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Refeeding Syndrome

Refeeding syndrome (RFS) is a potentially lethal condition defined as severe electrolyte and fluid imbalance associated with metabolic abnormalities in malnourished patients undergoing refeeding orally, enterally or parenterally. Children at risk for developing RFS include those with chronic malnutrition, protein malnutrition, marasmus, cancer, alcoholism, following prolonged starvation, massive weight loss, prolonged IV repletion and anorexia nervosa. Predominant features of RFS include severe hypophosphatemia, altered glucose metabolism, hypokalemia, hypomagnesemia and thiamine deficiency leading to serious cardiac, pulmonary, hematological and central nervous system complications. RFS can occur after five to ten days of starvation. Fats become the predominant source of energy. Insulin is suppressed and glucagon increases. Intracellular and extracellular ions are chronically depleted including sodium, potassium, magnesium and phosphorus. When normal nutrition is resumed insulin secretion increases and glycogen, fat and protein synthesis begins. The decreased phosphate depletes ATP and other phosphate-based molecules like 2,3-DPG intracellularly leading to poor oxygen delivery and electrolyte abnormalities from movement of ions into the cell. The patient with RFS can develop arrhythmias and cardiac dysfunction, gastrointestinal complaint, muscle weakness, myalgia, dyspnea, respiratory failure, hematologic disturbances and even death. Labs performed reveal hyponatremia, hypokalemia, hypomagnesemia, hypophosphatemia, hyperglycemia and trace and vitamins' deficiencies. Management of RFS includes slow nutritional supplementation approximately 20 Kcal/kg/day or 25% of calories requirements daily advancing three to five days for the total value with administration of deficient nutrients, trace elements, mineral and vitamins. RFS is most commonly reported in those receiving TPN but can occur also in patients who received intravenous saline-dextrose, tube feeding or an oral diet.

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Tap Water Iontophoresis

Idiopathic (primary) excessive hyperhidrosis is excessive sweating without known etiology. It usually occurs in the palmar, plantar, axilla, facial or cervical region usually bilateral causing in the child's social, psychological, emotional and professional difficulty. Hyperhidrosis usually starts in childhood and may continue for the rest of the life, does not occur during sleep for it is normally stimulated by emotion and stress. Management of hyperhidrosis includes aluminum chloride antiperspirants, systemic anticholinergic agents, injection of botulinum toxin A (Botox) and when is very severe surgical thoracic sympathectomy. Recently tap water or normal saline iontophoresis (TWI) has been found to be safe, effective and inexpensive treatment for idiopathic hyperhidrosis. Iontophoresis is an electrical treatment that includes absorption of a drug or chemical in the form of ions on the skin. It is used in local anesthesia, antibacterial modality, neurogenic pain relief, edema, chronic ulcer repair, skin fungi infection and hyperhidrosis. Pore obstruction of sweat ducts secondary to hyperkeratinization, impairment of the electrochemical gradient of sweat and biofeedback mechanism may be involved in hyperhidrosis following iontophoresis using tap water or normal saline. Iontophoresis is an effective treatment with an immediate result possibly due to mechanical or electrochemical occlusion of sweat ducts which can last for at least four weeks. The efficiency of normal saline iontophoresis has been found to be greater and 70% more efficient than that of tap water. Burning and pin pricking sensations are very common and may be felt by all treated patient. Dryness, cracking, erythema and vesiculation which are transient have also been reported after iontophoresis. Pregnant women, people with pacemaker or metal implants, cardiac condition or epilepsy are contraindications for use of iontophoresis therapy. TWI should be offered before considering more invasive surgical procedures.

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Transverse Testicular Ectopia

Transverse (or crossed) testicular ectopia (TTE) is a very rare congenital condition in which both testes migrate toward the same hemiscrotum through the same inguinal canal. Theories that explain this rare behavior of both testis residing in the same hemiscrotum include both testis being derived from the same germinal ridge through duplication of the gonadal primordium, mechanical effect of persistent Müllerian duct

structures preventing testicular decent and causing both testicles to descend toward the same scrotum and defective gubernacular formation. The ectopic testis may lie in the opposite hemiscrotum, inguinal canal or at the deep inguinal ring. TTE is usually associated with other anomalies such as persistent Müllerian duct syndrome, true hermaphroditism, inguinal hernia (most common association), renal agenesis and scrotal anomalies. The diagnosis of TTE is made on clinical examination and confirmed using scrotal ultrasonography, CT-Scan, MRI or laparoscopy. The patient usually present with uniform symptoms of an inguinal hernia on one side and an impalpable testis on the other side. Each testis has a corresponding spermatic cord but in most cases the two cords fuse to form an inseparable thick-walled structure several centimeters proximal to the testis. Each testis has its own blood supply. Both testes share a single patent processus vaginalis. TTE can be reclassified depending if it had a hernia (type 1), is accompanied with persistent Müllerian structures (type 2), or associated with other disorders mentioned above (type 3). Surgical management consists of either trans-septal orchiopexy or extraperitoneal transposition orchiopexy. Infertility and risk of seminoma are known long-term complications needing close follow-up.

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***Edited by: Humberto Lugo-Vicente, MD, FACS, FAAP**

**Professor of Pediatric Surgery, University of Puerto Rico - School of Medicine,
Rio Piedras, Puerto Rico. Director - Pediatric Surgery, San Jorge Childrens
Hospital.**

**Address: P.O. Box 10426, Caparra Heights Station, San Juan, Puerto Rico USA
00922-0426.**

Tel (787)-999-9450 Fax (787)-720-6103 E-mail: titolugo@coqui.net

Internet: <http://home.coqui.net/titolugo>

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