D-04 PREANALYTICAL PHASE AND INTERFERENCES IN IMMUNOCHEMICAL ANALYSIS

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The total testing process consists of three steps in medical laboratories: preanalytical, analytical and postanalytical phases. Each of these phases makes a different contribution to the total error which is extremely important for the interpretation of laboratory test results and patient safety. In many automated methods, including immunoochemistry analysers, the analytical phase is tightly controlled and the contribution of this phase to the total error is extremely low. Because the preanalytical procedures have not been standardized yet, the contribution of preanalytical errors to the total error is still important. Immunoochemical assays are analytically sensitive. However they may lack sufficient specificity and accuracy. Specifity, depends not only on the antigen binding properties of the antibody used in the assay method but also on the composition of the antigen and the matrix. Specificity can also be influenced by reagent composition and assay format. Substances may potentially cause interference in the measurement and can provoke changes in the measurable concentrations of the analyte or alter the antibody binding properties. In immunoochemical analysis interferences are analyte dependent or analyte independent and can lead to an increase (positive interference) or a decrease (negative interference) in measured analyte concentrations. Hemolysis, lipemia, icterus, anticoagulant effects and sample storage conditions may all cause interferences independently from analyte concentrations. Interactions between sample constituents and one or more antibodies used in the assay method cause analyte dependent interference. These include heterophilic antibodies, human anti-animal antibodies, auto analyte antibodies, romatoid factor and other proteins. Erroneous analyte concentration obtained as a result of interference may have important clinical consequences such as unnecessary further investigations, inappropriate treatments and may threaten patient safety. Therefore, during the interpretation of immunoochemical test results, preanalytical error sources should be known and taken into account by laboratory professionals and procedures should be defined wherever possible to identify them.

D-05 THYROID IN ENERGY BALANCE

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Thyroid hormones are the main players in energy balance. We can clearly see the importance of thyroid hormone in energy use in advanced thyroid insufficiency, myxemia patients. Patients are almost as if they are slow-motion, they are not energized. However, although energy reserves, that is, fat and glycogen are sufficiently present, they can not be converted to ATP. With thyroid hormone treatment, everything returns to normal. Hyperthyroidism is also characterized by excessive energy use and associated clinical and laboratory findings. Despite overfeeding, they lose weight, lose energy, energy bangles become negative. There are two basic regulators of energy balance control for thyroid hormone. It is regulated at the center of the hypothalamus. It's a little slow running system. It assesses thyroid hormone regulation and energy balance by evaluating the whole body’s metabolic needs. External-thyroid function relation is also directed by the hypothalamus. For example, the need to increase heat generation in extreme cold and the increase in thyroid activity in the context of this is a function controlled by the hypothalamus. In the context of thyroid energy balance, the other regulator is all peripheral cells. The thyroid hormones in the cells are weighted energy control. In this control, the energy needs of the cell and the basic approach to how it can be met. Here the cell increases or decreases the activity of thyroid hormone itself according to the energy need. Naturally, the instruments of the cell are deodorizing while this is ensured. It will increase the formation of deiodinase-1 and deiodinase-2 and T3 and 3,5 T2 when it wants to increase energy use, it will bring weight to the resting metabolic pathway by activating the deiodinase-3 pathway if energy demand is reduced or the energy economy wants to do it.In the hypothalamus, the arcuate nucleus works like a basic control unit. Controls other hypothalamic nucleus and hypothalamic areas in energy balance and nutrition. The hypothalamus increases the arcuate nucleus, the paraventricular nucleus, the ventromedial nucleus toughness tonus, as well as the autonomic system of the adrenergic pathway. Leptin from fatty tissue, GLP-1 and PYY secreted from ileum L-cells increase satiety tone by stimulating satiety neurons (proopiomelanocortin neurons), paraventricular neurons and ventromedulary nucleus neurons, respectively, in the arcuate nucleus. The arcuate nucleus and the lateral hypothalamus function mainly in the context of nutrition and energy recovery by another approach. Ghrelin stimulates NPY neurons in the arcuate nucleus, directing the person to feed and suppresses the feeling of satiety. The effect of hypothalamic neurons on fasting and satiety is changing the thyroid secretion and activity. For example, we can see this in obese people or weight gainers. In obesity, in other words in excess of energy, leptin elevation directly increases TRH secretion in the paraventricular nucleus. TRH increase naturally increases serum TSH level and FT3 / FT4 ratio. In another approach, the hypothalamus aims to combat the pathological energy turnover by using the TRH pathway. On the other hand, the exchange of thyroid hormone secretion as a primer (depending on the primary disease of the thyroid gland) can also make changes in the context of energy gain or loss in hypothyroidism which is also an energy balance. T3 elevation reaching the hypothalamus nuclei in hyperthyroids activates the mTOR signaling pathway in the nutrient neurons (NPY neurons) in the arcuate nucleus, increasing NPY and AGRP synthesis and expression and inducing side-to-side feeding through the lateral hypothalamus; while TRH expression is suppressed in the paracentric nucleus on the other hand. In contrast, high T3 suppresses the expression of α-MSH, which is a satiety hormone in the satiety neurons (in the proopiomelanocortin neurons), thereby reducing or eliminating the feeling of satiety. Overeating and eating in hyperthyroid patients is a known fact. Naturally, hypothyroidism is also the opposite of certain measures. As a result, thyroid hormones are an important means of controlling energy balance in the body. These tools are used both by the hypothalamus in energy balance control and by the peripheral cells independently.

D-06 INTRAOPERATIVE BIOMARKERS

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As a biomarker, intraoperative parathormon monitoring (IoPTH) is probably unique method to predict operative success during surgical intervention. The most common indication for parathyroid surgery is primary hyperparathyroidism (PHPT) due to adenoma, hyperplasia, cancer or rare some hereditary conditions. The only definitive cure for PHPT is parathyroidecmy of hyperfunctioning or abnormally enlarged parathyroid glands. The aims of the surgery is to achieve normalcalemia as well as to avoid recurrence and persistance of PHPT. Recent developments in the field of imaging techniques and IoPTH assays resulted in remarkable paradigm shift in surgery. Traditional surgical approach (bilateral neck exploration) mostly moved to focused or minimaly invasive parathyroidectomy. Three important features of parathormone (PTH) make it an ideal biomarker intraoperatively: It has a short half-life of 4-5 minutes. Gland devascularization effects hormon levels immediately. PTH is produced only by the parathyroid glands. Excessive PTH release from hyperfunctioning gland inhibit PTH secretion from the normal pathological glands surusus by using the thyroid hammer. In rapid decelerations, for example, in bariatric surgery, the ratio of TRH secretion and thus serum TSH, FT3 and FT3 / FT4 is decreasing. Here, the hypothalamus aims to make energy economy by reducing thyroid function. On the other hand, the exchange of thyroid hormone secretion as a primer (depending on the primary disease of the thyroid gland) can also make changes in the context of energy gain or loss in hypothyroidism nuclei related to energy balance. T3 elevation reaching the hypothalamus nuclei in hyperthyroids activates the mTOR signaling pathway in the nutrient neurons (NPY neurons) in the arcuate nucleus, increasing NPY and AGRP synthesis and expression and inducing side-to-side feeding through the lateral hypothalamus; while TRH expression is suppressed in the paracentric nucleus on the other hand. In contrast, high T3 suppresses the expression of α-MSH, which is a satiety hormone in the satiety neurons (in the proopiomelanocortin neurons), thereby reducing or eliminating the feeling of satiety. Overeating and eating in hyperthyroid patients is a known fact. Naturally, hypothyroidism is also the opposite of certain measures. As a result, thyroid hormones are an important means of controlling energy balance in the body. These tools are used both by the hypothalamus in energy balance control and by the peripheral cells independently.