**Biochem sheet 22-11**

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**Transaminase :enzyme catalase the reaction between an amino acid and alpha keto acid to synthesis nonessential amino acid**

**\*The body can synthesize nonessential amino acids.**

**-The mechanism to produce nonessential amino acid is transamination**

**\*Oxidative deamination**

**\*Oxidative decarboxylation ( an example of that is the production of histamine from histidine)**

**Glutamate+ alpha keto acid**

**Alpha kg +alpha amino acid (reversible reaction )**

**There are two enzyme :**

**الانزيم بتسمى حسب المتفاعلات او حسب النواتج\***

**1)ALT (according to the product =SGPT)**

**2) AST(according to the product =SGOT)**

**Both of them are found in liver and heart**

**But ALT is more specific in the liver and AST is more specific in heart) used in cardiac markers :AST, Mb ,LDH,IMA,CK-Mb,troponin )**

**MB : the first marker after the damage to cardiac muscles & it leaks to blood.**

**Other enzymes also leak out to the blood such as CPK ,CPKMB and homocysteine.**

**\*ارتفاع الهوموسيستين يدل على انك معرض لتصاب**

**Myocardial infarction**

**\*Homocystiene is a predictable marker for myocardial infarction.**

**\*LDH isoenzyme in heart :LDH1 composed of H4.**

**\* AST and ALT normal limits may record up to 40 international units per liter. Moreover, in myocardial infarction and liver cirrhosis they increase thousands of times.**

**Enzymes cause a problem if they increase 5 or 10 times the upper limit of normal.**

**\*LDH increases as a result of exercising or any physical activity.**

**coenzyme in transaminases :**

**pyridoxal phosphate**

**vitamin B6 phosphate**

**urea cycle : consumption 4 ATP**

**net body use 1 for urea formation**

**arginase enzyme found only in liver , because of that urea synthesis occurs in liver.**

**urea nitrogen sources :**

**(Carbamoyl phosphate واحد من اول تفاعل(**

 **والتاني من ال aspartate**

**Starvation(different from fasting )and severe exercise cause increase urea formation**

**Protein intake, starvation and severe muscular exercise increase urea up to 10-20 times.**

**\*proteins are not excreted in urine.**

**\*nitrogenous products that are excreted in urine are uric acid and urea.**

**Proteins differ from lipids and carbohydrates that they don’t have storage forms.**

**the coenzyme N-acetyl glutamate which activate carbamoyl phosphate come from the proteins so it increase protein intake , increase urea formation.**

**\*Waste product( end product of protein degradation in human )is urea**

**why glutamine is a carrier for ammonia from peripheral tissue?**

**because it neutral (nontoxic ) so it can pass all barriers while ammonia is toxic (to the brain)**

**Ammonia reacts with alpha ketoglutarate which leads to a decrease in alpha ketoglutarate thus resulting in a malfunction in the citric acid cycle.**

* **Hepatic coma origin in liver because of the increase in ammonia concentration**

**\*ammonia has no normal range in individuals, each patient has its own limit.**

**If ammonia concentration doesn’t fall down with time(every 6 hours) then the patient is not responding to the drug given and will remain in coma.**

**Albinisim : deficiency of tyrosinase**

**\*phenylketonuria is a genetic disease that results from a deficiency in phenlyalanine hydroxylase.**

**In the case of phenylketonuria, phenylalanine accumulates and it’s converted into phenyl lactate, acetate and pyruvate which are excreted in urine.**

**\*Alkaptonuria is characterized by brown/dark/black urine due to the auto oxidation of homogenetisic acid.**

**\*maple syrup urine disease is characterized by orange urine and is a result of branched-chain alpha keto acid dehydrogenase complex or (BCKDC)\*deficiency.**

**\*WIKIPEDIA.**