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Getting Physical before a Medical Physical and How It Leads to Doctor Becoming Patient: A Curious Case of Transient Raised Transaminases

Peter Cassar[1], Nicholas Delicata[2]

[1]Department of Medicine, Department of Nephrology and General Medicine, Mater Dei Hospital, Msida, Malta
[2]Department of Medicine, Department of Gastroenterology, Mater Dei Hospital, Msida, Malta
Email: peter.cassar@gov.mt

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Abstract

This case report sheds light on a curious case of rhabdomyolysis in an otherwise healthy twenty-eight-year-old male and its direct effect on liver function tests. This case report provides a fresh perspective on the doctor-patient dynamic where a medic becomes a doctor. Attention is also drawn to the mental and socioeconomic repercussions of having a health issue whilst applying for a home loan.

Keywords

Rhabdomyolysis, Transaminase, Creatinine Kinase

1. Introduction

There have been several documented cases of rhabdomyolysis associated with biochemical signs of suspected liver injury. A quick search of the literature available online reveals that reported cases go back as far as the 1980s [1]. Some studies concluded that during the course of rhabdomyolysis, elevated aspartate transaminase (AST) levels can occur in up to 93% of patients [2]. Studies have shown serial serum AST concentrations rising and falling in tandem with creatinine kinase (CK) concentrations [2]-[4]. Alanine transaminase (ALT) elevation on the other hand is significantly rarer (up to 75% of rhabdomyolysis cases). ALT levels are slower to fall and still do not do so in tandem with peak CK. This is also seen in **Figure 1** about this particular case. This may suggest that, in cases of rhabdomyolysis, abnormal aminotransferases may not necessarily prove hepatic injury [2].

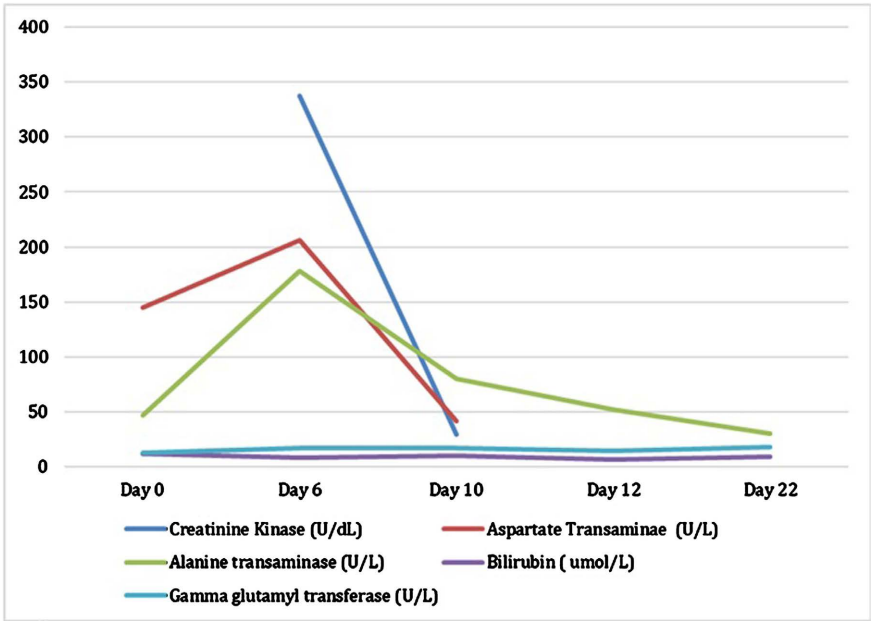

Figure 1. Biochemical progression.

2. Case Presentation

2.1. Patient's Perspective

Leading up to a medical physical for a life insurance policy to secure a home loan, a twenty-eight-year-old male felt motivated to attend the local gym he used to frequent in days gone by. The patient presented himself at the gym one morning and struggled to carry out the same workout routine he once performed effortlessly. Incredibly sore and some days later the patient underwent his physical which included bloodwork. Several days later, the patient was contacted by an insurance physician in view of newly deranged liver function tests. Being a doctor himself the patient suddenly finds himself in a very unique situation. On the one hand, he was suddenly concerned that there might be a potential health scare and on the other hand anxious to get his life insurance policy issued to secure the home loan for himself and his family. Being a medical doctor, a patient's inquisitive mind races to get a correct and timely diagnosis.

2.2. Detailed History and Examination

28-year-old previously well with no past medical history presents to a gastroenterologist with a special interest in the liver. The patient denies any weight loss, change in bowel habits, anorexia, or abdominal pain. Admits to some chest pain on torsional movements however no further symptoms, pain that started a week after exertion at a local gym. Claims to have presented solely in view of g elevated levels of alanine transaminase and aspartate transaminase on recent blood tests.

Previously otherwise healthy. Denies any past medical history. History of sebaceous cyst removal. Denies any allergies and does not take any regular medication. Works as specialist trainee in nephrology and general medicine at local hospital. Denies smoking and social drinking of less than 5 units a week.

On examination, there were no obvious signs of jaundice, ascites, spider naevi, tremor, or any obvious hepatomegaly. Chest and heart sounds were unremarkable.

The patient presented with a blood pressure of 118/70 millimeters of mercury (mmHg), a heart rate of 80 beats per minute, was afebrile and oxygen saturations above 96% on room air.

2.3. Investigations

Was decided to carry out further biochemical as well as immunological and serological tests to rule out further causes of liver dysfunction. Please find investigations tabulated together with their temporal relation for biochemical tests that were repeated over the course of weeks in Table 1.

Table 1. Biochemical investigations.

Biochemical Investigation (range)	07/03/2023	13/03/24	17/03/24	19/03/24	29/03/24
Bilirubin (<17.0 umol/L)	12.4	8.4	10.5	6.4	9.0
Alkaline phosphatase (40 - 129 U/L)	55	64	60	71	63
Gamma glutamyl transferase (8 - 61 U/L)	13	17	17	15	18
Aspartate transaminase (<50 U/L)	145	206	42		
Alanine transaminase (<41 U/L)	47	178	80	52	30
Creatinine Kinase (39 - 308 U/L)		3370	298		
HIV 1 Ag and HV1&2 Total Abs (Negative/Positive)			negative		
Hep screen (positive/negative)					
Antimitochondrial antibodies + Liver kidney microsomal antibody (negative/positive)		negative		Negative Titre (<1/1000)	

2.4. Working Diagnosis, Treatment and Outcome

Given the temporal association between creatine kinase levels and transaminase levels as well as patients history of physical exertion, the gastroenterologist treated patient conservatively as a case of rhadomyolysis induced transient raised trans-

aminases. A Conservative approach was taken to the resolution of rhabdomyolysis.

Patient was encouraged to drink plenty of fluids and avoid strenuous physical activity. Blood tests improved spontaneously with time. Over 4 weeks, transaminitis improved with no need for further interventions, biochemical tests, or imaging. Liver function tests normalized completely with no immediate or long-term sequelae.

2.5. Physician Becomes Patient

A few words from patient in question who also happens to be a medical doctor:

Being both a physician and patient in this particular scenario was a very unique experience. Felt firsthand the helplessness and uncertainty of being told there was something wrong with my bloodwork. Not only did I initially feel worried about newly deranged liver function tests, my clinical thinking kicked in soon to try and come up with a working differential. Felt anxious not only because of a potential health scare but also because of the psychological repercussions of potentially being refused life insurance coverage. Felt embarrassed as to date I was not aware that rhabdomyolysis could lead to a transient elevation in transaminase levels and that was so unfit at the time that just a single session of exerting myself led to such a dramatic biochemical picture. Was appreciative of colleagues who took an objective approach to reaching a diagnosis whilst all the while respecting the particular psychosocial stresses I was being subjected to in the process.

3. Discussion

As suggested in the introduction of this case report, temporal rises in aminotransferases have been wildly documented in cases of rhabdomyolysis. This case study shows the same correlation, with a clearly depicted downward trend in levels of AST and ALT with decreasing levels of creatinine kinase.

ALT and AST are found in varying tissue types including muscle, kidney, and liver in various species [5] [6]. Yang *et al.* [7] assessed the dissemination of ALT1 and ALT2 mRNA in rodents. ALT1 was found to be mainly expressed (from low to high) in the intestines, liver, fat, colon, muscle, and heart. ALT2 mRNA on the other hand is more restricted in distribution, to the brain, white adipose, and muscle.

In the case of the human liver, ALT is expressed to a greater extent when compared to AST. The latter is known to be found in skeletal muscle, red blood cells, brain, kidney, and heart muscle [8]. Lack of data persists as regards the quantitative discrepancies between tissues. Apple and Rogers proposed a ratio of 1:4 as regards the amount of ALT in muscle compared to the liver [9]. Other studies suggest different ratios [8], reflecting differing methods of measurement, leading to suggest that a more detailed analysis may be helpful.

Without a history of muscle injury or disease, one may mistakenly associate

raised aminotransferases with liver injury. Studies have shown that even with known rhabdomyolysis, further serological, molecular, and biochemical tests for liver disease have mostly been negative. Up to half of liver ultrasounds and computed tomography were unremarkable and most deviations from the norm were secondary to hepatic steatosis [10]. As a consequence of over-investigating potential liver disease, there could be an unfortunate failure to investigate or even recognize muscle disease.

A particular 27-year-old man underwent several years of biochemical and radiological tests for deranged aminotransferases, including two liver biopsies, until an elevated CK led to an eventual final diagnosis of muscular dystrophy [11].

There have not been many recent studies delving into the nature of liver biochemical liver dysfunction accompanying rhabdomyolysis.

An extensive study published in 2020, involving 528 patients, confirmed that an isolated rise in serum ALT can occur due to rhabdomyolysis. The highest CK levels, indicating the severity of muscle damage, are the primary factors accounting for the increased ALT. Age, acute kidney injury (AKI), and chronic liver disease had smaller effects, but together these four factors explained half of the variation in ALT levels. Predicting the increase in ALT based on peak CK levels might be feasible [10].

4. Limitations

The case documented here is an individual case. One cannot assume that each case of rhabomyolysis will give a transient rise in transaminase levels.

The cause for raised transaminase may have potentially not been identified through the potential lack of further biochemical or radiological tests that were deemed unnecessary by investigating clinician.

5. Conclusion

This case report joins the many other observational case reports peppering the literature and the limited studies suggesting a strong correlation between rhabdomyolysis and liver function tests. With this particular case and many others with an accompanying clear history of muscle injury and exertion, further serological, molecular, and biochemical tests into potential causes of liver injury are perhaps not only not warranted but may lead to patient anxiety.

6. Research Method, Material and Ethics

Data collection was done by accessing clinical software available at Mater Dei Hospital Malta as well as gaining permission from patients to publish results.

This is a sole case report based on one individual case. The case report was written up in retrospect and all investigations carried out were done in order to reach a working diagnosis, resolution, or amelioration of illness and not for the purposes of any study.

Written informed consent was obtained from the individual(s) for the publi-

cation of their thoughts and any potentially identifiable images or data included in this article.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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