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Case Report

CASE STUDY ON MUSCULAR DYSTROPHY

Syam Prashanth Pedada[1], Nirosha Pedada[1], Jyotsna Allamsetty[1*], G.V Nagaraju[2].

[1]Department of Pharmacy, Avanthi Institue Of Pharmaceutical Sciences,Tagarapuvalsa, Vizayanagaram,AP,India.

[2]Assiatant Professor,Department of Pharmacy Practice, QIS College of Pharmacy,Ongole, AP,India.

Abstract

29 yrs old patient was presented for the difficulty in walking and muscle weakness with difficulty, particularly in vigorous physical activities. The patient had experienced frequent falls and admitted in hospital given with symptomatic treatment. The patient's creatinine kinase was 2290 Iu/L, liver enzymes also abnormal. The Mechanism through which levels of liver enzymes become abnormal in patients with MD still unknown. Since serum CK is markedly elevated with a breakdown of muscle and is considered a diagnostic marker of muscular dystrophy's. Duchenne muscular dystrophy is an X linked recessive progressive myopathy, with an incidence of about 1 in 3500 male live birthday was described by french neurologist Benjamin in the 1860s. Conservative management, active physiotherapy, genetic counseling, and other supportive therapies hold the key to the successful management of these cases.

Keywords: Pancreatitis, Age, Observational Study, Risk Factors, Previous Abdominal Surgery.

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***Corresponding Author**

Jyotsna Allamsetty

Avanthi Institue Of Pharmaceutical Sciences, Tagarapuvalsa, Vizayanagaram.

Email: jyotsnabittu@gmail.com

INTRODUCTION

Muscular dystrophy refers a group of disorders that involve a progressive loss of muscle mass and consequent loss of strength.The main forms of muscular dystrophy may affect up to 1 in every 5000 males.The most common form is Duchenne muscular dystrophy. Muscular dystrophies are a heterogenous group of inherited myopathies that share similar clinical features and dystrophic changes on muscle biopsies [1]. DMD is an X linked recessive myopathy, with an incidence of about 1in 3500male live birth [2]. It was first described by french neurologist Guillaume Benjamin Amand Duchenne in 1860's. DMD is described as progressively leading to muscle weakness and eventually it's degradation. Inspite of known disease symptoms,the diagnosis of muscular dystrophy continues to be challenging in entire life potentially because unsuspected myopathy in children with hypertransaminasemia can be erroneously attributed to liver disease [3,4]. ALT,AST,ALP and lactic dehydrogenase are components of routine or comprehensive blood panels and collectively demonstrate liver function. Consequently ,in apparently health volunteer, analysis of these liver enzymes is performed more frequently than analysis of creatine kinase (ck), a more specific marker of muscle disease [5].

SYMPTOMS OF DUCHENNE TYPE MUSCULAR DYSTROPHY

Early symptoms :

1.frequent falls
2.difficulty rising from a lying and sitting position.
3.trouble running
4.walking on the toes
5.large muscle calf
6.muscle pain and stiffness.

AS THE TIME GOES ON THE FOLLOWING BECOME MORE LIKELY

1. Unable to walk
2. Limiting of movement of muscles and tendon.
3. Severe breathing problem.
4. The muscles of heart can be weakend leading to heart problems.
5. Risk of pneumonia

CASEREPORT

An 29yrs old male patientwho wasadmitted to Pinnacle star hospital, visakhapatnam, andhrapradesh for difficulty in walking and muscle weakness with difficulty in running and particularly in vigorous physical activities. He also had decreased quality or state of being physically active. His parents haveconsagineous marriage ,his brother was healthy. Patient had a history of frequent falls and admitted to hospital given with symptomatic treatment. The patient's creatininekinase was 2290 IU/L (normal 50-150 IU/L) and his liver enzymes we're abnormal since birth. He was diagnosed as a case of duchenne muscular dystrophy. He was treated with regular physiotherapy,periodical follow up of cardiac and respiratory infections and other protective therapies.

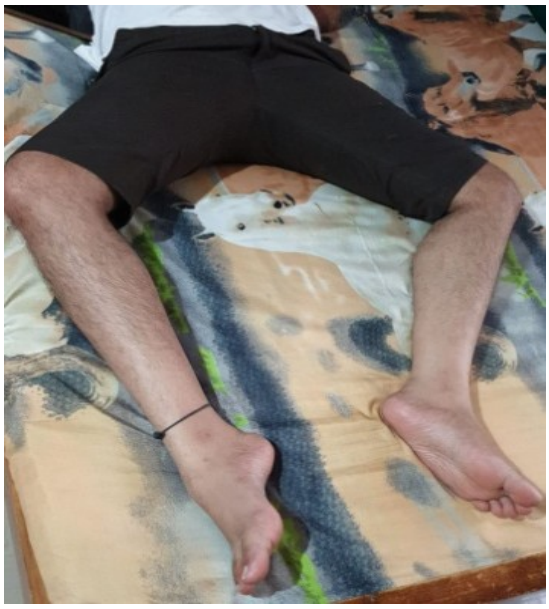

Figure 01:Before Treatement

Figure 02: After Treatment

Table 01:Patient Demogrpahy Levels of Enzymes

Age	29yrs
Sex	Male
Creatine kinase	2,290U/L
SGOT	43 IU/L

DISCUSSION

The mechanism through which levels of ALP and LDH become abnormal in patients with MD is still unknown. Elevation in ALT levels are common indicators of hepatocellular damage. DMD occurs as a result of mutations in the dystrophin gene. Mutation lead to an absence of or defect in protein dystrophin. Dystrophin is structural component in skeletal muscle ,cardiac muscle and brain. It interacts with multimeric protein complex associated with sarcolemma proteins which plays major role to maintain complexity of muscle membarane. AST is found in cardiac muscle,skeletal muscle ,kidneys,brain ,pancreas,lungs,leukocytes, and erythrocytes. Since serum CK is markedly elevated with breakdown of muscle and is considered a diagnostic marker of MDs, we assume that leakage of transaminase from muscle membarane would occur along with leakage of CK under pathological conditions such as in patients with MD. We follow up the patient for 7days, medication chart includes physiotherapy,tab deflazacort ,genetic counselling Was provided to the Patient and Patient Attenders and he is free from symptoms ater 7 Days, he is Discharged from the Hospital.

CONCLUSION

Duchenne muscle dystrophy is a progressive inherited myopathy with an early onset in childhood.It progresses to the bed-bound state in the second decade of life and patients usually prone to respiratory or heart complications. 3. Conservative management,active physiotherapy ,genetic counselling and other supportive therapies hold the key to successful management of the cases.

CONFLICTOFINTEREST

All authors declare that there is no conflict of interest regarding the publication of this paper

ABBREVATIONS

ALT-Alanineaminotransaminase;AST-Asparateanimotransaminase,
SGPT-Serum glutamate pyuruvatetransaminase,
SGOT-Serum glutamate oxalacetatetransaminase,
ALP-Alkalinephosphotase,
LDH-Lactate dehydrogenase,
CK-Creatininekinase,
MD –Muscular dystrophy,
DMD-Duchenne muscular dystrophy

REFERENCES

1. Mercuri E., Muntoni F. Muscular dystrophies. *The Lancet*. 2013;381(9869):845–860. doi: 10.1016/s0140-6736(12)61897-2.
2. Nussbaum R, McInnes RR, Willard HF (2007) Thompson & Thompson genetics in medicine. Elsevier Health Sciences.
3. T. Begum, M. R. Oliver, A. J. Kornberg, and X. Dennett, "Elevated aminotransferase as a presenting finding in a patient with occult muscle disease," Journal of Paediatrics and Child Health, vol. 36, no. 2, pp. 189–190, 2000.
4. E. Bradley, M. M. Aw, and A. Dhawan,"Elevatedtransaminases: of liver or muscle origin?" Journal ofPediatric Gastroenterology and Nutrition, vol. 34, no. 1, p. 93, 2002.
5. A. A. El-Bohy and B. L. Wong, "The diagnosis of muscular dystrophy," Pediatric Annals, vol. 34, no. 7, pp. 525–530, 2005.