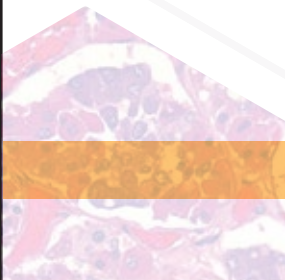
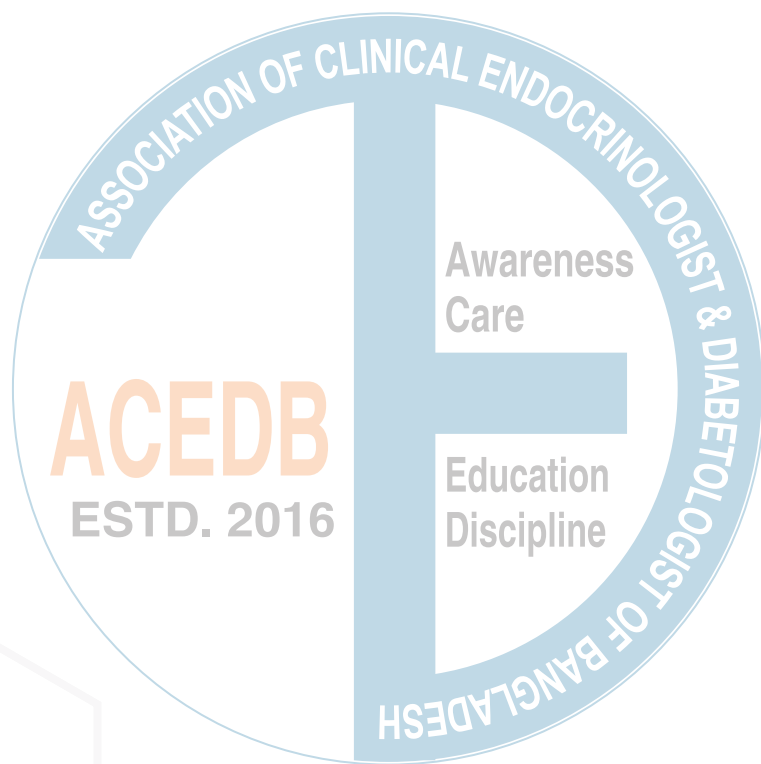
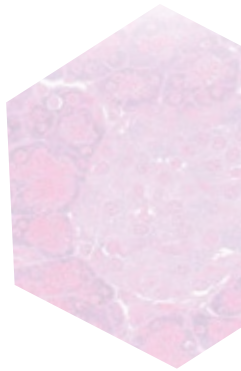


# Scientific papers



## Reducing the risk of NCDs by improving nutritional status during pregnancy, intrauterine life and infancy

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### Abstract

There is now increasing evidence that the Fetal Origin of Adulthood Diseases (FOAD) is important for the understanding and control of non-communicable diseases such as type 2 diabetes mellitus and cardiovascular disease. Fetal programming starts in intrauterine life which results in preferential flow of blood and nutrients to the brain. This can happen because of maternal undernutrition. There is partitioning of nutrients and the end result is intrauterine growth retardation and low birth weight (LBW). The infant may not recover from the nutritional deprivation and remain malnourished. About 30% of women of child bearing age in Bangladesh are underweight, LBW is prevalent in about 25% neonates, and stunting affects 24% of children under the age of 5 years. A study is in progress in Dhaka investigating the impact of early life nutritional status and feeding and the subsequent risk of non-communicable diseases. What is needed now is a program that would field interventions to improve nutritional status of adolescent girls, pregnant women and the first two years of life of the child, a period known as the First 1000 Days. Novel interventions have been discovered based on the knowledge of the gut microbiome. These interventions are food-based and improve nutritional status of children and women by modulating the gut microbiome. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S2*]

**Keywords:** NCDs, Nutritional status Pregnancy, Intrauterine life, Infancy

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## Management of delayed puberty

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### Abstract

Delayed puberty in females is defined as the lack of breast development by 13 years, or a delay of over 4 years between thelarche and completion of puberty or lack of menarche by 16 years. In males, a pubertal delay is evident by a lack of testicular enlargement by 14 years or more than 5 years between testicular enlargement and completion of puberty.

Broadly the causes of delayed puberty in both genders may be classified into three groups -Constitutional delay in growth and development, Hypergonadotropic hypogonadism and hypogonadotropic hypogonadism. The management of delayed puberty involves the identification of the etiology of the delay. The evaluation includes a thorough history, detailed physical examination and specific laboratory investigations (LH, FSH, estradiol in females, and total testosterone in males) and bone age estimation. Other investigations are lead by clinical and laboratory findings. Treatment primarily involves supplementation of sex steroids, the duration and dosage of which is decided according to the cause. Multidisciplinary input, open communication, shared decision-making help in improved outcome of patients with delayed puberty. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S3*]

**Keywords:** Delayed puberty, Management, Hypogonadism

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## Pediatric & adolescent obesity- The new age epidemic

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### Abstract

The last couple of decades have witnessed a pandemic of childhood and adolescent obesity. Most children and adolescents with obesity do not have an underlying disorder; obesity in itself is becoming a harbinger of disease in these children.

School studies have demonstrated an increase in the prevalence of Pediatric Obesity in India from 7% to 21% in the period. The situation is further exacerbated by a greater tendency of metabolic complications at a relatively early stage of adiposity, as reflected by our study that demonstrates the presence of complications in over 50% of obese children. Besides the well-established complications of dysglycemia and dyslipidemia, obese children have a very high prediction of metabolic liver disease and hypertension. High blood pressure in asymptomatic school children is a particular cause of concern. A lack of easily identifiable markers of steatohepatitis makes its early identification difficult. We have demonstrated a high prevalence of transient elastography-identified hepatic fibrosis with ALT levels above 69 IU/L, acting as a surrogate for the same.

Dysglycemia remains a significant cause of concern in pediatric obesity, being present in around 19% of cases. Pediatric type 2 diabetes has a rapid course, as indicated by our study, which showed a need for multiple agents within two years of identification. This highlights the need for early identification, a process marred by a need for more correlation with adult parameters. Marginally elevated HbA1C levels, particularly, have a very low specificity in identifying dysglycemia in our study, indicating the need for higher cutoffs (above 6%). Sleep-disordered breathing is a common correlate of obesity present in 50% of subjects and has been shown to modulate the development of metabolic complications. Mild thyroid dysfunction is common in obesity and usually does not need intervention. The higher prevalence of steatohepatitis and dyslipidemia in these children with “subclinical hypothyroidism” suggests the need for further exploration.

Therapeutic options for childhood obesity are limited, with a lack of long-term efficacy. The use of GLP1 receptor analogs represents a potent option, though currently limited by cost and availability. Bariatric surgery, the most effective long-term treatment of morbid obesity, should be restricted to recalcitrant cases with significant complications. The rapid rise in the prevalence of pediatric obesity, clubbed with a high rate of complication and a lack of therapeutic strategies, highlights the need for primary prevention for the condition. Lifestyle interventions starting from the antenatal period are pivotal to achieving the goal. Exclusive breastfeeding, reduced screen time, increased physical activity, and sleep time are cornerstones of a successful strategy for countering obesity. Timely identification of obesity using local growth references is pivotal, as international charts tend to underestimate the disease burden. Further research into the etiopathogenesis of obesity complications and therapeutic options is expected to reduce the burden of children who have already become obese. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S4*]

**Keywords:** Obesity, Pediatric, Adolescent

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## Diabetic kidney disease: Management update

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### Abstract

Diabetic kidney disease is an all-encompassing term that includes albuminuric DKD and non albuminuric DKD. On the other hand, Diabetic nephropathy denotes the classical clinical course of DKD, characterized by a progressive increase in albuminuria followed by a decline in glomerular filtration rate (GFR). It is a serious microvascular complication that affects approximately 40% of individuals with diabetes. Presently it is the leading cause of end-stage kidney disease (ESKD) worldwide.

The pathogenesis of diabetic nephropathy is similar in type-1 and type-2 diabetes. It involves metabolic, hemodynamic, growth, inflammation and fibrotic factors. But the relative contribution of these factors may vary among patients and overtime. Genetic and environmental factors can modify the appearance of the kidney disease.

Usually, diabetic kidney disease is a clinical diagnosis. A Kidney biopsy is the gold standard test for diagnostic and prognostic information but in most cases is only preferred when an alternative renal pathology is suspected.

In T1 DM, diagnosis of DKD can be made when there is persistent moderate (A2) or severe (A3) albuminuria or a persistent reduction in eGFR to  $<60\text{ml}/\text{min}/1.73\text{m}^2$  occurring at least 5 year after onset of diabetes. In over 95% of cases, diabetic retinopathy will also be present. In T2 DM the clinical diagnosis of DKD can be more challenging due to increased heterogeneity of clinical presentation. DKD can be present at diagnosis. Reduction in eGFR can occur in the setting of normal urinary albumin excretion in both T1 DM and T2 DM. Non-proteinuric DKD often points towards aetiologies that are ischemic in nature or in which tubulo-interstitial pathologies predominate.

Optimal management of DKD is a complex, multi-disciplinary, cross-functional team effort. It bridges from diabetes management in general practice or diabetology settings to CKD management in nephrology setting.

Specific treatment of patient with diabetic nephropathy can be divided in 4 major areas: Inhibition of RAS, Glycemic control, BP control and cardiovascular risk reduction. The single best evidence-based therapy for diabetic nephropathy is therapy with a RAS blocking medication. Sodium-glucose Cotransporter 2 inhibitors, Non-Steroidal Mineralocorticoid receptor antagonists and, to some extent, Glucagon-like peptide 1 receptor agonists (GLP-1 RAs) are game changers in maximizing treatment for better outcome of DKD. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S5*]

**Keywords:** Diabetic kidney disease, Management, Update

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## Flozins in Type 1 DM patients: What to expect?

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### Abstract

Because of their unique mechanism of action of producing renal glycosuria, SGLT-2 inhibitors have been investigated over the past few years as a potential treatment option for Type 1 DM. Moreover, Extraglycemic benefits of SGLT-2 inhibitors like renal & cardiovascular benefits are also two major advantages that type 1 DM patients are likely to get, if they are allowed to take it. Two of the SGLT-2 inhibitors, dapagliflozin & sotagliflozin once temporarily licensed for use by the European Medical Agency (EMA) as an adjunct to insulin therapy in adults with T1D with a body mass index of 27 kg/m<sup>2</sup> or higher, subsequently got withdrawal of their authorization because of the high frequency of DKA. Beyond DKA, both dapagliflozin and sotagliflozin can cause intravascular volume depletion, which can be overcome by dose reduction. The EASE Trials, with empagliflozin 10 and 25 mg doses plus a unique lower dose (2.5 mg) showed that ketoacidosis rate was comparable between empagliflozin 2.5 mg and placebo but increased with 10 mg and 25 mg with improved glycemic control & weight reduction. In a real-world clinical experience in the off-label use of empagliflozin, Fallatah et al showed a significant reduction in HbA1c of 0.8% from baseline and an average non-significant weight reduction of 1.7 kg over 15 months. Despite the improvements in glycaemic control, weight loss, and other risk reductions with SGLT2i, several studies have documented an important increase in the risk of DKA. Further studies will be needed to determine the safety of this therapy in T1D and to determine the type of patient those are potentially to be benefitted based on the risk-benefit estimation. Success of Flozins in type 1 DM will largely depend on careful selection of the patients who will enjoy its glycemic as well as extra-glycemic benefits while minimizing risk of DKA. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S6*]

**Keywords:** Flozins, Type 1 DM, SGLT-2 inhibitors

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## Fructosamine: A new frontier in Diabetes care

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### Abstract

Diabetes mellitus has emerged as the leading non-communicable disease which has affected the whole globe and across the age groups. It is directly responsible for the causation and the outcome of other non-communicable diseases like coronary artery disease, cerebrovascular disease, peripheral vascular diseases etc. There have always been search for markers for its early detection, detection in susceptible groups like first degree relatives of diabetics and those with metabolic syndrome and also for the close monitoring of recent past blood sugar levels in special situations. This is also important for the prevention or delay of microvascular and macro-vascular complications of Diabetes mellitus.

Glycosylated hemoglobin has been used for the diagnosis and the monitoring of the blood sugar levels of previous 3 months. However, the correlation between glycaemia and HbA1c is not perfect. For instance, in both diabetics and non-diabetics, HbA1c levels are genetically determined, with heritability of 50%. Other factors like age, hemoglobinopathies, drugs and some diseases may also have an effect.

Increased blood sugar levels promote non-enzymatic glycation of proteins through the Maillard reaction with Schiff base formation through Amadori rearrangements. Glycated hemoglobin A1c (HbA1c) is formed when the N-terminal valine residue of the beta chain is glycated. Approximately 50% of HbA1c reflects blood glucose in the past 1 month, 25% for the past 1 to 2 months, and 25% for the past 2 to 4 months. Hemoglobin (Hb)A1c is adult Hb (HbA) with glucose bound to its b chain N-terminal valine, resulting from non-enzymatic glycation in erythrocytes. Hence, HbA1c concentration reflects the concentration of glucose to which erythrocytes are exposed over their lifespan.

Fructosamine is a ketoamine formed when the carbonyl group of glucose reacts with an amino group of a protein, thus forming glycated serum proteins (mainly albumin). Fructosamine determination is the most widely used alternative to HbA1c. Some studies have attempted to interpret the parallel measurement of fructosamine and HbA1c. Compared to glycated hemoglobin (HbA1c), serum fructosamine has a shorter half-life and reflects the physiology of glucose in the extracellular space, whereas HbA1c indicates glycosylation in the intra-erythrocyte compartment. Thus, fructosamine is of clinical utility to show short-term glycemic control (2–4 weeks) and when underlying conditions make HbA1c interpretation difficult (i.e., anemic states, hemoglobinopathies or renal diseases). [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S7*]

**Keywords:** Fructosamine, Diabetes, Ketoamine

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## Management of youth-onset type-2 diabetes: Is it different from adult?

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### Abstract

Youth-onset type 2 diabetes mellitus (T2DM) presents a growing public health challenge worldwide. It results from a complex interaction between epigenetic, genetic, and environmental factors. It has been linked to a rising trend of childhood obesity. The early onset of diabetes significantly increases the risk of long-term complications and poses unique management challenges. Effective management strategies require a comprehensive approach, integrating medical treatment, lifestyle interventions, and psychosocial support. Early diagnosis, continuous monitoring, individualized treatment plans, and strong support systems are vital for optimizing outcomes and minimizing the impact of the disease on young lives. This presentation will cover the overall management of type-2 diabetes in young people, highlighting specific issues that are different from late-onset diabetes. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S8*]

**Keywords:** Youth-onset type-2 diabetes, Management, T2DM

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## Youth-onset diabetes: Sharing experience from a nationwide prevalence study by ‘Study on diabetes in young’ group

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### Abstract

Youth-onset DM is progressively rising to be a medical and social concern in developing countries like Bangladesh. The ‘Study on Diabetes in Young’ (SODY) group, BSMMU, carried out several studies on different aspects of youth-onset DM, including pathophysiological mechanisms, role of cytokines, micronutrients, and genetic predisposition. Recently, the group carried out a nationwide study funded by the Non-Communicable Disease Control (NCDC) program, DGHS, to estimate the prevalence of DM in young (10-34 years) and to identify related factors at individual, household, and community levels. The SODY group traveled to all eight administrative divisions of Bangladesh during the last 6 months and screened 2152 pre-registered young participants selected by multistage random sampling. OGTT was performed in individuals not known to have DM to ascertain the glycemic status. Necessary laboratory equipment (including semi-automatic biochemical analyzers) was carried to the study sites to ensure a uniform, standard biochemical assay. The study was greatly facilitated by the involvement of local endocrinologists and other medical professionals. It was observed that 1 in every 25 Bangladeshi youth had DM (prevalence 4.2%; higher in young adults, 7.2%, than in adolescents, 1.4%), and two-thirds of them are not aware of it. The prevalence of prediabetes was more alarming – 1 in every 5 (18.5%). The study also investigated the predisposing factors for dysglycemia at an early age, which include family history of DM, overweight/obesity, smoking, smokeless tobacco use, urban living, and higher socioeconomic conditions. To combat the possible future catastrophe brought about by the surge of young-onset DM, promoting healthy living and screening young people at risk is essential. SODY group aims to continue research on the topic and seeks national and international collaboration. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S9*]

**Keywords:** Youth-onset diabetes, SODY group, Prevalence

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## Changing phenotypes of PCOS and diagnostic update 2023

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### Abstract

Polycystic ovary syndrome (PCOS) is the most prevalent endocrine and metabolic disorder, affecting 6 to 10% of reproductive-aged women worldwide. As a heterogeneous disorder, PCOS reflects various potential etiologies with diverse clinical presentations. Therefore, the definition of PCOS is still a matter of debate. The features of PCOS are clinical and/or biochemical hyperandrogenism (HA), ovulatory dysfunction (OD), and polycystic ovarian morphology (PCOM). There are three different diagnostic criteria sets (NIH criteria 2012, Rotterdam 2003 and AE-PCOS criteria 2006) and the syndrome could present with four sub phenotypes. Finally, in 2012, NIH consensus panel proposed the phenotypic approach to classify PCOS. PCOS phenotypes are currently classified as phenotype-A (full blown PCOS: HA + OD + PCOM), phenotype-B (non-PCO PCOS: HA + OD), phenotype-C (HA + PCOM), and phenotype-D (non-hyperandrogenic PCOS: OD + PCOM). Based on the sub phenotype classification introduced by the NIH PCOS workshop in 2012, Phenotype-A is the most common phenotype reported in clinical patients whereas phenotype-C is more common in unselected populations, phenotype D is least common.

2023 updated guideline strongly emphasis on HD +/- OD along with exclusion of other causes such as CAH, CS, ovarian or adrenal tumor. PCOM or AMH is not a must in adults and is strongly discouraged in adolescents because it is falsely labeled as PCOS.

The phenotypic approach of categorizing PCOS has a number of practical implications. Full-blown PCOS (phenotype A) is at a higher risk of adverse metabolic and cardiovascular outcomes as compared with the others, and phenotype D is the least severe phenotype. Also, this group is more likely to be clomiphene-resistant. Timely diagnosis and identification of specific sub phenotypes is essential to address presenting complaints of a patient, determine individualized treatment targets, and prevent long-term health consequences of the syndrome. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S10*]

**Keywords:** Polycystic ovary syndrome, Phenotypes, Diagnostic update

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## Approach to a case with disorders of sex development

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### Abstract

Disorders of sex development (DSD) occur in infants who exhibit a discrepancy between their genetic, gonadal, and phenotypic sex. The estimated incidence of these disorders is about 1 in 4,500 live births. In newborns with a 46 XX karyotype, the most frequent cause of genital ambiguity is congenital adrenal hyperplasia, resulting from 21-hydroxylase deficiency. This leads to excessive androgen production and subsequent virilization in genetic females. Another relatively common cause is mixed gonadal dysgenesis, often due to 45X/46XY mosaicism. Cases involving under-virilized genetic males are rarer. The birth of a child with DSD is both a medical and social emergency, requiring the prompt involvement of a multidisciplinary team to ensure a timely and accurate determination of the child's gender. This process, however, should not be rushed. Parents are often confused and overwhelmed by the diagnosis and need empathetic and informed guidance from health professionals. While gender assignment is crucial, decisions must be made with careful consideration, allowing parents to make informed choices after thorough consultation with the medical team.

The once common practice of early gender assignment and surgical intervention has faced significant scrutiny and opposition from patients, advocacy groups, and healthcare providers. In most cases, genital ambiguity is recognized at birth, which immediately raises the question of how to assign the child's sex. The expert should promptly meet with the parents, explaining that the baby's genitals are incompletely developed, and that a specialized team led by an endocrinologist will conduct a comprehensive evaluation. This evaluation will involve the parents' input and consent before any decisions are made regarding gender.

It is suggested that the doctor communicate with parents using the following language: "There is an issue with how the baby's genitals have formed, and at this time, we cannot determine whether the child should be raised as a boy or a girl." During this period, it is important to refer to the infant as "the baby" without assigning a specific gender. There is no medical justification to separate the mother and baby. Both sex and name assignment should be delayed, and a prompt, thorough evaluation by a multidisciplinary team should be initiated, aiming to make a gender determination within two weeks. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S11*]

**Keywords:** Disorders of sex development, Genital ambiguity, Sex assignment

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## Adrenal insufficiency test selection and updated management

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### Abstract

Adrenal insufficiency (AI) is frequently misdiagnosed due to non-specific symptoms and signs early in the disease's course, making timely identification difficult. AI is a medical disorder characterized by adrenal cortical hypofunction, which includes insufficient cortisol synthesis that fails to meet the demands of severe physiological stress. This disease can be functionally classified into three types based on its underlying causes: Primary, central, and hypothalamic-pituitary-adrenal (HPA) axis suppression. The significant contrast among these three is that HPA axis suppression is by definition reversible with a progressive discontinuation of therapy, whereas primary and central AI are usually permanent. Hormonal assays required for diagnosis are routinely available throughout the world; nonetheless, significant barriers remain in both diagnosing AI and determining the most appropriate replacement therapy. A serum cortisol level of  $<4 \mu\text{g/dL}$  ( $110 \text{ nmol/L}$ ) at 8:00 AM, paired with a plasma ACTH level that exceeds the normal reference range (typically  $>100 \text{ pg/mL}$ ), can diagnose primary AI without dynamic testing. On the contrary, the diagnosis of central AI is frequently more difficult than primary AI, due to the absence of dynamic tests in most published studies of diagnostic criteria. Furthermore, even after being diagnosed, it is difficult to replicate the natural cortisol and mineralocorticoid cycles. Well-designed, double-blind, randomized studies comparing diverse treatment regimens and formulations are still required to address these ongoing issues. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S12*]

**Keywords:** Adrenal Insufficiency, Management, Tests

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## Primary aldosteronism: Updates and future directions

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### Abstract

Primary aldosteronism (PA), or Conn's syndrome, is a condition characterized by overproduction of aldosterone, a hormone produced by the adrenal glands. This overproduction leads to sodium retention, hypokalemia and hypertension. In patients with only one abnormal adrenal gland, laparoscopic surgery offers an opportunity for cure of hypertension. Despite PA being the most common treatable and curable cause of hypertension, <1% of patients worldwide are diagnosed and treated. This lecture will be covering newer aspects in our understanding of PA, its diagnosis, and subtyping modalities. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S13*]

**Keywords:** Primary aldosteronism, Conn's syndrome, aldosterone

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## Pituitary incidentaloma: An endocrinologists' dilemma

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### Abstract

A pituitary incidentaloma (PI) is a previously unsuspected pituitary lesion that is discovered on an imaging study performed for an unrelated reason. PIs have become common in clinical practice because of increased use of imaging and radiologic advances. The average frequency of a PI is 10.6% with the majority being pituitary adenoma. All patients with PI should be screened for hyperprolactinemia and acromegaly whereas testing for hormonal deficiency is recommended for lesions larger than 6.0 mm. Most micro-incidentalomas are small nonfunctioning adenomas or cysts, which can be conservatively managed. For larger lesions, appropriate medical or surgical treatment should be offered depending on hormonal status, compressive or pressure effects on surrounding structures. For incidentally detected lactotroph, somatotroph, and corticotroph adenomas, disease-specific management guidelines apply. Here, we highlight the necessity of proper evaluation of a PI through some real life case scenario and emphasize on development of population specific guideline of management. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S14*]

**Keywords:** Pituitary incidentaloma, Micro-incidentaloma, Non-functioning adenoma

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## Management of thyroid carcinoma: Role of an Endocrinologist

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### Abstract

Thyroid cancer, which includes papillary, follicular, medullary, and anaplastic carcinoma, the most common of all malignant endocrine tumors, has seen an increasing incidence globally over the past few decades. However, the number of thyroid cancer survivors has increased substantially with the advent of new risk stratification and appropriate management.

The management of thyroid cancer is a complex and multifaceted process that requires the involvement of multiple healthcare professionals at different stages. Among these professionals, the endocrinologist plays a pivotal role in the multidisciplinary team.

Endocrine expertise is essential in the evaluation of thyroid nodules, interpretation of biochemical and imaging studies, conduction of fine needle aspiration (FNA) biopsies, and determination of the nature of thyroid cancer for diagnosis. Once malignancy is confirmed, the endocrinologist collaborates with surgeons, nuclear medicine specialists, and sometimes oncologists to establish a comprehensive treatment plan, which may include thyroidectomy, radioactive iodine therapy, thyroid hormone suppression therapy, or systemic therapy.

In post-treatment, they play a key role in monitoring recurrence through regular clinical evaluations, imaging, and serum markers such as thyroglobulin. Additionally, an endocrinologist monitors the management of hypothyroidism, maintains TSH suppression, and addresses complications like hypocalcemia and other metabolic changes.

Moreover, endocrinologists provide long-term surveillance and individualized care to ensure optimal quality of life and survival rates in thyroid cancer patients.

In summary, the endocrinologist's role in thyroid cancer management is pivotal, spanning diagnosis, risk stratification, treatment planning, and long-term surveillance, all in collaboration with a multidisciplinary team to optimize the patient outcomes. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S15*]

**Keywords:** Thyroid carcinoma, Management, Endocrine perspective

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## Improving the quality of life of people with hypoparathyroidism

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### Abstract

Parathyroid hormone is one of the major hormone that regulates serum calcium via effects on bones, kidney and gastrointestinal tract. Hypoparathyroidism occurs when there is destruction of parathyroid gland, abnormal development, altered regulation or impaired action of parathyroid hormone. Hypoparathyroidism may be associated with a spectrum of clinical manifestations ranging from few to life threatening seizure. Clinical manifestations depend upon severity of hypocalcemia, rate of development of hypoparathyroidism and chronicity. Hypoparathyroidism should be suspected in any patient with mild or severe symptoms of neuromuscular irritability or incidental laboratory findings of hypocalcemia. Diagnosis is confirmed by simultaneously measuring serum calcium, serum magnesium, phosphorus, serum creatinine and intact PTH level. The goal of therapy in all patients with hypoparathyroidism are to relieve symptoms, to raise and maintain serum calcium in low normal range and to prevent kidney stones. Advances in our knowledge of hypoparathyroidism have led to greater understanding of the disease itself and approach to it. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S16*]

**Keywords:** Hypoparathyroidism, Quality of life, Hypocalcemia

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## Ongoing challenges in success or failure in osteoporosis care

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### Abstract

Osteoporosis is the most common bone disease in humans and represents a major public health problem. It remains silent until complicated by fractures that can occur following minimal trauma. The most common fractures are those of the vertebrae (spine), proximal femur (hip), and distal forearm (wrist). Non-pharmacological management of osteoporosis includes adequate calcium and vitamin D intake, weight-bearing exercise, smoking cessation, limitation of alcohol/caffeine consumption, and fall-prevention techniques. The goal of pharmacological therapy is to reduce the risk of fractures. Medications to treat osteoporosis are categorized as either antiresorptive (i.e., bisphosphonates, selective estrogen receptor modulators (SERMs), estrogens, calcitonin, and denosumab) or anabolic (i.e., teriparatide). Antiresorptive medications primarily decrease the rate of bone resorption while anabolic medications increase bone formation more than bone resorption. While several medications have overlapping indications, it is important to note that not all osteoporosis medications are approved by the Food and Drug Administration (FDA) to treat Post-menopausal Osteoporosis (PMO), osteoporosis in men, and/or Glucocorticoid Induced Osteoporosis (GIO). According to AACE/ACE guidelines, first-line treatment for most PMO patients at high risk of fracture includes alendronate, risedronate, zoledronic acid, and denosumab. For those who cannot use oral therapy and are at high risk of fracture, use of teriparatide, denosumab, or zoledronic acid is recommended. This recommendation is also reflected in the ACP guidelines. Treatment duration of three to five years for PMO. Bisphosphonates remain the first-line and most cost-effective treatment option for osteoporosis, but there is increasing concern about their long-term safety. Medications with novel mechanisms to treat osteoporosis can be expected in the near future. Although appropriate BMD screening and treatment with medication is important, osteoporosis is preventable with proper management of diet, lifestyle, and fall prevention interventions. [*J Assoc Clin Endocrinol Diabetol Bangladesh, 2024;3(Suppl 1): S17*]

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