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Rhabdomyolysis-induced acute kidney injury in a patient with undifferentiated connective tissue disease

A case report and literature review rhabdomyolysis-induced AKI in a patient with UCTD

Hongxia Mai, MD[a], Yuliang Zhao, MD[a], Stephen Salerno, MS[b], Yi Li, PhD[b], Letian Yang, MD[a], Ping Fu, MD, PhD[a,*]

Abstract

Rationale: Acute kidney injury (AKI) accounts for 8% to 16% of hospital admissions and can quadruple hospital mortality, placing a serious burden on the health economy. Acute kidney injury (AKI) is mainly caused by dehydration, shock, infection, sepsis, heart disease, or as a side-effect of nephrotoxic drugs. About 10% to 60% of patients with rhabdomyolysis develop AKI, and 10% of AKI is attributable to rhabdomyolysis. However, rhabdomyolysis-induced AKI secondary to undifferentiated connective tissue disease (UCTD) has rarely been reported before.

Patient concerns: We report the case of a 50-year-old male of UCTD presented with dark brown urine, swelling and edema of the upper limbs, and decreased urine output.

Diagnosis: The patient was diagnosed with rhabdomyolysis-induced AKI secondary to UCTD.

Interventions: The patient was successfully treated with intravenous methylprednisolone with other supportive treatment.

Outcomes: After 3 days of initiating treatment of medicinal charcoal tablets, sodium bicarbonate and intravenous fluids upon admission, the patient's serum creatinine changed mildly from 145.0 μmol/L to 156.0 μmol/L, but the urinary output increased from 1000 mL/24 h to 2400 mL/24 h, with his creatine kinase (CK) and myoglobin rose from 474 IU/L to 962 IU/L and from 641.5ng/mL to 1599 ng/mL, respectively. We then tried to empirically initiate UCTD therapy by giving corticosteroids. After the administration of the 40 mg of methylprednisolone daily, the serum creatinine level dropped to 97 μmol/L the second day, CK decreased to 85 IU/L within 1 week and myoglobin decreased to 65.05 ng/mL within 10 days. When maintenance dose of 4 mg daily was given, the patient showed no abnormalities in creatinine or CK levels.

Lessons: There have been few reports on the association between rhabdomyolysis-induced AKI and UCTD and its mechanism remains unclear. Clinicians should be aware of UCTD as a possible cause to rhabdomyolysis-induced AKI.

Abbreviations: AKI = acute kidney injury, UCTD = undifferentiated connective tissue disease, CK = creatine kinase, CRRT = continuous renal replacement therapy, CTD = connective tissue diseases, SLE = systemic lupus erythematosus, ANA = anti-nuclear antibody, ANCA = antineutrophil cytoplasmic antibodies.

Keywords: acute kidney injury, rhabdomyolysis, undifferentiated connective tissue disease

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[a] Division of Nephrology, Kidney Research Institute, West China Hospital of Sichuan University, Chengdu, China, [b] Department of Biostatistics, University of Michigan, Ann Arbor, MI.

[*] Correspondence: Ping Fu, Division of Nephrology, Kidney Research Institute, West China Hospital, Sichuan University, No. 37, Guoxue Alley, Chengdu, 610041, Sichuan Province, China (e-mail: fupinghx@scu.edu.cn).

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1. Introduction

Acute kidney injury (AKI) is a life-threatening complication of rhabdomyolysis. A recent Chinese epidemiological survey found that the overall incidence of acute kidney injury (AKI) is 11.6%, with in-hospital mortality of AKI being 8.8%.[1] Risk factors that can lead to rhabdomyolysis and future AKI include trauma, severe muscle strain, exposure to extreme temperatures, dehydration, infection, autoimmune diseases, and genetic disorders.[2,3] Trauma and crush injury that result from natural disasters such as earthquakes can cause rhabdomyolysis and AKI.[4] Rhabdomyolysis is characterized by acute and extensive destruction of rhabdomyocytes in lesion areas and muscle fiber necrosis accompanied by scattered muscle fiber regeneration. This is followed by the release of muscle cell contents, including intracellular metabolites (potassium, phosphate, and urate) and intracellular proteins (aldolase, myoglobin, creatine kinase [CK], lactate dehydrogenase, and aspartate transaminase), into the blood.[5] Myoglobin is the major nephrotoxin in rhabdomyolysis and can cause renal tubule obstruction, oxidant damage, and vascular contraction, which can lead to AKI. Myoglobin is easily filtered by glomeruli in the kidney and precipitated in the renal tubules, especially in acidic environments where it is more likely to precipitate in the renal tubules by interacting with the Tamm-Horsfall protein. Oxidative stress injury is caused by the dissociation of iron in myoglobin, which results in free radical release and oxidative injury of the renal parenchyma. Other studies have shown that myoglobin induces lipid peroxidation, which produces isoprostanes, leading to renal arteriolar dysfunction and hypoperfusion. Of course, while myoglobin is a key factor in rhabdomyolysis onset, but other factors such as low blood volume, secondary injuries, metabolic abnormalities, and drug effects are also important causes.[6] The clinical presentation of rhabdomyolysis varies,[7] and while current diagnostic criteria are lacking, the rapid elevation of serum creatine kinase levels is widely recognized as an indicator of rhabdomyolysis.[3] Treatment may include volume resuscitation, urine alkalization and diuresis, maintaining homeostasis, and renal replacement therapy when necessary.[8] Early and active volume resuscitation can restore renal perfusion and increase urine flow to dilute nephrotoxin and promote the excretion of myoglobin and toxic products. Urine alkalization (pH >6.5) can hinder the formation of an acidic environment and reduce the precipitation of myoglobin in the renal tubules, however, there is insufficient clinical evidence for its efficacy in treating rhabdomyolysis. Diuresis is also a common therapy for rhabdomyolysis, as it can increase urine volume and reduce renal tubule obstruction.[6] Although continuous renal replacement therapy (CRRT) has been reported to be beneficial for rhabdomyolysis, there is not enough evidence to support that CRRT for patients with rhabdomyolysis can effectively prevent secondary AKI.[9] In order to prevent hyperkalemia, metabolic acidosis, metabolic alkalosis, and hypervolemia, it is important to monitor electrolyte balance and homeostasis during therapy for rhabdomyolysis. It has also been reported that rhabdomyolysis is associated with connective tissue diseases (CTD), such as polymyositis,[10–15] dermatomyositis,[16–20] systemic lupus erythematosus (SLE),[8,21–23] adult still disease,[24] and vasculitis.[25,26] This report details the development and treatment of a case of rhabdomyolysis-induced AKI in the setting of an undifferentiated connective tissue disease (UCTD) relapse. We speculate that UCTD may be a potential cause of rhabdomyolysis. The patient has provided informed consent for publication of this case.

2. Case presentation

A 50-year-old male was admitted to our department for oliguria, dark brown urine, and swelling and edema of the upper limbs for 1 day. The patient had a history of interphalangeal joint pain of the hands and a positive cytoplasmic type of anti-nuclear antibody (ANA) with high titer (1:10000) for the past 2 years. In this case, he had no exertional causes of over-exercise, status epilepticus, hyperkinetic syndrome, or severe dystonia, as well as no history of non-exertional causes of alcohol/drug abuse, medication, toxic agents, infection, temperature extremes, and muscle ischemia, or trauma leading to rhabdomyolysis.[27] Laboratory tests clearly showed a rise in creatinine levels from 59 μmol/L, observed a few weeks ago, to 182.0 umol/L at admission. We also observed a rapid decrease in the amount of urine output over the course of 2 days, leading to the diagnosis of AKI. In addition, increased CK (962 IU/L) and myoglobin (1599 ng/mL) indicated possible rhabdomyolysis. An ultrasound showed that the direction of muscle fibers in the lesion area was disordered and the striated muscle texture was blurred, appearing cloudy.[28] As an effective means of diagnosing rhabdomyolysis, further ultrasound examination revealed edema in subcutaneous tissue on upper and lower limbs, and the muscle layer of the limbs was disordered and appeared glassy. Electromyography revealed myogenic injury of the upper arms, which strengthened the evidence of rhabdomyolysis diagnosis. In addition, the patient developed a cough and fever at the time of admission. Computed tomography (CT) showed signs of pulmonary infection, and sputum culture confirmed a diagnosis of *Klebsiella pneumoniae*.

The patient was given medicinal carbon tablets, piperacillin-tazobatan, sodium bicarbonate, intravenous saline infusion, and furosemide diuretic treatment after admission. After 3 days of initial treatment, the serum creatinine levels decreased from 182.0 μmol/L to 156.0 μmol/L, while CK and myoglobin rose from 474 IU/L to a peak of 962 IU/L and from 641.50 ng/mL to a peak of 1599 ng/mL, respectively. In addition, serological examination was positive for a cytoplasmic type of ANA with high titer (1:10000), specific to SLE, but negative for other types of autoimmune antibodies such as rheumatoid factor, anti-SCL70, anti-Jo-1, anti-RNP, anti-SS-A, anti-SS-B, c-antineutrophil cytoplasmic antibodies (ANCA), and p-ANCA. These results suggested a diagnosis of UCTD, however, there is currently no generally accepted diagnostic criteria for UCTD. The clinical features of UCTD manifest like systemic autoimmune diseases such as arthralgias, arthritis, Raynaud's phenomenon, leukopenia, anemia, xerostomia, or xerophthalmia, but about 90% of UCTD patients are ANA positive. Positive expression of other autoimmune antibodies such as anti-SCL70 (skin sclerosis, pulmonary fibrosis, and tumors), anti-Jo-1 (dermatomyositis specific antibodies), anti-SS-A (Sjogren's syndrome, SLE, and rheumatoid arthritis), anti-SS-B (Sjogren's syndrome and SLE), c-ANCA, and p-ANCA (vasculitis) is rare, and is not up to current CTD diagnostic criteria. However, UCTD may evolve into CTD, where SLE is more common.[29]

This case suggests that UCTD might be a notable addition the list of causes of rhabdomyolysis and rhabdomyolysis-induced AKI. A causal association between UCTD and rhabdomyolysis was unclear because muscle biopsy was rejected by the patient, and so we empirically initiated UCTD therapy to achieve AKI and rhabdomyolysis remission after conventional therapies for AKI and rhabdomyolysis failed. An intravenous methylprednisolone infusion (40 mg, once a day) was administered on the fourth day of

admission, along with the aforementioned conventional therapy. The patient's indicators of AKI and rhabdomyolysis decreased notably. His creatinine levels dropped to 97 μmol/L within 1 day, CK levels dropped to 85 IU/L within 1 week, and myoglobin levels dropped to 65.05 ng/mL within 10 days and showed no increasing trend thereafter (Figs. 1 and 2). After 12 days of methylprednisolone administration, the patient was discharged and followed regularly by department of Rheumatology. After 6 weeks of oral methylprednisolone therapy, the methylprednisolone dose was gradually reduced to 16 mg, once a day, to 12 mg 1 month later, and again to 10 mg the following month. The maintenance therapy of oral methylprednisolone (10 mg, once a day) was reduced to 8 mg after 5 months, and to 6 mg after 1 month. Currently, maintenance treatment of methylprednisolone (4 mg, once a day) is given after 2 months for the methylprednisolone of 6 mg. During methylprednisolone reduction, the patient was reviewed monthly and showed no abnormalities in creatinine or CK levels.

3. Discussion

The presented case illustrated an unusual presentation of rhabdomyolysis-induced AKI secondary to UCTD. The patient featured rhabdomyolysis and AKI, and had a history of UCTD for two years. However, a definitive initial diagnosis was delayed because of the unclear cause of rhabdomyolysis. The concept of UCTD was developed from the basis of undifferentiated connective tissue syndrome and postulated mainly by LeRoy in 1980 when he put forward the question of whether certain patients experienced UCTD, rather than definite connective tissue disease.[30] However, there is currently no generally accepted diagnostic classification and criteria for UCTD. The clinical features of UCTD share the same manifestations as systemic autoimmune diseases, but the autoimmune spectrum is mainly only positive for ANA, not up to any of the connective tissue disease diagnostic criteria.[29] This case suggests that UCTD might be added to the list of causes of rhabdomyolysis and rhabdomyolysis-induced AKI.

Rhabdomyolysis was first reported in Germany in 1881 and was described in greater detail after the battle of London during the Second World War.[27] Rhabdomyolysis is primarily caused by acute muscle injury and results in muscle pain, weakness, tightness, and edema from the release of muscular contents into the bloodstream.[31] This may lead to a series of physiological pathologies, which are characterized by the increasing of serum

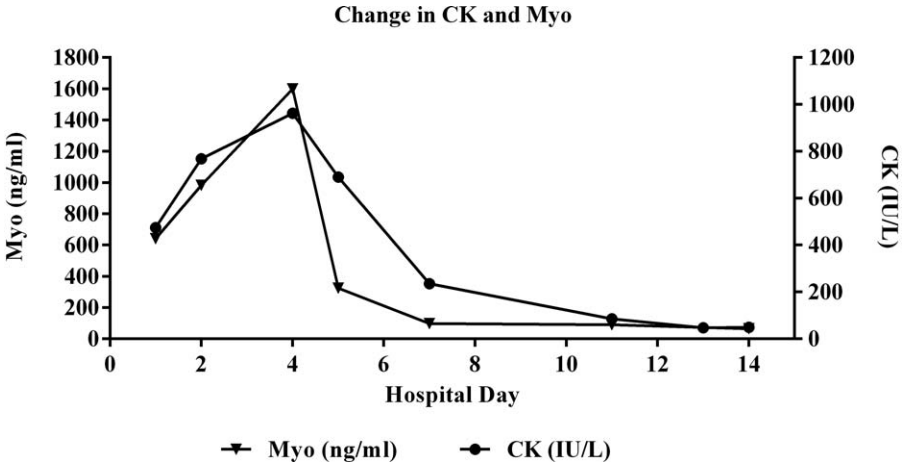

Figure 1. Trends in CK and Myo levels for the patient over time. CK = creatine kinase, Myo = myoglobin.

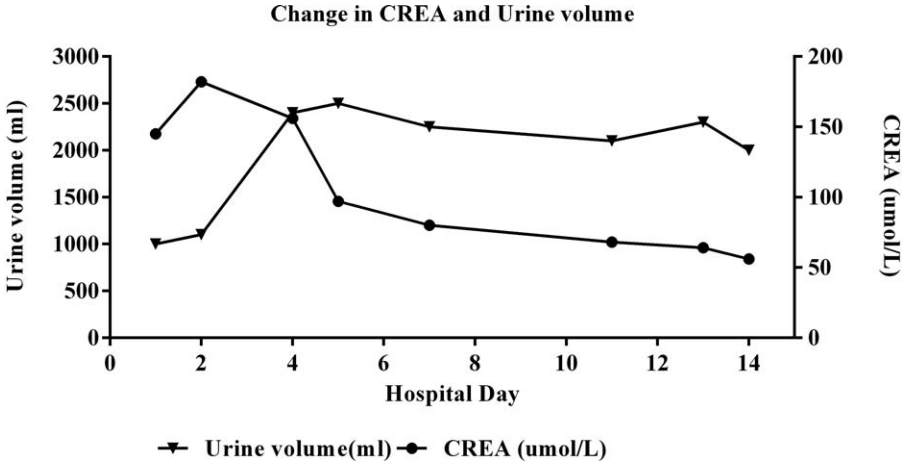

Figure 2. Trends in urine volume and serum CREA for the patient over time. CREA = creatinine.

Table 1

Patients with CTD combined with rhabdomyolysis.

No.	CTD	Reference	Year	Age (yr)	Sex	Treatment	Outcome
1	dermatomyositis	Caccamo et al[16]	1993	25	F	prednisone, methotrexate and plasmapheresis	remission
2	vasculitis	Berlit et al[26]	1993	31	F	Corticosteroids and chloroquine	remission
3	dermatomyositis	Fukunaga et al[18]	2002	31	M	IVIG and plasma exchange	remission
4	polymyositis	Tsai et al[10]	2004	40	F	hemodialysis, corticosteroid, dintravenous albumin and furosemide, intravenous fluid supplement, and alkalization	remission
5	polymyositis	Kim et al[11]	2005	57	F	prednisone, intravenous hydration	remission
6	dermatomyositis	Joshi et al[19]	2009	20	F	methylprednisolone, symptomatic dialysis, and alkalinization and hydration treatment	remission
7	SLE	de Carvalho et al[22]	2011	36	F	hyperhydration and alkalinization	death
8	polymyositis	Naha et al[12]	2012	35	M	prednisolone, rifampicin, and doxycycline	remission
9	SLE	Verdolin et al[23]	2014	39	F	methylprednisolone, alkalization and hydration, chloroquine, prednisone and azathioprine	remission
10	polymyositis	Farooq et al[13]	2015	22	F	steroids	remission
11	polymyositis	Bilen et al[14]	2016	73	M	IVIG and plasmapheresis	remission
12	polymyositis	Pinto-Lopes et al[15]	2017	51	M	prednisolone, azathioprine, methotrexate, mycophenolate mofetil, tacrolimus, and IVIG	remission
13	SLE	Nguyen et al[24]	2018	36	F	methylprednisolone and hemodialysis	remission
14	vasculitis	Iida et al[27]	2018	71	M	prednisolone, cyclophosphamide, and hydration	remission

F = female, M = male, IVIG = intravenous immunoglobulin.

creatine kinase, myoglobin, uric acid, potassium, and phosphorus, influencing the stability of internal environment, and leading to AKI.[32,33] Many symptoms of rhabdomyolysis are nonspecific, including myalgia, swelling, weakness, and so on. Severe cases have had diffuse intravascular coagulation, liver damage, respiratory distress syndrome, circulatory collapse, multiple organ dysfunction, coma, or even death. The most important complication is AKI, which is mainly caused by both obstruction and toxicity to the renal tubules by myoglobin. The occurrence rate of which varies from 14% to 46%.[27] Despite there being many pathogenic factors of rhabdomyolysis, autoimmune diseases are an uncommon cause. Since symptoms of autoimmune diseases such as muscle pain, muscle weakness, and muscle enzyme elevation all resemble rhabdomyolysis, which is prone to misdiagnosis, it is necessary to screen autoantibodies series for CTD or UCTD in rhabdomyolysis patients presenting with significantly elevated serum creatinine, CK, and myoglobin, so as to undergo a timely, positive, and effective treatment.

Although a few cases of rhabdomyolysis-induced AKI with autoimmune diseases have been reported (Table 1), the mechanism is still largely unknown. To the best of our knowledge, only a few of cases of rhabdomyolysis secondary to CTD have been described in literature. In 2005, Kim et al[11] reported the case of a 57-year-old female with polymyositis presenting with rhabdomyolysis, myoglobinuria, and AKI. She was stabilized with intravenous prednisone, volume resuscitation, and other supportive treatment. In 2018, Daniel et al[23] described the case of a 36-year-old female with an SLE exacerbation, as well as concurrent rhabdomyolysis-induced AKI with massively elevated creatine phosphokinase (CPK) (304,700U/L) and massive myoglobin stain in the tubular casts on renal biopsy specimen. Her laboratory and clinical picture improved after hemodialysis and corticosteroid treatment. In this case, after the diagnosis of UCTD, AKI and rhabdomyolysis were equally relieved with methylprednisolone treatment.

Our current patient was diagnosed, not only with rhabdomyolysis-induced AKI and UCTD, but also a severe pulmonary infection caused by *K pneumoniae*. Two previous studies have indicated that infectious rhabdomyolysis accounts for 5% to 31% of all cases.[34,35] We postulate that such infection may be a trigger for rhabdomyolysis. In a case report and literature review on a rhabdomyolysis patient with *K pneumoniae*, the authors reviewed 77 reported cases of bacterial rhabdomyolysis and found that the most common organismal causes were Legionella spp., Streptococcus spp., and Salmonella spp.[36] Rhabdomyolysis caused by primary infection occurs mainly in the respiratory system. Compared to noninfectious rhabdomyolysis, cases of infectious rhabdomyolysis typically have lower CK levels.[36] While there is no proven mechanism for the association between rhabdomyolysis and infection, some hypotheses have been proposed. It is often the case that muscle biopsies show inflammation or lymphocytic infiltration to severe muscle necrosis. Muscle virulence factors, especially tumor necrosis factor (TNF) in respond to virus infection, may be a cause of muscle damage.[22] In the present case, rhabdomyolysis and AKI both occurred before the onset of pneumonia, which was thus unlikely to be the cause. Early diagnosis and treatment are necessary to minimize kidney damage and protect renal function, but in the event of acute renal failure or hyperkalemia, hemodialysis should be given if indicated.[11] This case we report provides a possible cause and effective treatment for rhabdomyolysis-induced AKI. Further studies are warranted to shed light on the pathophysiology of rhabdomyolysis-induced AKI in the setting of a UCTD relapse.

4. Conclusions

Our case suggests that there might be an association between rhabdomyolysis-induced AKI and UCTD, which is rarely reported. Treatment mainly consists of volume resuscitation, urine alkalization and diuresis, and hemodialysis when necessary for rhabdomyolysis-induced AKI and corticosteroid treatment for UCTD.[33] Early and accurate diagnosis and management of the primary disease, UCTD, is of vital importance for halting AKI progression. Clinicians should be aware of possible underlying connective tissue disease when treating patients with rhabdomyolysis-induced AKI. We believe this warrants screening of the autoimmune antibody spectrum as a clinical routine for such patients.

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Author contributions

Conceptualization: Ping Fu.
Investigation: Hongxia Mai, Yuliang Zhao.
Methodology: Hongxia Mai, Yuliang Zhao.
Writing – original draft: Hongxia Mai, Yuliang Zhao, Stephen Salerno, Yi Li, Letian Yang.
Writing – review & editing: Ping Fu, Yuliang Zhao, Stephen Salerno, Yi Li.

References

[1] Xu X, Nie S, Liu Z, et al. Epidemiology and clinical correlates of AKI in Chinese hospitalized adults[J]. Clin J Am Socie Nephrol 2015;10:1510–8.
[2] John F, Oluronbi R, Pitchumoni CS. Levofloxacin-induced rhabdomyolysis: a case report[J]. J Med Case Rep 2016;10:235.
[3] Nance JR, Mammen AL. Diagnostic evaluation of rhabdomyolysis. Muscle Nerve 2015;51:793–810.
[4] Safari S, Yousefifard M, Hashemi B, et al. The value of serum creatine kinase in predicting the risk of rhabdomyolysis-induced acute kidney injury: a systematic review and meta-analysis. Clin Exp Nephrol 2016;20:153–61.
[5] Guis S, Mattei JP, Cozzone PJ, et al. Pathophysiology and clinical presentations of rhabdomyolysis. Jt Bone Spine 2005;72:382–91.
[6] Zimmerman JL, Shen MC. Rhabdomyolysis. Chest 2013;144:1058–65.
[7] Gangadhara S, Gangadhara S, Gandhy C, et al. Rhabdomyolysis and autoimmune variant stiff-person syndrome[J]. Clin Pract 2016;6:
[8] de Carvalho JF, da Mota LMH, Bonfa E. Fatal rhabdomyolysis in systemic lupus erythematosus. Rheumatol Int 2011;31:1243–5.
[9] Zeng X, Zhang L, Wu T, et al. Continuous renal replacement therapy (CRRT) for rhabdomyolysis[J]. Cochrane Database Sys Rev 2014;6:
[10] Tsai CN, Liu MF, Lin TS, et al. Rhabdomyolysis and acute renal failure in a polymyositis patient. Mod Rheumatol 2004;14:422–3.
[11] Kim HW, Choi JR, Jang SJ, et al. Recurrent rhabdomyolysis and myoglobinuric acute renal failure in a patient with polymyositis. Nephrol Dialysis Transpl 2005;20:2255–8.
[12] Naha K, Karanth S, Dasari S, et al. Polymyositis–like syndrome with rhabdomyolysis in association with brucellosis. Asian Pacific J Tropical Med 2012;5:755–6.
[13] Farooq A, Choksi V, Chu A, et al. Severe rhabdomyolysis without systemic involvement: a rare case of idiopathic eosinophilic polymyositis [J]. Case Rep Rheumatol 2015;2015:
[14] Bilen MA, Subudhi SK, Gao J, et al. Acute rhabdomyolysis with severe polymyositis following ipilimumab-nivolumab treatment in a cancer patient with elevated anti-striated muscle antibody[J]. J Immunother Cancer 2016;4:36.
[15] Pinto-Lopes P, Carneiro-Leão L, Morais R, et al. Acute heart failure and rhabdomyolysis: a clue for the diagnosis of polymyositis with cardiac involvement. Reumatismo 2017;69:78–83.
[16] Caccamo DV, Keene CY, Durham J, et al. Fulminant rhabdomyolysis in a patient with dermatomyositis. Neurology 1993;43:844–1844.
[17] Kofteridis PI, Malliotakis F, Sotsiou NK, et al. Acute onset of dermatomyositis presenting in pregnancy with rhabdomyolysis and fetal loss: case report. Scandinavian J Rheumatoly 1999;28:192–4.
[18] Fukunaga E, Kunishige M, Mitsui T, et al. Severe dermatomyositis with rhabdomyolysis and paralytic ileus: a case successfully treated with plasmapheresis and intravenous immunoglobulin. Eur J Neurol 2002;9:697–8.
[19] Joshi D, Kumar N, Rai A. Dermatomyositis presenting with rhabdomyolysis and acute renal failure; an uncommon manifestation[J]. Annals Indian Academy Neurology 2009;12:45.
[20] Mizoguchi F, Takada K, Ishikawa K, et al. A case of dermatomyositis with rhabdomyolysis, rescued by intravenous immunoglobulin. Mod Rheumatol 2015;25:646–8.
[21] Schubert J, Manschwetus H, Walter GF, et al. Systemic lupus erythematosus (SLE) with panniculitis and rhabdomyolysis. Immunitat Infektion 1991;19:58–9.
[22] Verdolin LD, Borner AR, Mussi H, et al. Rhabdomyolysis associated with dengue fever in a lupic patient. Revista Brasileira Reumatol (Engl Ed) 2014;54:318–21.
[23] Nguyen D, Alsaati F, Deitrick J, et al. Rhabdomyolysis secondary to systemic lupus erythematosus[J]. Autoimmunity Highlights 2018;9:5.
[24] Samuels AJ, Berney SN, Tourtellotte CD, et al. Coexistence of adult onset Still's disease and polymyositis with rhabdomyolysis successfully treated with methotrexate and corticosteroids. J Rheumatol 1989;16:685–7.
[25] Berlit P, Härle M, Buss J, et al. Rhabdomyolysis in vasculitis[M]//ANCA-Associated Vasculitides. Springer, Boston, MA, 1993: 427-430.
[26] Iida H, Hanaoka H, Asari Y, et al. Rhabdomyolysis in a patient with polyarteritis nodosa. Intern Med 2018;57:101–6.
[27] Zutt R, Van der Kooi AJ, Linthorst GE, et al. Rhabdomyolysis: review of the literature. Neuromuscul Disord 2014;24:651–9.
[28] Carrillo-Esper R, Galván-Talamantes Y, Meza-Ayala CM, et al. Manifestaciones ultrasonográficas en rabdomiólisis. Cirugía y Cirujanos 2016;84:518–22.
[29] Mosca M, Tani C, Neri C, et al. Undifferentiated connective tissue diseases (UCTD). Autoimmunity Rev 2006;6:1–4.
[30] Mosca M, Tani C, Talarico R, et al. Undifferentiated connective tissue diseases (UCTD): simplified systemic autoimmune diseases. Autoimmunity Rev 2011;10:256–8.
[31] Aguiar DT, Monteiro C, Coutinho P. Recurrent rhabdomyolysis secondary to hyponatremia in a patient with primary psychogenic polydipsia. Revista Brasileira Terapia Intensiva 2015;27:77–81.
[32] Childs SG. Rhabdomyolysis. Orthopaed Nurs 2005;24:443–7.
[33] Criddle LM. Rhabdomyolysis pathophysiology, recognition, and management. Crit Care Nurse 2003;23:14–30.
[34] Blanco JR, Zabalza M, Salcedo J, et al. Rhabdomyolysis of infectious and noninfectious causes. South Med J 2002;95:542–4.
[35] Byrd RPJr, Roy TM. Rhabdomyolysis of infectious and noninfectious causes. South Med J 2002;95:1356–7.
[36] Deng L, Jia R, Li W, et al. A Klebsiella pneumoniae liver abscess presenting with myasthenia and tea-colored urine: A case report and review of 77 cases of bacterial rhabdomyolysis[J]. Medicine 2017;96: