Diabetes Insipidus

Diabetes insipidus (DI) is a condition that occurs when there is insufficient production of antidiuretic hormone (ADH), also known as vasopressin. ADH is a hormone that helps the kidney and body to conserve the correct amount of water by controlling the output of urine. ADH is secreted by the hypothalamus to decrease the amount of urine output so that dehydration does not occur, stored in the pituitary gland and released into the blood. Diabetes insipidus can be of central and nephrogenic origin. Central DI occurs after damage to hypothalamus or pituitary due to head injury, surgery, genetic disorders or brain tumors (craniopharyngioma), while nephrogenic DI is a lack of response of the kidney to normal ADH levels due to drugs, chronic disorders, sickle cell disease or polycystic kidney disease. Most common symptoms of DI are excessive thirst (polydipsia) and urine production (polyuria), inability to concentrate the urine along with hypernatremia, seizures and dehydration. In addition children develop irritability, poor feeding, failure to thrive and fever. DI is diagnosed with history, labs (urine/blood tests), water deprivation test DDAVP trial (which should be done in the hospital) and imaging (MRI/CT Scan). Diabetes insipidus can lead to further brain damage, impaired mental function, intellectual disability, hyperactivity and restlessness. Management of diabetes insipidus depends on the cause of DI. Treatment includes adequate hydration and antidiuretic hormone medication (oral, injection or nasal spray), or medications that stimulate the production of ADH such as Non-steroidal antiinflammatory or chlorpropamide. Depending on the cause of diabetes insipidus the disease can be temporary or permanent. Children with central and nephrogenic diabetes insipidus can lead full healthy lives with proper and monitored management.

References:
Bariatric Surgery

Childhood obesity has reached global epidemic proportions. Obesity is defined as BMI > 95th percentile for sex and age, with severe obesity within the 99th percentile. Pediatric obesity affects health by causing metabolic syndrome, hypertension, dyslipidemia, diabetes (insulin resistance), obstructive sleep apnea, musculoskeletal complaints (fracture, low extremity misalignment), nutritional deficiencies (Vit D and iron deficiency), depression and poor self-esteem. Screening for obesity after age of two years should be standard of care for primary physicians. Breast feeding has a protective effect in childhood obesity and should be encouraged. The basis of management of child obesity remains a goal of energy expenditure that surpasses energy consumption. Daily physical activity of 60 minutes daily should be encouraged between families. If lifestyle modification does not produce significant weight loss bariatric surgery might be needed in the adolescent age. Adolescent considered for bariatric surgery should be severely obese with BMI > 40 and comorbid conditions described above, attained adult stature, failed six months of conventional weight management, demonstrate comprehensive psychological evaluation, avoid pregnancy for at least one year postoperatively, be capable to adhere to nutritional guidelines postoperatively and have decisional capacity. Both Roux-en-Y gastric bypass (RYGB) and laparoscopic sleeve gastrectomy (LSG) are effective in achieving significant weight loss. Obesity related comorbidities resolve almost universally with remission of diabetes, hypertension, dyslipidemia and sleep apnea. Metabolic syndrome and concomitant cardiovascular morbidity and mortality is decreased in severely obese adolescent undergoing bariatric surgery. RYGB increases insulin sensitivity by four times in adolescent with and without diabetes after surgery. Perioperative complications seen in 22% and include anastomotic stricture, reoperation, anastomotic leak, dumping syndrome and dehydration. In absence of supplementation inadequate absorption of calcium, Vit D, iron, Vit B1, B12, A and folate can occur resulting in nutritional deficiencies.

References:
**London Sign**

The London sign is a trivial looking circular bruising in the epigastric area after blunt abdominal trauma. The London sign usually corresponds to the shape and size of the bicycle handle. The sign means that the impact was sharp and strong enough to cause some kind of internal visceral damage to the child. This type of epigastric blunt trauma blow can cause pancreatic or duodenal injury of sufficient severity since these two organs get crushed between the blow and the vertebral column. Presence of the London sign needs hospital admission and further imaging investigation in most cases as an underlying internal organ injury might be missed. The shape and size of the bruising depends on the object transmitting the impacting force. As such the victim can present bicycle handle, tire marks, shirt buttons, knuckles of hand or wrist watch marked in the epigastrium all conforming and representing the London sign. The damage to the internal viscera might include pancreatic contusion with or without main duct transection, intramural duodenal hematoma and perforation of the duodenum. A similar sign, known as the seatbelt sign occurs after high impact blunt injury from motor vehicle accidents. The seatbelt sign is a linear ecchymosis of the abdominal wall corresponding to the belt. The seatbelt complex describes an injury to the intestine (including the rectum), lumbar spine and other abdominal organs associated with the belt. Chest injury, fracture of the sternum and ribs along with injury of the major vessels are also included in this complex. The prevalence of intestinal injury in patients with the seatbelt sign is almost 15%. Physical examination is sometimes inaccurate in the diagnosis of blunt abdominal injury and requires a high index of clinical suspicion. Children with the London and Seatbelt sign should undergo further abdominal imaging such as US and CT-Scan in search of missed visceral injury.

**References:**

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