A case report of systemic lupus erythematosus coinciding with rhabdomyolysis: a dramatic recovery via therapeutic plasma exchange

Seda Altiner^, Alper Ekinci^

Department of Immunology and Allergy, Ankara University Faculty of Medicine, Ankara, Turkiye

Contributions: (I) Conception and design: S Altiner; (II) Administrative support: None; (III) Provision of study materials or patients: S Altiner; (IV) Collection and assembly of data: Both authors; (V) Data analysis and interpretation: Both authors; (VI) Manuscript writing: Both authors; (VII) Final approval of manuscript: Both authors.

Correspondence to: Seda Altiner, MD. Assistant Professor, Division of Immunology and Allergy, Ankara University Faculty of Medicine, Hacettepe Mahallesi, Talatpasa Bulvarı, A. Adnan Saygun Caddesi No. 35, 06230 Altındağ, Ankara, Turkiye. Email: altiners@ankara.edu.tr.

Background: In cases of systemic lupus erythematosus (SLE) neurological, cardiac, and renal involvement are among the most frequently encountered clinical challenges that may ultimately be life-threatening. Rhabdomyolysis is a rare complication reported on a case-by-case basis in SLE. Therapeutic plasma exchange (TPE) is being utilized increasingly to treat SLE, with encouraging results. In contrast, data on the use of TPE in the treatment of rhabdomyolysis is scarce.

Case Description: Herein, we report a case of a 23-year-old woman with a history of SLE experiencing complications involving her heart and kidneys. Despite previous treatments, she discontinued medications due to psychosocial issues. Upon admission, she exhibited symptoms such as shortness of breath and edema. Laboratory tests revealed organ damage and urinary Escherichia coli infection, while imagings showed lung and heart abnormalities. Treatment included steroids, antibiotics, and oxygen therapy. Rhabdomyolysis was diagnosed and treated with TPE. Her condition improved over 2 weeks, and she was discharged with a comprehensive medical regimen, including medications for lupus management and psychological support. SLE complicated by rhabdomyolysis, a rare and potentially deadly condition. The patient had been successfully treated using TPE, leading to recovery.

Conclusions: Rhabdomyolysis accompanying SLE is an uncommon situation that raises the risk of morbidity and mortality. TPE is an effective and safe treatment option in SLE where there is an inadequate response to the classical treatments, where the side effects of treatment agents were hazardous, or where treatment agents are contraindicated.

Keywords: Case report; rhabdomyolysis; systemic lupus erythematosus (SLE); therapeutic plasma exchange (TPE)

Received: 28 December 2023; Accepted: 02 July 2024; Published online: 13 August 2024.
doi: 10.21037/amj-23-255
View this article at: https://dx.doi.org/10.21037/amj-23-255

Introduction

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease whose etiology is not thoroughly illuminated and is mostly seen in young women (1). Cells of both the innate and adaptive immune system that are reactive to various self-antigens, especially double stranded deoxyribonucleic acid (dsDNA), play a role in disease pathogenesis (1). Histopathological observations in many tissues indicate the presence of immune complex

^ ORCID: Seda Altiner, 0000-0001-5648-4284; Alper Ekinci, 0000-0002-6269-5990.

© AME Medical Journal. All rights reserved.

accumulation and vasculitis (1).

Multi-system involvement is a hallmark of SLE. During the disease, involvement of the kidney, heart and nervous system can be observed (1). SLE nephritis is a spectrum and may be presented with asymptomatic proteinuria, hematuria, nephrotic syndrome, nephritic syndrome, or end-stage renal failure (1). Although SLE most commonly causes pericarditis of the heart, it can also present with myocarditis, Libman-Sacks endocarditis, atherosclerosis, arrhythmia, and congestive heart failure (1). Headache, epileptic seizures, aseptic meningitis, demyelinating illness, ischemic stroke, peripheral neuropathy, depression, and psychosis are among the neuropsychiatric symptoms linked to SLE (1).

Rhabdomyolysis, which is rarely observed in SLE, is a clinical condition characterized by necrosis of skeletal muscle cells and the entry of necrotic cell content into the bloodstream (2,3). A variety of factors predisposing to rhabdomyolysis have been suggested. Infections, autoimmune diseases, metabolic disorders, drug- or toxin-related conditions, as well as ischemic and physical damage, are well-established acquired conditions that result in rhabdomyolysis (2). As a consequence, a number of metabolic complications, such as renal failure, severe electrolyte imbalance, hypovolemia, or hypercoagulation might take place (2).

Therapeutic plasma exchange (TPE) is an extracorporeal treatment method that removes pathogenic components, including autoantibodies, immune complexes, complement components, and cytokines, from the circulation (4). Studies have indicated that TPE may serve as an adjunctive therapy to treat SLE, resulting in positive outcomes (5). TPE can effectively treat SLE by removing harmful molecules, reducing organ damage, and decreasing mortality and morbidity (5).

On the other hand, data on the use of TPE for the treatment of rhabdomyolysis are limited and contradictory (6,7). Rhabdomyolysis is not stated as a clear indication for TPE in current guidelines (8). In a recent retrospective study, the results of TPE treatment in the rhabdomyolysis cases followed-up in the intensive care unit were analyzed, and the mortality rates did not differ significantly between 19 cases in which TPE was applied and 47 cases in which TPE was not applied (6). However, in the same study, it was determined that long-term renal failure did not develop in any of the rhabdomyolysis cases treated with TPE (6). Furthermore, there are data indicating that TPE can improve clinical findings and prevent possible complications in rhabdomyolysis (7,9-11). TPE may provide benefit in rhabdomyolysis by removing toxic products including myoglobin, as well as by removing substances like drugs, toxins, and inflammatory cytokines that may cause or aggravate rhabdomyolysis (7,9-11).

Here we report a severe case of SLE diagnosed with renal, cardiac, and neurological involvement and also developed rhabdomyolysis during follow-up, which we treated timely and effectively with TPE. We present this case in accordance with the CARE reporting checklist (available at https://amj.amegroups.com/article/view/10.21037/amj-23-255/rc).

Highlight box

Key findings

- We report a case of a systemic lupus erythematosus (SLE) patient with nephritis, pancarditis, and neurological involvement. She also developed rhabdomyolysis during SLE treatment. She responded well to the therapeutic plasma exchange (TPE) treatment.

What is known and what is new?

- Rhabdomyolysis is a rare complication of SLE. Few cases have been documented on this topic.
- A description of the effective management via TPE in a case of rhabdomyolysis coexisting with SLE is provided.

What is the implication, and what should change now?

- This study highlights that rhabdomyolysis accompanying SLE is a rare and potentially mortal situation. TPE is an outstanding treatment option in such cases when the other treatment options are inefficient or inappropriate.

Case presentation

A 23-year-old female patient, who had been diagnosed with SLE since the age of 11 years (April 2003), was admitted to our inpatient clinic in June 2015 with complaints of shortness of breath, decreased urine output, and widespread edema for 1 week. She was put on high dose systemic steroid and cyclophosphamide treatments in May 2012 due to lupus nephritis and lupus myocarditis; however, treatment with cyclophosphamide was ceased due to a hypersensitivity reaction with respiratory and circulatory collapse, namely anaphylaxis. At discharge, she was prescribed methylprednisolone, hydroxychloroquine, and mycophenolate mofetil treatments; however, she did not show up for planned follow-up and treatment in June 2012 due to psychosocial problems.

© AME Medical Journal. All rights reserved.

On physical examination, the patient's general condition was poor, agitated, and confused. Arterial blood pressure was 150/90 mmHg, pulse was 120/minute. She was hyperthermic, tachypneic and hypoxemic; temperature was 39 ℃, respiratory rate was 30/minute, and fingertip oxygen saturation was 88%. Lung auscultation revealed roughening of breath sounds and rales in the bilateral basal and middle zones. Cardiac auscultation revealed tachycardia, decreased S1 and S2 intensity, and the existence of S3 and S4 additional sounds. Bilateral pretibial edema was prominent, due to renal and cardiac fluid overload. Appearance of the skin of upper extremities was compatible with livedo reticularis, suggestive of vasculitis. There were no heliotrope rash, shawl sign, and Gottron's papules, compatible with dermatomyositis.

In laboratory tests, there was significant hypoxemia in arterial blood gas evaluation (PO_2: 71.7 mmHg). Hematologic involvement due to SLE was evident, hemoglobin was 8.3 g/dL, white blood cell (WBC) count was 7.1×10^9/L with a shift to the left, lymphocyte count was 1.2×10^9/L and platelet count was 184×10^9/L. The blood urea nitrogen (BUN) was 105 mg/dL, serum creatinine was 4.61 mg/dL, serum albumin was 2.3 g/dL, serum total protein was 4.3 g/dL, erythrocyte sedimentation rate (ESR) was 85 mm/h, C-reactive protein (CRP) was 48.2 mg/L. Troponin I was 0.13 ng/mL. Thyroid function tests, namely thyroid stimulating hormone (TSH), free T4 (FT4), and free T3 (FT3), were in normal ranges. Serum complement levels were significantly low; C3 was 0.469 g/L, C4 was 0.0853 g/L. Antinuclear antibody (ANA), anti-dsDNA, anti-nucleosome, anti-histone, anti-Smith, perinuclear anti-neutrophil cytoplasmic antibody (p-ANCA) were positive. Haematuria and pyuria were present in urinalysis. Protein level in 24-hour urine was 3,480 mg. There was extended spectrum beta lactamase positive *Escherichia coli* (*E. coli*) growth in the urine culture. The remainder of the laboratory tests were not remarkable (*Table 1*).

Thorax computerized tomography (CT) examination revealed peribronchovascular infiltration and ground glass opacities, primarily located in the perihilar areas, and pleural effusion. Although the findings were considered consistent with pulmonary edema, *Pneumocystis jirovecii* pneumonia was also considered in the differential diagnosis (*Figure 1*).

Sinus tachycardia and diffuse T wave negativity were detected by electrocardiogram. In echocardiographic (ECO) evaluation, ejection fraction (EF) was measured as 15%; also, ECO revealed enlargement of all heart chambers, significant hypokinesia in the left ventricle, minimal pericardial effusion, a bright appearance suggesting inflammatory cell infiltration in the myocardium and pericardium, and a semi-mobile structure with a soft echogenicity with a diameter of 1.0 cm × 0.6 cm on the posterior mitral leaflet. All these findings were compatible with Libman-Sacks endocarditis, myocarditis, and pericarditis, namely pancarditis.

One thousand mg/day pulse methylprednisolone for 3 consecutive days, hydroxychloroquine 200 mg twice a day, acetyl salicylic acid 100 mg/day, pantoprazole 40 mg once a day, amlodipine 10 mg/day, furosemide 20 mg four times a day and 6 mL/min oxygen treatments were commenced. Besides, clarithromycin 500 mg twice a day, piperacillin-tazobactam 2.25 g four times a day, trimethoprim-sulfamethoxazole 400 mg/80 mg twice a day, and daptomycin 500 mg/day were initiated. Since the patient was not hyperlipidemic, statin had not been commenced. Additionally, hemodialysis was performed concomitantly.

On the 5th day of hospitalization, the patient complained of generalized muscle weakness and widespread subtle pain. Aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) levels increased up to twice normal limits; however, alanine aminotransferase (ALT) levels were normal. In the tests performed on suspicion of rhabdomyolysis, creatine kinase (CK) levels were 3,707 U/L, potassium (K) level was 5 mEq/L, phosphorus (P) level was 7.3 mg/dL, myoglobin level was 108.6 ng/mL. Urinalysis revealed hemoglobinuria and myoglobinuria (*Table 2*).

Daptomycin was immediately discontinued, and the patient received TPE for 10 consecutive days beginning on the 6th day of hospitalization. The amount of the exchanged plasma was 3,000 mL/day. Only perioral paresthesia and carpopedal spasm developed as complications during treatment; and the symptoms responded rapidly to calcium replacement.

On the 15th day of hospitalization, urine output increased to 3,000 mL/day, and serum creatinine level decreased to 1.4 mg/dL. Hypoxemia resolved. EF was measured as 40% in follow-up ECO. Pleural and pericardial effusion resolved. All of the symptoms were resolved afther the treatments. Laboratory parameters were returned to their baseline levels (*Table 3*). Considering that non-compliance with medical treatment could be a sign of lupus-related psychiatric involvement, the patient was consulted to the Psychiatry Department. On the 20th day of hospitalization, she was discharged with medical treatments including valsartan

© AME Medical Journal. All rights reserved.

Table 1 Initial laboratory values of the patient

Variables	Patient's value	References
Hemoglobin (g/dL)	8.3	11.7–15.5
Hematocrit (%)	25.1	35–45
WBC count (×10^9/L)	7.1	1.8–7.7
Lymphocyte count (×10^9/L)	1.2	1.5–4
Platelet count (×10^9/L)	184	150–400
AST (U/L)	33	0–35
ALT (U/L)	21	0–35
Alkaline phosphatase (U/L)	60	35–104
Gamma-glutamyl transferase (U/L)	27	5–36
BUN (mg/dL)	105	6–20
Creatinine (mg/dL)	4.61	0.8–1.5
Albumin (g/dL)	2.3	3.5–5.2
Total protein (g/dL)	4.3	6.6–8.7
ESR (mm/h)	85	0–20
CRP (mg/L)	48.2	0–5
TSH (μIU/mL)	0.35	0.34–5.6
FT4 (pmol/L)	10.69	7–16
FT3 (pmol/L)	5.65	3.1–6.8
C3 (g/L)	0.469	0.9–1.8
C4 (g/L)	0.0853	0.1–0.4
CK (U/L)	48	26–140
Troponin I (ng/mL)	0.13	<0.06
Protein (24-hour urine) (mg/24 h)	3,480	<140
Sodium (Na) (mEq/L)	138	136–145
Potassium (K) (mEq/L)	4.4	3.5–5.1
Chloride (Cl) (mEq/L)	104	98–108
Calcium (Ca) (mg/dL)	8.7	8.6–10.2
Magnesium (Mg) (mg/dL)	2.28	1.6–2.6
Phosphorus (P) (mg/dL)	3.16	2.7–4.5
Ferritine (ng/mL)	121	11–307
LDH (U/L)	195	125–220
PO_2 (arterial blood gas) (mmHg)	71.7	80–100
PCO_2 (arterial blood gas) (mmHg)	21.7	35–45
HCO_3 (mmol/L)	16.5	22–26
Lactate (mmol/L)	1.5	0.4–2.2
pH (arterial blood gas)	7.396	7.35–7.45
ANA	1:640 positive (nuclear coarse specked pattern)	Negative
Anti-dsDNA	1:320 positive	Negative
Anti-Nucleosome	1:320 positive	Negative
Anti-Histon	1:320 positive	Negative
Anti-Sm	1:320 positive	Negative
p-ANCA	Positive	Negative

WBC, white blood cell; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BUN, blood urea nitrogen; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; TSH, thyroid stimulating hormone; FT3, free T3; FT4, free T4; LDH, lactate dehydrogenase; CK, creatine kinase; ANA, antinuclear antibody; dsDNA, double stranded deoxyribonucleic acid; p-ANCA, perinuclear anti-neutrophil cytoplasmic antibody.



Figure 1 Thorax CT image of the patient was compatible with pulmonary edema and *Pneumocystis jirovecii* pneumonia. CT, computed tomography.

plus hydrochlorothiazide 160/12.5 once a day, lercanidipine 10 mg/day, furosemide 40 mg/day, acetyl salicylic acid 100 mg/day, prednisolone 50 mg/day, pantoprazole 40 mg/day, hydroxychloroquine 200 mg twice a day, mycophenolate mofetil 500 mg twice a day, and escitalopram 20 mg/day. The timeline process for this patient is illustrated in *Figure 2*. The patient was recommended to visit our clinic after 1 month for follow-up. However, since the patient moved to a different city, she stated that she continued her follow-up in a different center when contacted by phone. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national

© AME Medical Journal. All rights reserved.

Table 2 Laboratory values of the patient during rhabdomyolysis

Variables	Patient's value	References
Hemoglobin (g/dL)	9.1	11.7–15.5
Hematocrit (%)	28	35–45
WBC count (×10^9/L)	8.4	1.8–7.7
Lymphocyte count (×10^9/L)	1.6	1.5–4
Platelet count (×10^9/L)	192	150–400
AST (U/L)	82	0–35
ALT (U/L)	26	0–35
Alkaline phosphatase (U/L)	97	35–104
Gamma-glutamyl transferase (U/L)	64	5–36
BUN (mg/dL)	73	6–20
Creatinine (mg/dL)	3.85	0.8–1.5
TSH (μIU/mL)	0.58	0.34–5.6
FT4 (pmol/L)	9.74	7–16
FT3 (pmol/L)	5.46	3.1–6.8
Albumin (g/dL)	2.5	3.5–5.2
Total protein (g/dL)	4.7	6.6–8.7
Sodium (Na) (mEq/L)	136	136–145
Potassium (K) (mEq/L)	5	3.5–5.1
Chloride (Cl) (mEq/L)	107	98–108
Calcium (Ca) (mg/dL)	8.8	8.6–10.2
Magnesium (Mg) (mg/dL)	2.08	1.6–2.6
Phosphorus (P) (mg/dL)	7.3	2.7–4.5
LDH (U/L)	437	125–220
CK (U/L)	3,707	26–140
Myoglobin (ng/mL)	108.6	14–66

WBC, white blood cell; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BUN, blood urea nitrogen; TSH, thyroid stimulating hormone; FT3, free T3; FT4, free T4; LDH, lactate dehydrogenase; CK, creatine kinase.

Table 3 Laboratory values of the patient following TPE treatment

Variables	Patient's value	References
Hemoglobin (g/dL)	8.5	11.7–15.5
Hematocrit (%)	26.4	35–45
WBC count (×10^9/L)	6.8	1.8–7.7
Lymphocyte count (×10^9/L)	1.7	1.5–4
Platelet count (×10^9/L)	169	150–400
AST (U/L)	26	0–35
ALT (U/L)	19	0–35
Alkaline phosphatase (U/L)	76	35–104
Gamma-glutamyl transferase (U/L)	34	5–36
BUN (mg/dL)	18	6–20
Creatinine (mg/dL)	1.4	0.8–1.5
TSH (μIU/mL)	0.62	0.34–5.6
FT4 (pmol/L)	9.18	7–16
FT3 (pmol/L)	5.21	3.1–6.8
Albumin (g/dL)	2.4	3.5–5.2
Total protein (g/dL)	4.8	6.6–8.7
Sodium (Na) (mEq/L)	139	136–145
Potassium (K) (mEq/L)	3.7	3.5–5.1
Chloride (Cl) (mEq/L)	107	98–108
Calcium (Ca) (mg/dL)	8.9	8.6–10.2
Magnesium (Mg) (mg/dL)	1.99	1.6–2.6
Phosphorus (P) (mg/dL)	4.4	2.7–4.5
LDH (U/L)	216	125–220
CK (U/L)	107	26–140
Myoglobin (ng/mL)	36	14–66

TPE, therapeutic plasma exchange; WBC, white blood cell; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BUN, blood urea nitrogen; TSH, thyroid stimulating hormone; FT3, free T3; FT4, free T4; LDH, lactate dehydrogenase; CK, creatine kinase.

research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

To the best of our knowledge, our case is the first that displayed nephritis, carditis, and neuropsychiatric involvement, as well as rhabdomyolysis, and that responded dramatically to TPE.

Additionally, in our case, besides being ANA positive, p-ANCA positivity is also present. In a recent study, it has been mentioned that p-ANCA positivity may be associated with renal dysfunction in SLE cases, but it is also noted

© AME Medical Journal. All rights reserved.

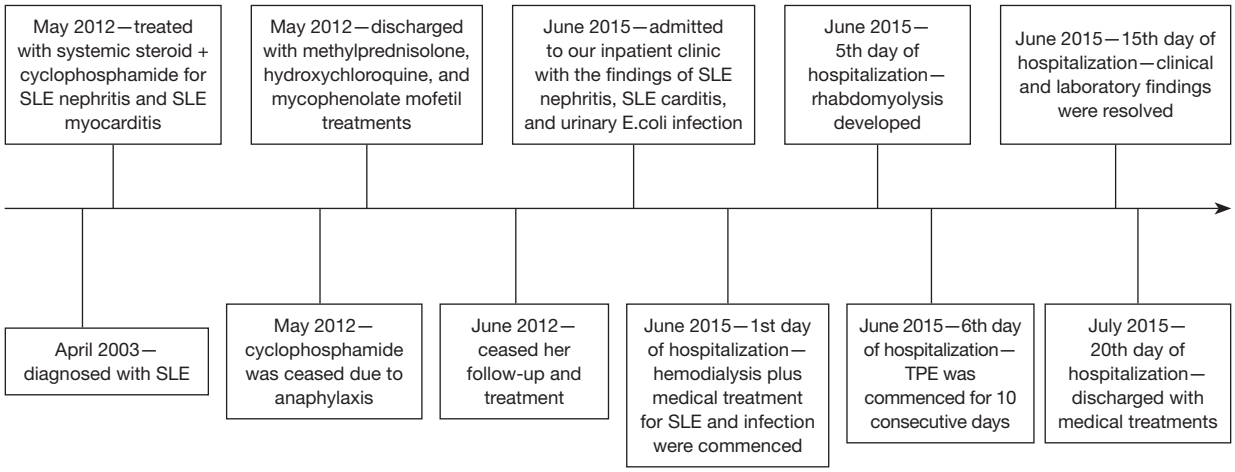


Figure 2 Timeline process of the patient. SLE, systemic lupus erythematosus; TPE, therapeutic plasma exchange.

that anti-dsDNA positivity in SLE cases may lead to p-ANCA false positivity (12). Accordingly, the observed p-ANCA positivity in our case may be associated with SLE pathophysiology without ANCA-associated vasculitis.

There are some limitations to our case presentation. Firstly, we cannot share the long-term outcomes of TPE therapy in our case as we could not continue the long-term follow-up of our patient in our clinic. Additionally, the lack of measurement of myositis-specific antibodies in our case is another limitation. However, since there were no dermatomyositis-compatible skin rashes in our case and due to the use of medications strongly associated with rhabdomyolysis, our case was not considered consistent with idiopathic inflammatory myopathy.

For SLE treatment, non-specific anti-inflammatory and immunosuppressive treatments including corticosteroids, hydroxychloroquine, cyclophosphamide, mycophenolate mofetil, calcineurin inhibitors, azathioprine, and methotrexate are used, as well as various biological agents, including belimumab, which targets the B lymphocyte stimulator (BlyS), anifrolumab, which targets the type 1 interferon (IFN) receptor, and rituximab, which targets cluster of differentiation 20 (CD20) (13). Although SLE's mortality and morbidity can be significantly reduced with adequate immunosuppressive treatment modalities, there are certain risks and complications. The development of secondary infections, organ toxicity, malignancy, or the possibility of treatment failure are hallmarks of predisposing factors for mortality and morbidity (1).

During TPE, the patient's blood is cleaned extracorporeally and returned to the circulation with replacement fluid (14). TPE is generally considered as a safe treatment method; the most common complication is hypocalcemia, as seen in our case, and it responds well to calcium replacement (14).

Particularly, SLE cases with diffuse alveolar hemorrhage, neuropsychiatric involvement, thrombotic microangiopathy, catastrophic antiphospholipid antibody syndrome, thrombotic thrombocytopenic purpura, and renal involvement constitute the main indications for TPE, which has been shown to significantly reduce mortality rates in these cases (5). In addition, there are data indicating that TPE provides clinical benefit on a case-by-case basis in cases of lupus carditis (15). TPE may be an outstanding treatment option in SLE patients who have inadequate response to the immunosuppressive and immunomodulatory treatments, where the use of these treatments is contraindicated, or which have side effects due to these treatments, including infection and allergic reactions (4,15,16).

Rhabdomyolysis, a clinical condition in which the necrotic muscle cell contents enter the vascular circulation, may develop secondary to traumatic or non-traumatic reasons (2). The most common causes in adults are trauma, drug exposure and infections (17). Several drugs that can cause rhabdomyolysis include various antibiotics including daptomycin, macrolides, trimethoprim-sulfamethoxazole, piperacillin-tazobactam, as well as systemic corticosteroids (2,18). In a recent analysis, the association of rhabdomyolysis with daptomycin treatment was significantly stronger compared with most other antibiotics (18). Many viral, bacterial, fungal, and protozoal infections, including urinary E.coli infection, may provoke rhabdomyolysis, and the accused mechanisms include tissue

© AME Medical Journal. All rights reserved.

hypoxia, direct invasion of muscle tissue by the infectious agent, endotoxin-mediated muscle damage or lysosomal enzyme activation (2,19). Prolonged immobilization may also cause rhabdomyolysis (2).

Rhabdomyolysis causes non-specific symptoms including muscle pain, swelling in muscle groups, cramps, difficulty in movements, and darkening of the urine (2). Systemic symptoms such as fever, abdominal pain, nausea, and vomiting, as well as conditions with high morbidity and mortality such as cardiovascular deterioration due to electrolyte imbalance, and acute renal failure may occur (2). Laboratory findings include increased serum levels of various components of muscle cells such as CK, myoglobin, LDH, AST, aldolase, and hyperkalemia, hyperphosphatemia, hypocalcemia (2). Fluid replacement and mannitol, urine alkalinization, treatment of electrolyte disorders, and dialysis, in necessary cases, may be preferred for treatment (2).

To date, 10 cases of rhabdomyolysis with lupus, 8 of which were SLE, other than ours, have been reported (3). Of these 8 cases, 4 were infection-related, 2 were drug-related, one was related to hyponatremia, and the etiological factor was not identified in one case (3).

In our patient, possible etiological factors of rhabdomyolysis were assumed as immobilization, urinary E.coli infection, and use of daptomycin as well as corticosteroids; however, the extent to which these factors affected the development of rhabdomyolysis could not be evaluated. Since the patient had severe congestive heart failure, intravenous fluid therapy and mannitol could not be administered due to concerns that it might worsen pulmonary edema. Although lupus nephritis requires heavy immunosuppressive treatment; it could not be applied because the patient had both renal failure and a history of anaphylaxis with cyclophosphamide. The patient was at high risk for permanent nephropathy due to both the tubular toxicity of myoglobin and the immune complexes present in the circulation. In this context, TPE was considered to be the most rational treatment approach to prevent negative outcomes.

In our case, potential mechanisms of benefit from TPE therapy include the removal of autoantibodies and immune complexes associated with SLE activation from the circulation, clearance of muscle damage markers elevated in the blood due to rhabdomyolysis which could lead to organ damage, and normalization of electrolyte imbalances resulting from rhabdomyolysis.

Further studies are required to illuminate the relationship between SLE and rhabdomyolysis and the effectiveness of TPE in rhabdomyolysis more clearly.

Conclusions

Rhabdomyolysis accompanying SLE is an uncommon situation that raises the risk of morbidity and mortality. TPE may be an effective and safe treatment option in SLE where there is an inadequate response to the classical treatments, where the side effects of treatment agents were hazardous, or where treatment agents are contraindicated. Larger-scale studies on the use of TPE for the treatment of rhabdomyolysis are a must.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://amj.amegroups.com/article/view/10.21037/amj-23-255/rc

Peer Review File: Available at https://amj.amegroups.com/article/view/10.21037/amj-23-255/prf

Conflicts of Interest: Both authors have completed the ICMJE uniform disclosure form (available at https://amj.amegroups.com/article/view/10.21037/amj-23-255/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Open Access Statement: This is an Open Access article distributed in accordance with the Creative Commons Attribution-NonCommercial-NoDerivs 4.0 International License (CC BY-NC-ND 4.0), which permits the non-

© AME Medical Journal. All rights reserved.

commercial replication and distribution of the article with the strict proviso that no changes or edits are made and the original work is properly cited (including links to both the formal publication through the relevant DOI and the license). See: https://creativecommons.org/licenses/by-nc-nd/4.0/.

References

1. Justiz Vaillant AA, Goyal A, Varacallo M. Systemic Lupus Erythematosus. Treasure Island (FL): StatPearls Publishing; 2023 [cited 2023 Oct 22].
2. Cabral BMI, Edding SN, Portocarrero JP, et al. Rhabdomyolysis. Dis Mon 2020;66:101015.
3. Lee IH, Cho S, Ahn DJ, et al. Systemic lupus erythematosus presenting as hyponatremia-associated rhabdomyolysis: A case report. Medicine (Baltimore) 2021;100:e27390.
4. Zanatta E, Cozzi M, Marson P, et al. The role of plasma exchange in the management of autoimmune disorders. Br J Haematol 2019;186:207-19.
5. Aguirre-Valencia D, Naranjo-Escobar J, Posso-Osorio I, et al. Therapeutic Plasma Exchange as Management of Complicated Systemic Lupus Erythematosus and Other Autoimmune Diseases. Autoimmune Dis 2019;2019:5350960.
6. Boparai S, Lakra R, Dhaliwal L, et al. Therapeutic Plasma Exchange in Severe Rhabdomyolysis: A Case-Control Study. Cureus 2023;15:e39748.
7. Levin PD, Levin V, Weissman C, et al. Therapeutic plasma exchange as treatment for propofol infusion syndrome. J Clin Apher 2015;30:311-3.
8. Padmanabhan A, Connelly-Smith L, Aqui N, et al. Guidelines on the Use of Therapeutic Apheresis in Clinical Practice - Evidence-Based Approach from the Writing Committee of the American Society for Apheresis: The Eighth Special Issue. J Clin Apher 2019;34:171-354.
9. Raj VM, Alladin A, Pfeiffer B, et al. Therapeutic plasma exchange in the treatment of exertional heat stroke and multiorgan failure. Pediatr Nephrol 2013;28:971-4.
10. Yang KC, Fang CC, Su TC, et al. Treatment of fibrate-induced rhabdomyolysis with plasma exchange in ESRD. Am J Kidney Dis 2005;45:e57-60.
11. Kuroda M, Katsuki K, Uehara H, et al. Successful treatment of fulminating complications associated with extensive rhabdomyolysis by plasma exchange. Artif Organs 1981;5:372-8.
12. Wang Y, Yu X, Xie X, et al. Clinical features and outcomes of patients with antineutrophil cytoplasmic antibody-positive systemic lupus erythematosus. Ren Fail 2023;45:2235431.
13. Katarzyna PB, Wiktor S, Ewa D, et al. Current treatment of systemic lupus erythematosus: a clinician's perspective. Rheumatol Int 2023;43:1395-407.
14. Cervantes CE, Bloch EM, Sperati CJ. Therapeutic Plasma Exchange: Core Curriculum 2023. Am J Kidney Dis 2023;81:475-92.
15. Xing ZX, Yu K, Yang H, et al. Successful use of plasma exchange in fulminant lupus myocarditis coexisting with pneumonia: A case report. World J Clin Cases 2020;8:2056-65.
16. Soyuöz A, Karadağ Ö, Karaağaç T, et al. Therapeutic plasma exchange for refractory SLE: A comparison of outcomes between different sub-phenotypes. Eur J Rheumatol 2018;5:32-6.
17. Chavez LO, Leon M, Einav S, et al. Beyond muscle destruction: a systematic review of rhabdomyolysis for clinical practice. Crit Care 2016;20:135.
18. Teng C, Baus C, Wilson JP, et al. Rhabdomyolysis Associations with Antibiotics: A Pharmacovigilance Study of the FDA Adverse Event Reporting System (FAERS). Int J Med Sci 2019;16:1504-9.
19. Blanco JR, Zabalza M, Salcedo J, et al. Rhabdomyolysis of infectious and noninfectious causes. South Med J 2002;95:542-4.

doi: 10.21037/amj-23-255
Cite this article as: Altiner S, Ekinci A. A case report of systemic lupus erythematosus coinciding with rhabdomyolysis: a dramatic recovery via therapeutic plasma exchange. AME Med J 2024.

© AME Medical Journal. All rights reserved.