Rare and Dangerous Complication of Rhabdomyolysis in Critically Ill Patients: a Case Study

Lin Shan[1], Meng Fan[2], Zheheng Bao[2*]

[1] Department of Surgical Center, Tianjin First Central Hospital, Tianjin 300192, China

[2] Department of Orthopedic Surgery, Tianjin First Central Hospital, Tianjin 300192, China

DOI: 10.32629/jcmr.v5i1.1780

Abstract: Rhabdomyolysis in critically ill patients is rare and can easily lead to serious adverse complications. We present the case of a young male patient who experienced acute liver failure resulting from closed abdominal injury, accompanied by severe complications of rhabdomyolysis and compartment syndrome. The patient underwent orthotopic liver transplantation surgery to restore liver function. Compartment syndrome of the lower legs was treated with percutaneous deep fasciotomy. Upon discharge, the patient was in a post-liver transplant state and exhibited partial dysfunction in lower limbs. Therefore, rhabdomyolysis occurs insidiously in severe patients and can easily lead to serious adverse outcomes. Clinicians should recognize symptoms of rhabdomyolysis and intervene promptly.

***Keywords*:** acute liver failure, rhabdomyolysis, compartment syndrome

1. Introduction

Rhabdomyolysis(RML) is a condition characterized by the necrosis and breakdown of muscle tissue, resulting in the destruction of muscle cell membranes and the release of cellular contents into the bloodstream[1] . The typical clinical manifestations include myalgia, muscle weakness and dark urine. However, the majority of patients present with atypical clinical symptoms that may result in a missed diagnosis[2] . The causes of RML are diverse, including trauma, substance abuse and infection. Especially in critically ill patients, the occurrence of RML is insidious and fatal. Here we report the therapeutic process of a young male patient with acute traumatic liver failure who suffered from RML and leading to severe compartment syndrome during hospitalization.

2. Case presentation

A 20-year-old male suffered a closed abdominal injury due to a robotic arm impact. Abdominal CT confirmed liver rupture. We promptly performed exploratory laparotomy, removed the damaged liver and performed hemostasis. The patient presented with severe circulatory instability and liver failure after surgery. On the 7th day of hospitalization, the patient suffered from acute hemorrhagic shock. And we performed another exploratory laparotomy and partial hepatectomy. On the 11th day of hospitalization, the patient underwent orthotopic liver transplantation due to liver failure, and the liver function gradually returned to stability.

After liver transplantation, the patient gradually developed acute kidney injury (AKI). On the 4th day after liver transplantation surgery, the patient complained of limited dorsiflexion of both ankle joints and lower limbs edema. Ultrasound showed normal blood perfusion in the lower limbs. On the 10th day after surgery, the patient experienced severe pain in both lower legs, accompanied by squeezing pain. The pressure of the intermuscular compartment of both lower legs increased significantly (Figure 1A). The ultrasonic manifestations of lower limbs musculature were thickening, hypoechoic and blurred muscle texture (Figure 1B). The blood perfusion was still normal. Magnetic resonance imaging(MRI) of the waist and lower limbs showed muscular edema and peri-muscular effusion (Figure 1C & D).

3. Diagnosis

Based on the patient's clinical manifestations and imaging test results, bilateral calf compartment syndrome could be clearly diagnosed. The laboratory results showed the value of creatine kinase (CK) was 1913.9U/L (reference 50-310U/L) and began to multiply on the 7th day of hospitalization. The follow-up CK on the same day was 6437.6 U/L and the value of myoglobin (MYO) >4095 ng/ml (reference value 17.4-105.7 μg/L). On the 9th day of hospitalization, the peak CK was 136387 U/L, about 430 times the normal (Table 1). Therefore, we considered that RML occurred before the patient received the liver transplant and eventually led to the occurrence of secondary compartment syndrome.

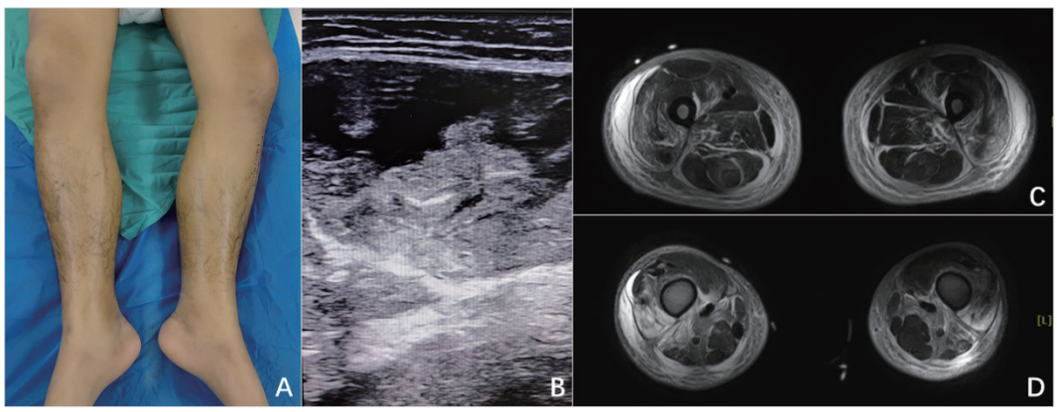

Figure 1. Manifestations of lower limbs. (A. Swelling of the lower legs with high compartment pressure. B. Ultrasound showed structural disorder of the calf muscles. C, D. MRI of the proximal and distal thighs showed musculature edema and perimuscular space effusion.)

Table 1. Laboratory results during hospitalization

Parameter (reference range)	Day 1	Day 4	Day 7		Day 8	Day 9	Day 15	Day 19	Day 20	Day 24
RBC (4.3-5.8*10^9/L)	3.51	7.71	1.37		2.45	1.97	2.90	2.46	2.12	1.24
HGB (130-175 g/L)	109.00	3.73	41.00		72.00	60.00	87.00	75.00	65.00	38.00
PLT (125-350*10^9/L)	65.00	115.00	33.00		10.00	11.00	76.00	128.00	138.00	191.00
Myoglobin (17.4-15.7ng/ml)		137.20	>4126	>4095	>4095	>4126	>4105	2915.5	3170.5	1187.7
Creatine kinase (50-310U/L)			1913.9	6437.6	86582	136387	16873	3311.3	2219.6	1226.7
Creatinine (59-104 umol/L)	91		165	196	160	150	340	522	379	350
Uric acid (208-428 umol/L)	318.4	71	372.9	535.7	190.1	127.6	395.3	332	221	353.4
ALT (9-50U/L)	1312.1	53.6	1813.3	774.34	886.3	794.5	352	126.1	85	362.9
AST (15-40U/L)	1078.5	1002.9	2416.3	2071.3	3199.8	3151.6	630	157.8	104.1	677.9

4. Treatment and outcome

During hospitalization, we closely monitored the changes in laboratory indicators including CK and MYO. Blood purification and plasma exchange were performed intermittently to remove macromolecular substances. We performed four debridement surgeries to decompress and remove necrotic tissue in the lower limbs. We found that necrosis was mainly distributed in the lateral compartment and posterior compartment of the calf. A large amount of necrotic tissue fluid and blood clot-like tissue were released from the intermuscular space. The wound was gradually closed with a staged suture. We carried out rehabilitation on the range of motion of lower limb joints and muscle strength. The symptoms of neuralgia were satisfactorily controlled with analgesic and sedative drugs. In addition, the patient was often complicated with local symptoms such as foot edema, blisters and pressure sores during rehabilitation. The patient is currently able to walk with assistance.

5. Discussion

Currently, the diagnosis of RML is established by clinical symptoms and serum CK levels. The CK rise within 12 hours after muscle injury. It reaches peak within 24 to 72 hours and returns to normal about 5 days [1, 3] . The CK greater than 1000 U/L or at least five times the upper limit of the normal range is generally considered to be diagnostic of RML. Moreover, the CK correlates well with the degree of muscle injury and the risk of compartment syndrome[1] . On the 7th day of hospitalization, this patient experienced a rapid increase in CK and MYO. Although the patient was in shock, we were able to assess the extent of muscle damage and diagnose RML by monitoring the change of CK.

Trauma and medications are the main pathogenic factors of RML in adults[4]. Among drug-induced RML, statins have been generally confirmed to be associated with RML[5]. We reviewed medication prescriptions during hospitalization and found there was no clear association with RML. Therefore, we considered the main reason for the occurrence of RML was ischemia-reperfusion injury of the muscle tissue due to circulatory instability. Of course, electrolyte imbalances and disseminated intravascular coagulation(DIC) aggravated the progression of damage. The muscle tissue is relatively more tolerant to ischemia and hypoxia, but long-term ischemia would still cause irreversible complications such as necrosis and

apoptosis of muscle cells [6].

Compartment syndrome was another serious complication that occured during the course of the disease. The risk factors include the young age and the male gender[7]. Young male with abundant muscle volume tend to generate high pressure in the fixed volume compartment after muscle necrosis, and resulting in a vicious cycle of ischemia- necrosis. The symptoms of compartment syndrome include muscle swelling, numbness, muscle pain and paresthesias.

6. Conclusion

Although RML is a relatively uncommon disease, constant vigilance towards its risk should be maintained. Clinicians with a better comprehensive understanding of RML can help reduce the risk of adverse outcomes.

Acknowledgments

This work was supported by Natural Science Foundation of Tianjin Municipality (No. 20JCYBJC00990).

References

[1] Elsayed EF, Reilly RF. Rhabdomyolysis: a review, with emphasis on the pediatric population. Pediatr Nephrol. 2010;25(1):7-18.
[2] Panconesi R, Widmer J, Carvalho MF, Eden J, Dondossola D, Dutkowski P, et al. Mitochondria and ischemia reperfusion injury. Curr Opin Organ Transplant. 2022;27(5):434-45.
[3] Stahl K, Rastelli E, Schoser B. A systematic review on the definition of rhabdomyolysis. J Neurol. 2020;267(4):877-82.
[4] Gupta A, Thorson P, Penmatsa KR, Gupta P. Rhabdomyolysis: Revisited. Ulster Med J. 2021;90(2):61-9.
[5] Wen Z, Liang Y, Hao Y, Delavan B, Huang R, Mikailov M, et al. Drug-Induced Rhabdomyolysis Atlas (DIRA) for idiosyncratic adverse drug reaction management. Drug Discov Today. 2019;24(1):9-15.
[6] Zhou T, Prather ER, Garrison DE, Zuo L. Interplay between ROS and Antioxidants during Ischemia-Reperfusion Injuries in Cardiac and Skeletal Muscle. Int J Mol Sci. 2018;19(2).
[7] Rodriguez, J, Suneja, N, von Keudell, A, & Zhang, D. Surgical demographics of acute thigh compartment syndrome. Injury. 2022;53(10), 3481-3485.

Author bio

Name: Shan Lin; gender: Male; birth: June 1981; Nationality: Han Nationality; native place: Tianjin; Education: Bachelor degree; professional title: attending physician; Research direction: Bone and joint.

[Corresponding author] Name: Bao Zheheng; gender: male; born: September 1990; ethnic: Han Nationality; native place: Shanxi; Education: Doctor; title: attending physician; research direction: Bone and joint.