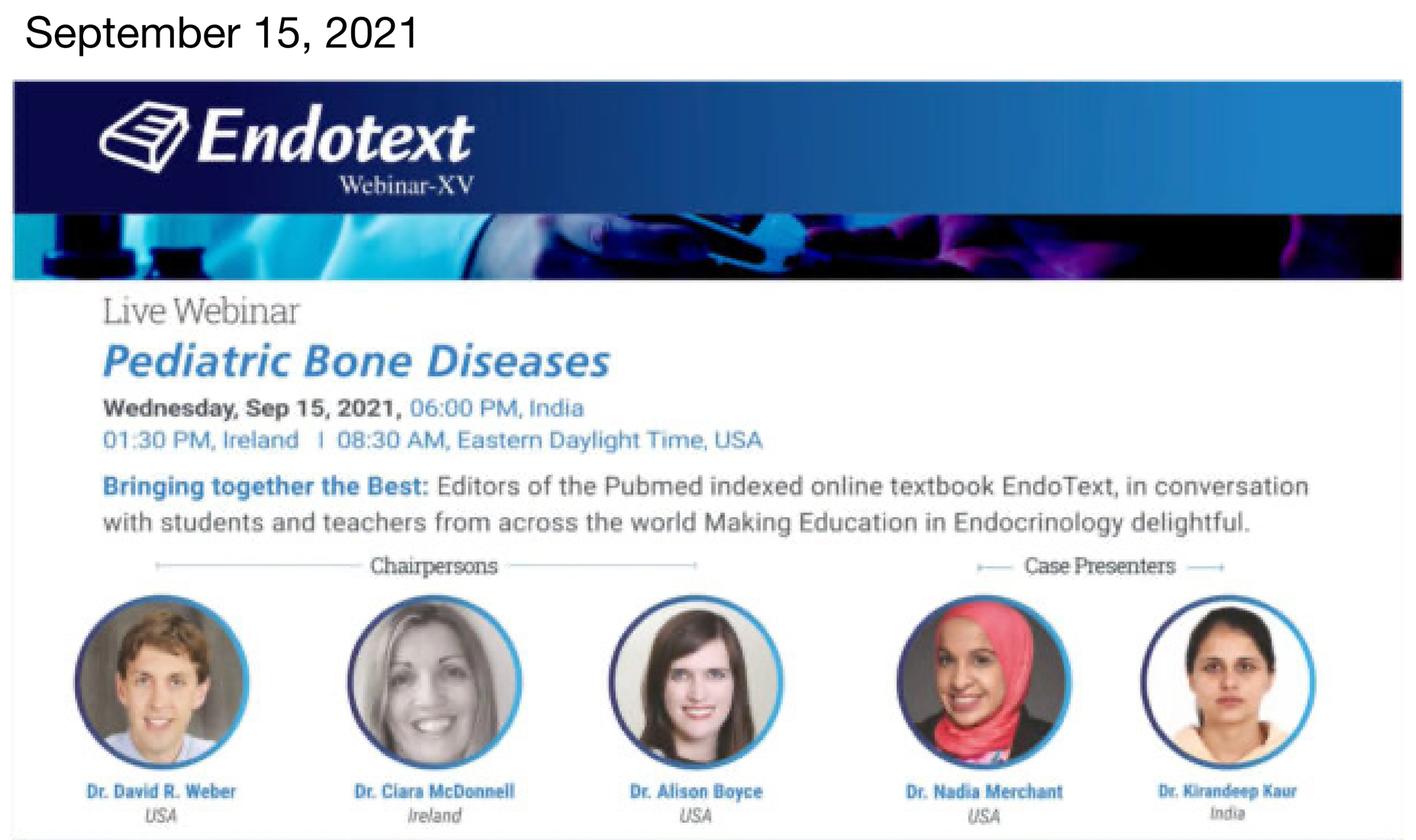

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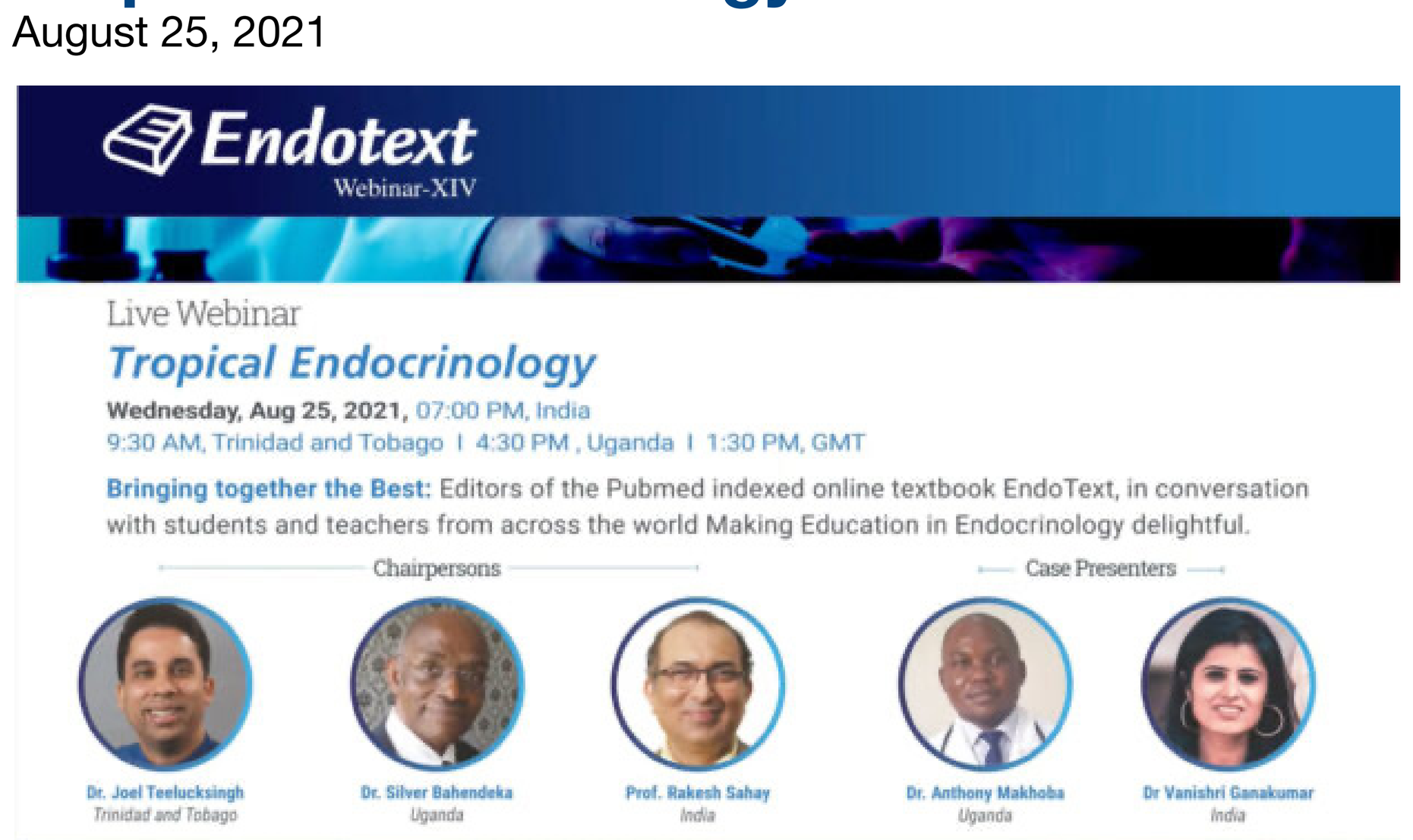

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Cholesterol and Triglycerides

LOW DENSITY LIPOPROTEIN CHOLESTEROL

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Table 1. Etiology of Cushing's Syndrome
ACTH-dependent
Pituitary-dependent Cushing's syndrome (Cushing's disease)
Ectopic ACTH syndrome
Ectopic CRH syndrome (very rare)
Exogenous ACTH administration
ACTH-independent
Adrenal adenoma
Adrenal carcinoma
ACTH-independent bilateral macronodular adrenal hyperplasia (AIMAH) – now known as bilateral macronodular hyperplasia (BMAH)
AIMAH secondary to abnormal hormone receptor expression/function
Primary pigmented nodular adrenocortical disease (PPNAD), associated with Carney complex or sporadic
McCune-Albright syndrome
Exogenous glucocorticoid administration

OTHER

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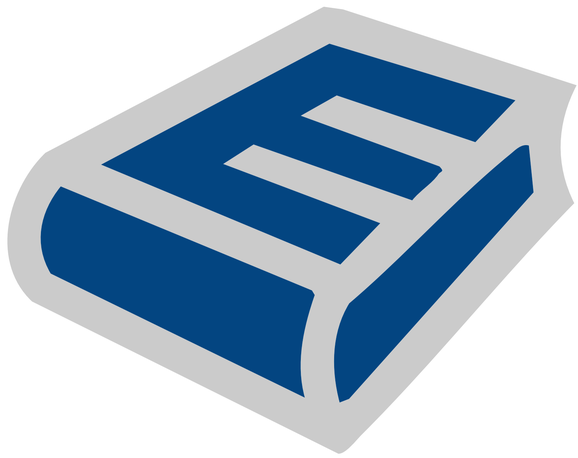

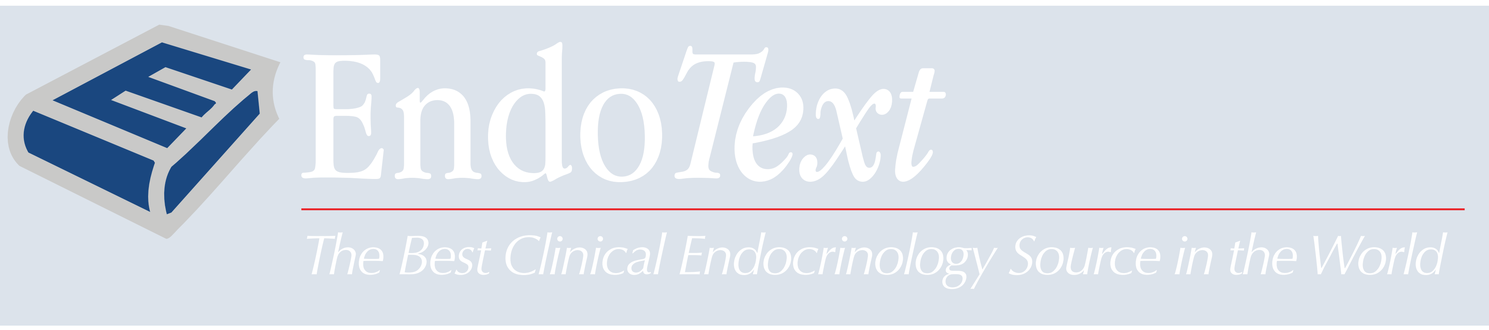

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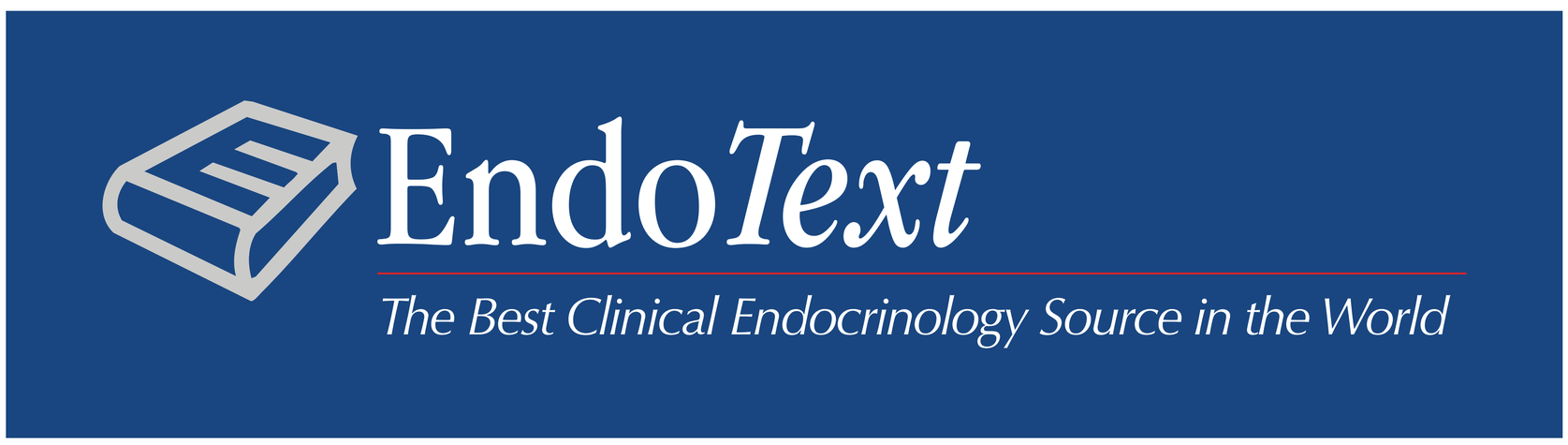

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POST-TRANSPLANT OSTEOPOROSIS

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ABSTRACT

Organ transplantation has become an established treatment for end-stage diseases, and in recent decades, survival rates have significantly improved. This progress has made diagnosing osteoporosis and other complications essential for prevention, treatment, and enhancing the quality of life for transplant patients. Patients who undergo solid organ transplantation often have risk factors for bone loss and fractures, and these risks can increase after transplantation. Post-transplant fractures have been identified as an independent risk factor for overall mortality in these patients. Preoperative low bone mass increases the likelihood of these complications. Osteoporosis is a significant concern that can develop, worsened by glucocorticoids and immunosuppressive therapy, used after transplantation to prevent organ rejection. A major consequence of this is an elevated risk of fractures in bones with reduced strength and quality, leading to increased morbidity and mortality. Additionally, there are notable differences in bone loss and fracture rates among patients with different types of transplanted organs. Initially, reports indicated that in the first year following transplantation, there was a rapid loss of bone mass and an increased rate of fractures. Unfortunately, bone mass achieved after transplantation remains lower long-term compared to that of healthy individuals. Protocols involving less aggressive use of glucocorticoids and immunosuppressants have been introduced to reduce these complications, along with advancements in infection prevention and treatment, to improve the tolerability of treatments and long-term outcomes. Another strategy has been to optimize bone mass in transplant candidates, administering calcium, vitamin D, and bisphosphonates before surgery. Therefore, the prevention and management of bone loss in both transplant candidates and post-transplant patients should be prioritized to reduce the risk of fractures.

INTRODUCTION

Organ transplantation is a well-accepted procedure for treating end-stage diseases such as kidney disease, chronic liver failure, end-stage pulmonary disease, and heart failure. Over the past decade, advancements in this technique have significantly improved patient survival and quality of life. The number of transplants has steadily increased, rising from 106,879 in 2010 to 157,500 in 2020 (1). However, bone loss is a common complication affecting long-term survival and quality of life during patient follow-up.

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After transplantation, rapid and significant bone loss can occur within the first 3-6 months, along with a substantial increase in fracture risk (2,3). The rapid rate of bone loss is likely due to corticosteroids. Greater bone loss has been reported at vertebral and hip sites, along with high rates of fragility fractures. Over half of transplanted patients develop osteoporosis and one-third experience vertebral fractures (4). However, recent studies show a lower rate of bone loss and fractures following transplants, likely due to reduced glucocorticoid doses and modifications in immunosuppression regimens (5,6).

Several risk factors contribute to bone loss in patients including pretransplant disease, aging, hypogonadism, vitamin D deficiency, malabsorption, low body weight, physical inactivity, excessive tobacco or alcohol use, and immunosuppressive therapy (7) (Table 1). Improved management of pretransplant risk factors has led to better bone mineral density (BMD) levels before transplantation.

Table 1. Risk Factors for Bone Disease in Patients with Organ Transplantation

Organ	Potential Risk Factor
Pre-Transplant Factors Affecting All Transplant Patients	-Pre-existing low bone disease -Lower bone mineral density -History of Fractures
Factors Specific to Kidney Transplant Recipients.	-Female gender -Older age -β-microglobulin amyloidosis -Glucocorticoids -Secondary hyperparathyroidism -adynamic bone disease -Chronic metabolic acidosis -Hypogonadism -Vitamin D deficiency -Long-term hemodialysis -Diabetes
Factors Specific to Liver Transplant Recipients.	-Older age -Alcoholism -Hypogonadism -Abnormal vitamin D metabolism -Primary biliary cirrhosis -Cholestasis -Hyperbilirubinemia.
Factors Specific to Heart Transplant Recipients.	-Low levels of vitamin D -Hypogonadism -Long-term heparin -Loops diuretics -Secondary hyperparathyroidism -Physical inactivity, -Therapy with loop diuretics, -Tobacco, alcoholism.