Abstracts

Refractory hypoglycemia secondary to topical salicylate intoxication

Raschke R, Arnold-Capell PA, Richeson R, Curry SC.
Arch Intern Med 1991; 151:591–593.

We describe a case of severe refractory hypoglycemia secondary to topical salicylate intoxication. A 72-year-old man with psoriasis and end-stage renal disease was treated with a topical cream containing 10% salicylic acid. The patient presented with encephalopathy and subsequently developed hypoglycemia refractory to infusions of large amounts of glucose. A serum salicylate concentration was elevated at 3.2 mmol/L. Emergent hemodialysis was accompanied by rapid lowering of serum salicylate concentration and resolution of refractory hypoglycemia. Salicylate is well absorbed across normal and diseased skin. Salicylate markedly impairs gluconeogenesis and increases glucose utilization, resulting in hypoglycemia. To our knowledge, this is the first article on hypoglycemia due to the application of topical salicylate.

Antecedent medical diseases in patients with amyotrophic lateral sclerosis

Armon C, Kurland LT, O'Brien PC, Mulder DW.
Arch Neurol 1991; 48:283–286.

Odds ratios (ORs) were estimated for the prevalence of antecedent endocrine, metabolic, or vascular diseases among 45 patients with amyotrophic lateral sclerosis from the Rochester, Minnesota, population compared with 90 control subjects matched for sex, year of birth, period of observation, and residence. Hypertension occurred less frequently in male patients with amyotrophic lateral sclerosis (4%) than in control subjects (30%; OR = .10). Because of small population size, no conclusions can be drawn with respect to the following antecedent conditions: thyroid disease (OR = 1.61), coronary artery disease (OR = .58), obesity (OR = .52), diabetes (OR = 1.00), cerebrovascular disease (OR = .21), and peripheral vascular disease (OR = 1.23). The heterogeneity of antecedent thyroid disease makes it highly unlikely that any specific thyroid lesion is causally associated with most cases of amyotrophic lateral sclerosis. Hypertension may be a marker for protective factors against the development of amyotrophic lateral sclerosis in men.

Thoracic myelopathy caused by ossification of the ligamentum flavum clinicopathologic study and surgical treatment

Okada K, Oka S, Tohge K, Ono K, Yonenobu K, Hosoya T.
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The authors reviewed 14 patients with thoracic myelopathy caused by ossification of the ligamentum flavum (OLF). The premdomiant locality of sympotomatic OLF was at the thoracolumbar junction, particularly at T10–T11 followed by T11–T12. At the level of the thickest OLF in each patient, there were three types of OLF from computed tomography and operative findings: a lateral type in three patients, diffuse in eight, and thickened nodular in three. The diagnosis of OLF-related thoracic spinal canal stenosis was best made by enhanced computed tomography. Histologic study revealed that the developmental mode of OLF was mainly endochondral ossification. Numerous fibrocartilaginous cells were found in the increased and swollen collagen fibers forming the hypertrophic ligamentum flavum (HLF). Ossification extended along the superficial layer of HLF. The size or extension of OLF was relevant to the corresponding diathesis of spinalhyperostosis. Results of laminectomy for OLF were proof because of the high occurrence of complications early on or later deterioration. Therefore, laminoplasty is recommended as a successful procedure for OLF-related thoracic myelopathy, avoiding further local mechanical stress due to tensile force.

Central nervous system magnesium deficiency

Langley WF, Mann D. Arch Intern Med 1991; 151:593–596.

The central nervous system concentration of magnesium (Mg^{++}) appears to have a critical level below which neurologic dysfunction occurs. Observations presented suggested that the interchange of the Mg^{++} ion between the cerebrospinal fluid, extracellular fluid, and bone is more rapid and dynamic than is usually believed. This is especially so when the hypertrophied parathyroid gland is associated with significant skeletal depletion of Mg^{++} as judged by history rather than serum level. Magnesium, much like calcium, has a large presence in bone and has a negative feedback relationship with the parathyroid gland. A decline in central nervous system Mg^{++} may occur when the skeletal buffer system orchestrated largely by the parathyroid glands is activated by an increase in serum calcium. Observations in veterinary medicine and obstetrics suggest that the transfer of Mg^{++} from the extracellular fluid into bone during mineralization processes may be extensive. If the inhibition of the hypertrophied parathyroid gland is prolonged and the skeletal depletion of Mg^{++} extreme, serious neurologic symptoms, including seizures, coma, and death, may occur. Noise, excitement, and bodily contact appear to precipitate neurologic symptoms in Mg^{++}-deficient human subjects as it has been documented to occur in Mg^{++}-deficient experimental animals. The similarity of the acute central nervous system demyelinating syndromes with reactive central nervous system Mg^{++} deficiency is reviewed.

Causes and clinical management of vertebral osteomyelitis in Saskatchewan

Joughin E, McDougall C, Parfitt C, Yong-Hing K, Kirkaldy-Willis WH. Spine 1991; 16(3):261–264.

A retrospective review of all patients with vertebral osteomyelitis admitted to all Saskatchewan referral hospitals from 1973 to 1986 was undertaken to determine the incidence and clinical characteristics of the disease. There were 73 patients, an incidence of 5.3 cases/million per year. Erroneous initial diagnoses were common (41%). There was a significantly increased risk in patients older than 60 years. Staphylococcus aureus was the most frequent organism. Mycobacterium tuberculosis was present in 29.5% and was more common in native Indian patients. Surgery was performed in 31% of all patients, and in 50% of those with tuberculous infections. The outcome was excellent in 92% of patients. Diabetes and transurethral resection of the prostate were risk factors for vertebral osteomyelitis.