Myopathy In An Alcoholic Patient

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Abstract: Aim :- To describe a patient with elevated creatine kinase , the cause of which is not encountered commonly. The CPK level of a blood sample obtained in the laboratory was found to be very high . When investigated it was found to be of a patient who presented to emergency after taking Alcohol for 5 days without food.The patient was being diagnosed with alcoholic myopathy.Alcoholic myopathy is not well recognized condition and should be considered in such patient presenting with muscle weakness [Kamal S et al NJIRM 2013; 4(1) : 144-146]

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Introduction: : Creatine kinase is the most sensitive indicator of muscle damage . Skeletal muscle has the highest CK content of any tissue , more than three times as much as heart , brain and consequently nearly all CK activities in normal plasma is derived from skeletal muscle.[1]

Human tissues contain three forms of creatine kinase , comprising dimers of the muscle and brain type subunits , M and B. The combinations are CK-MM, CK – MB, & CK – BB. Skeletal muscle contains mostly CK-MM with only a small amount of CK-MB ranging from 0.2 – 15 % of the total enzyme activity.[1]

The serum creatine phosphokinase level in blood may increase in the following conditions [2] : A Inflammatory myositis ± vasculitis.Muscular dystrophy.Motor neuron disease.Alcohol , drugs Trauma , strenuous exercise

The myopathies are neuromuscular disorders in which the primary symptom is muscle weakness due to dysfunction of muscle fiber. Other symptoms include muscle cramps , stiffness and spasm. Myopathies can be inherited (such as muscular dystrophies) or acquired (such as common muscle cramps).

Clinical case:- Here is a case of elevated creatine kinase (EC: 2.7.3.2), the cause of which is not encountered commonly.

A 38 year old male patient presented to the emergency with vomiting in many episodes , with loose motions since two days and the patient had being taking alcohol without food for 5 days before developing the problem. His general condition was very poor , but he was conscious & he did not pass urine since two days. On taking past history the patient was found to a chronic alcoholic.On examination the abdomen was found to be distended and tender . The investigations done in biochemical lab were as follows:

- 5/2/2010 – CPK – 8465 U/L, TOTAL PROTEIN – 5.7g/dl ,ALBUMIN – 2.6g/dl , INORGANIC PHOSPHORUS – 3.0mg/dl
- 6/2/2010 – UREA – 87mg/dl , CREATININE – 4.3 mg/dl
- 7/2/2010 – UREA – 115mg/dl , CREATININE – 3.3mg/dl , CPK – 15180 U/L.
- 8/2/2010 – UREA – 79 mg/dl , CREATININE – 2.7mg/dl, Na+ - 145 mmol/l, K+ 4.0mmol/l.
- 10/2/10 – UREA – 48mg/dl , CREATININE – 2.0mg/dl , Na+- 143 mmol/l , K+ - 3.6 mmol/l .
- 11/2/10 – RBS – 131mg/dl , UREA – 44 mg/dl , CREATININE – 2.4mg/dl , Na+ - 146mmol/l , K+ - 3.4mmol/l, Ca2+ - 5.2mg/dl , CPK – 2060 U/L .
- 12/2/10 – UREA – 40mg/dl, CREATININE – 1.5mg/dl, Na+ - 146mmol/l, K+- 3.8 mmol/l.
- 15/2/10 – CPK – 800U/L
- 20/2/10 – CPK – 139U/L
- 23/2/10 – CPK – 98U/L

Ultra sound examination reveled – fatty liver , ascites & pancreatitis.The patient was referred to surgical department where exploratory laparotomy was done and necrotizing pancreatitis with ascites was found. Patient was shifted to ICU from OT , with a drain in situ , treated conservatively. On improvement of general condition , the patient was shifted from ICU to the surgical ward and

finally with proper advice the patient was discharged on 23/2/10.

Discussion: We know that in normal adult – CPK activity is almost entirely due to CK-MM isoform. Except for neurogenic muscle disease CPK-MM or CK-3 is increased in dystrophies & myopathies. Here the cause of increased CPK-MM (CK-3) is necrotizing polymyopathy (acute rhabdomyolysis) due to alcoholism.

The other causes of acute rhabdomyolysis include [3]– crush injuries, muscle infarction or necrosis, malignant hyperthermia, unaccustomed strenuous exercise, recurrent or paroxysmal myoglobinuria, viral infections or toxin. The effect of excessive alcohol on nervous system , heart and liver is well known. But the skeletal muscular system is also affected by excessive intake of alcohol is not well known.

Alcohol may cause either an acute or a chronic myopathy . The acute syndrome is characterized by painful localized or generalized muscular cramps and sometimes by weakness & tenderness of muscles with subcutaneous edema.[4,5] .

In chronic syndrome [5,6] muscular weakness and sometimes tenderness of proximal muscles occur. The neurological complications of alcoholism and cirrhosis are commonly associated. Biochemical abnormalities are similar to those in acute syndrome but less striking and electromyograph is myopathic.

Perkoff and his colleagues[5] showed that alcoholic patients ,in whom no muscular symptoms were present but from whom a history of muscle cramps was frequently obtained , often had a biochemical findings similar to those found in symptomatic subjects.

E .T. O'Brien & P.Goldstraw [6,7] reported a case of chronic alcoholic presenting with sharp pain in the limbs and swelling of the calf muscles, with difficulty to climb stairs and to rise from horizontal position in association with gastric pain, anorexia , vomiting & weight loss. They diagnosed the case to be alcoholic myopathy especially acute type, where symptoms were attributed to alcoholic neuropathy.
Our case also presented with chronic history of alcohol intake but with acute episodes of loose motion, vomiting , anuria , general condition poor, tender abdomen & muscle cramps.

Sharma A et al [8] also reported a similar case of a middle aged male who developed swelling and weakness in the lower limbs following heavy binge of alcohol. The case also reported of having acute renal failure due to myoglobinuria.

According to them acute alcoholic myopathy is not a well recognized condition and should be considered in any intoxicated patient who presents with muscle tenderness & weakness.

Jose'M Nicolas et al [9] reported histologic myopathy was present in 58% of alcoholics. It was related to life time ethanol consumption and more severe in the presence of protein malnutrition. They concluded that malnutrition is an additional developmental factor in functional and structural muscle damage induced by chronic ethanol consumption.

Preedy VR et al [10] discussed that alcoholic myopathy is characterized by biochemical and morphological lesions within muscle , ranging from impairment of muscle strength and loss of lean tissue to cellular disturbances & altered gene expression. The chronic form of the disease is five times more common than cirrhosis and is characterized by selective atrophy of type 11 (anaerobic) fibres. They said that acetaldehyde has an important role in the etiology of the disease by reducing muscle protein synthesis in vivo and proteolytic activity in vitro. There is formation of acetaldehyde protein adducts within muscle in response to either acute or chronic alcohol exposure and the adducts are located preferentially within the sarcolemmal and sub-sarcolemmal regions.

Conclusion: From the above discussion we conclude that myopathy due to alcohol may be

commoner than is generally realized and a history of alcoholism should be taken when examining a patient with neuromuscular disorder.

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