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Plasma exchange therapy in systemic lupus erythematosus: A single-center retrospective cohort study

Sistemik lupus eritematozusda plazma değişim tedavisi: Tek merkez retrospektif kohort çalışması

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Abstract

Objective: Few randomized controlled studies investigating the role of plasma exchange (PLEX) therapy shown no significant benefit in the management of lupus nephritis. However small case series have suggested potential efficacy in certain types of organ involvement in systemic lupus erythematosus (SLE).

Methods: We conducted a retrospective review of patient records who received PLEX therapy between October 2013 and March 2022 at our apheresis unit. Patients under the age of 18 and those who underwent PLEX therapy for non-rheumatic and rheumatic diseases other than SLE were excluded from the study. We collected comprehensive data including the primary indication for PLEX therapy, procedural details, concurrent immunosuppressive medications, overall survival, outcomes of organ involvement, and any complications associated with PLEX therapy.

Results: Among 58 patients with rheumatic diseases who underwent PLEX therapy we included 17 SLE patients. The main indication for PLEX was catastrophic antiphospholipid syndrome (n=5), diffuse alveolar hemorrhage (DAH) (n=5), neuropsychiatric involvement (n=4), thrombotic microangiopathy (n=2) and renal involvement (n=1). Nine patients experienced severe/opportunistic infections resulting in death only in 1 patient during PLEX. Additionally, 3 patients died due to active disease during PLEX. Among the survived patients PLEX therapy provided remission in 13 patients.

Conclusion: PLEX can be regarded as a supplementary treatment along with immunosuppressives, particularly for a subset of SLE patients experiencing conditions such as DAH and neuropsychiatric involvement. Despite high frequency of severe/opportunistic infections only one patient died.

Keywords: Plasma exchange, plasmapheresis, systemic lupus erythematosus, lupus

Öz

Amaç: Plazma değişimi (PLEX) tedavisinin lupus nefriti yönetiminde anlamlı bir fayda sağlamadığını gösteren az sayıda randomize kontrollü çalışma bulunmaktadır. Bununla birlikte, küçük olgu serileri, sistemik lupus eritematozusun (SLE) bazı organ tutulum tiplerinde plazma değişiminin etkili olabileceğini bildirmiştir.

Yöntem: Ekim 2013 ile Mart 2022 tarihleri arasında PLEX tedavisi alan hastaların kayıtlarını geriye dönük olarak inceledik. On sekiz yaşın altındaki hastalar, SLE dışında romatizmal hastalığı olanlar ve romatizmal hastalık dışı nedenlerle PLEX tedavisi yapılan hastalar dışlandı. PLEX tedavisinin başlıca endikasyonu, işlem detayları, eşzamanlı olarak kullanılan immünosüpresif ilaçlar, genel sağkalım, organ tutulumunun sonuçları ve PLEX tedavisi ilişkili komplikasyonlar gibi bilgiler not edildi.

Bulgular: Romatizmal hastalığı olup PLEX tedavisi uygulanan 58 hastadan 17 SLE hastası çalışmaya dahil edildi. PLEX tedavisinin birincil endikasyonları katastrofik antifosfolipid sendromu (n=5), diffüz alveolar hemoraji (DAH) (n=5), nöropsikiyatrik tutulum (n=4), trombotik mikroanjiyopati (n=2) ve renal tutulum (n=1) idi. PLEX sırasında 9 hastada ciddi/fırsatçı enfeksiyonlar geliştiği görüldü. Bir hasta enfeksiyona bağlı, 3 hasta aktif hastalık nedeniyle PLEX devam ederken kaybedildi. Sağ kalan hastaların 13'ünde PLEX tedavisi ile remisyon sağlandı.

Sonuç: PLEX, DAH ve nöropsikiyatrik tutulum gibi SLE hastalarının belli bir alt grubu için immünosüpresiflerle birlikte ek bir tedavi olarak değerlendirilebilir. Hastalarımızın yaklaşık yarısı ciddi veya fırsatçı enfeksiyonlarla karşılaşsa da, yalnızca bir hastada enfeksiyona bağlı mortalite gözlenmiştir.

Anahtar Kelimeler: Plazma değişimi, plazmaferez, sistemik lupus eritematozus, lupus

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Introduction

Systemic lupus erythematosus (SLE) is a complex autoimmune disease with a wide range of clinical symptoms and a variable disease course. The prognosis of patients has improved with the introduction of combined immunosuppressive and glucocorticoid (GC) therapy.[1] In general, management of SLE depends on disease severity, disease activity, clinical manifestations, and comorbidities. Cutaneous manifestations, musculoskeletal manifestations, and serositis are typically indicative of less severe disease, and may exhibit fluctuations in accordance with disease activity. Frequently, these conditions can be managed through the administration of nonsteroidal anti-inflammatory drugs or low-potency immunosuppressive agents, in addition to hydroxychloroquine and/or brief regimens of GCs. Organ or life-threatening disease manifestations, such as kidney, lung, and central nervous system involvement require more aggressive immunosuppression. In those patients, immunosuppressives (e.g. mycophenolate mofetil, cyclophosphamide) are mostly combined with high doses of systemic GCs. However, there is still some subgroup of patients who do not respond well to the standard of care (SOC).[2] Despite the lack of high-quality data, therapeutic plasma exchange therapy (PLEX) has been considered as an alternative treatment option in refractory and/or severe SLE patients.[3]

PLEX has been used for almost four decades in a wide variety of autoimmune diseases in which humoral factors play a role in the pathogenesis.[4] The underlying idea of the PLEX is based on the assumption that the reduction or elimination of specific pathological substances (e.g., autoantibodies, immune complexes, cryoglobulins) from the plasma can lead to the prevention of additional damage or even might help reversing the pathological condition.[5] The American Society for Apheresis (ASFA) has categorized the use of PLEX into four distinct groups based on the currently available evidence.[6] Category-I disorders are for which PLEX is the first-line therapy, either alone or with a combination of other therapies. PLEX is accepted as the second-line therapy in category-II disorders. If the benefit of PLEX has not been fully demonstrated, those group of disorders are classified as category-III. If the published evidence indicates or implies that PLEX could potentially be harmful or ineffective, those disorders are classified as category-IV. PLEX has been studied in few randomized controlled trials (RCT) in patients with lupus nephritis (LN) and none of them demonstrated any significant improvement in renal outcome compared to the SOC.[7-11] On the other hand, there are significant number of case reports and series have reported positive outcome especially in refractory and severe SLE patients.[3,12,13] According to ASFA guidelines, SLE patients with severe types of organ involvement, including central nervous system (CNS) involvement, diffuse alveolar hemorrhage (DAH), thrombotic microangiopathy, cryoglobulinemia, cytopenia, hyperviscosity, but not LN, are classified into category-II.[6] Additionally, the ASFA guideline published in 2019 upgraded catastrophic antiphospholipid syndrome (CAPS) from Category II to Category I.[14] In the ASFA guideline published in 2023, CAPS continues to be classified under Category I.[6]

The objective of this real-world study was to retrospectively evaluate the overall survival of patients, identify causes of mortality, assess the incidence of infectious and non-infectious complications, and determine the risks and benefits of PLEX therapy in patients with SLE [with or without antiphospholipid syndrome (APS)] in a real-world clinical setting.

Materials and Methods

A retrospective chart review was conducted on patients who received PLEX therapy between October 2013 and March 2022. We excluded patients who underwent PLEX treatment for conditions other than rheumatic diseases, such as thrombotic thrombocytopenic purpura (TTP), multiple sclerosis, or hyperviscosity syndrome, and who were under 18 years old. Additionally, patients with rheumatic diseases other than SLE, including ANCA-associated vasculitis (AAV) (n=28), cryoglobulinemic vasculitis (n=7), systemic sclerosis (n=2), Goodpasture syndrome (n=2), IgA vasculitis (n=1), and dermatomyositis (n=1), were also excluded. We have previously reported the outcome of PLEX therapy among our 28 AAV patients.[15] We obtained comprehensive data from patient charts, including information on the underlying rheumatic disease, the primary indication for PLEX therapy, specific procedural details (such as the use of albumin and/or, fresh frozen plasma (FFP) peripheral or central venous catheters, and a number of PLEX sessions), concomitant immunosuppressive treatments [such as steroid pulses, cyclophosphamide (CYC), rituximab (RTX), or intravenous immunoglobulin (IVIG)], overall survival rates, outcomes of organ involvement, and any complications associated with PLEX therapy. Mortality, and as well as the impact on organ function, were assessed both during the administration of PLEX and at the 3-month and 12-month post-treatment follow-up. We examined the occurrence of infections during the initial 5-week period since patients treated with PLEX have been shown to continue to exhibit low levels of immunoglobulin G until week 5.[16] We also assessed the mortality until the final follow-up

appointment for patients. The study was planned according to the Declaration of Helsinki, and an independent ethics committee of Cerrahpasa Medical Faculty gave permission to conduct this study (date: 02.06.2022, approval number: 396452).

Statistical Analysis

The demographic, baseline, and follow-up characteristics of the patients were presented with the descriptive statistics. Data are expressed as means and standard deviations (SD), median values with ranges (Q1-Q3) or frequency (%).

Results

Baseline Characteristics of Patients

The retrospective analysis of medical records identified a total of 318 individuals who received PLEX treatment between October 2013 and March 2022 at our apheresis facility. Following the exclusion of 253 patients who underwent PLEX for non-rheumatic conditions, 41 patients with rheumatic diseases other than SLE, and 7 patients who were below the age of 18, a total of 17 patients diagnosed with SLE were subjected to further evaluation (Figure 1).

As expected, the majority of the patients were female (n=15, 88%) and the mean age of the patients was 33.4±9.4. The main indications for PLEX were DAH in 5 patients, CAPS in 5, CNS involvement in 4, TTP in 2 patients, and rapidly progressive glomerulonephritis (RPGN) in 1 patient (Table 1). Hemodialysis was also started concomitant with PLEX therapy in 3 patients. The causes for hemodialysis were CAPS in 1, class IV LN in 1 and RPGN in 1 patient. Due to the patient's thrombocytopenia and the potential risk of hemorrhage associated with PLEX therapy, a renal biopsy could not be performed in the case of RPGN. Immunosuppressive agents (CYC, RTX), high dose intravenous pulse methylprednisolone (1 g/day for 3 days) followed by prednisolone 1 mg/kg/day were initiated in all but two patients in conjunction with PLEX. IVIG was used in combination with immunosuppressives in 11 patients and as a solo treatment in 2 patients. The treatment details of each patient are given in the Table 1.

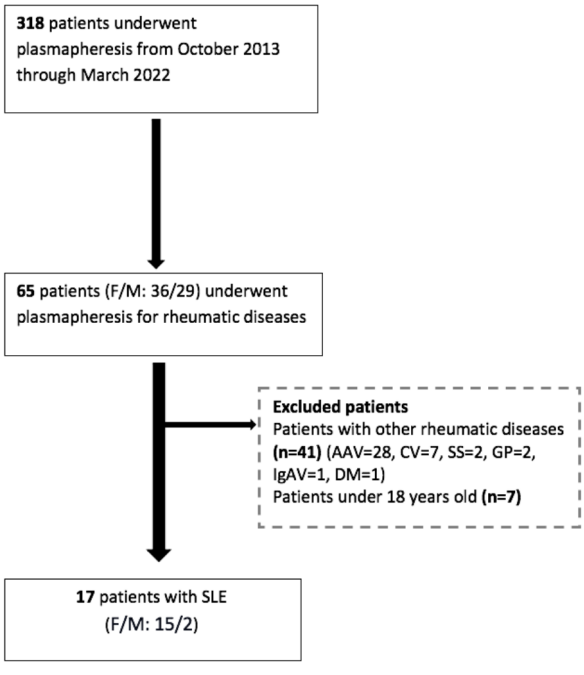

Figure 1. Flow-chart of the included patients
AAV: ANCA-associated vasculitis, CV: Cryoglobulinemic vasculitis, DM: Dermatomyositis, GP: Goodpasture syndrome, IgAV: IgA vasculitis, SLE: Systemic lupus erythematosus, SS: Systemic sclerosis

Ten (59%) out of 17 SLE patients had concomitant APS (Table 1). Three patients had triple positive antiphospholipid (aPL) profile, one had double positivity [lupus anticoagulant (LA) + anticardiolipin (aCL)] and the remaining had only one type of aPL [LA=3, aCL=2, antibeta-2 glycoprotein (anti-β2GPI) =1] antibodies. Anti-double-stranded DNA (anti-dsDNA) was positive in 11 patients, Ro/SSA was positive in 2, anti-Smith (anti-Sm) was positive in 2, and La/SSB was positive in one patient.

Features of Plasma Exchange Therapy

The median number of PLEX was 4 (Q1-Q3=3-5) (Table 2). A Fresenius Comtec 2010 machine (centrifugal technique) was used for the procedure to exchange an average of 1.3 plasma volumes. PLEX was performed using a central venous catheter and peripheral veins in 9 (53%) and 8 (47%) patients, respectively. Plasma was replaced with fresh frozen plasma (FFP) (n=5, 29%), albumin (n=2, 12%), or both (n=10, 59%).

Outcomes

Death and Complications

Four (24%) patients died during PLEX therapy. The cause of mortality in three patients was attributed to the underlying active disease, i.e. CAPS (n=2) and RPGN (n=1). Death in one patient was associated with both infection (CMV infection and pseudomonas aeruginosa pneumonia) and active disease (CNS involvement).

While no additional deaths occurred within the first three months, three more deaths were observed before month 12 related to COVID-19 infection, relapsing disease (DAH), and aspiration-related cardiopulmonary arrest.

Table 1. Clinical characteristics and outcome of 17 SLE patients who underwent PLEX therapy

Age/Sex	Concomitant APS	Main indication for PLEX	Concomitant IS	Infection during PLEX	Outcome
31/F	No	DAH	MP+CYC+IVIG	No	Remission at month 12
23/F	Yes	CAPS	MP+CYC+IVIG	No	Died at month 7 due to cardiopulmonary arrest
25/F	Yes	CAPS	MP+RTX+IVIG	No	Remission at month 12
30/F	No	CNS inv.	MP+RTX+IVIG	No	Remission at month 12
30/M	Yes	DAH	MP+CYC	No	Remission at month 12
28/F	No	RPGN	MP+CYC+IVIG	CMV	Died during PLEX
35/F	No	CNS inv.	MP+IVIG	CMV+ PAP	Died during PLEX
25/M	Yes	DAH	MP+CYC+IVIG	CMV	Died due to active disease at month 4
26/F	No	CNS inv.	MP+CYC	SMP	Remission at month 12
52/F	No	DAH	MP+IVIG	CMV + Lobar pneumonia	Died due to COVID-19 after 4 months of PLEX
47/F	Yes	DAH	MP+CYC+IVIG	PJP	Remission at month 12
49/F	Yes	CAPS	IVIG	Pneumonia	Died during PLEX
26/F	Yes	CAPS	MP+RTX+IVIG	PJP	Remission at month 12
47/F	Yes	CAPS	IVIG	No	Died during PLEX
32/F	Yes	TTP	MP+CYC+IVIG	Pneumonia + Soft tissue infection	Remission at month 12
34/F	Yes	CNS inv.	MP+RTX	No	Remission at month 12
28/F	No	TTP	MP+CYC	No	Remission at month 12

APS: Antiphospholipid syndrome, CAPS: Catastrophic antiphospholipid syndrome, CMV: Cytomegalovirus, CNS: Central nervous system, CYC: Cyclophosphamide, DAH: Diffuse alveolar hemorrhage, F: Female, IS: Immunosuppressives, IVIG: Intravenous immunoglobulin, M: Male, MP: Methylprednisolone, PAP: Pseudomonas aeruginosa pneumonia, PJP: Pneumocystis jirovecii pneumonia, PLEX: Plasma exchange, RPGN: Rapidly progressive glomerulonephritis, RTX: Rituximab, SMP: Stenotrophomonas maltophilia pneumonia, TTP: Thrombotic thrombocytopenic purpura

Table 2. Features of plasma exchange therapy

Median number of PLEX sessions (Q1-Q3)	**4 (3-5)**
PLEX with	
FFP, n (%)	5 (29)
Albumin, n (%)	2 (12)
FFP and albumin, n (%)	10 (59)
Route of venous access	
Peripheral venous, n (%)	8 (47)
Central venous, n (%)	9 (53)

FFP: Fresh frozen plasma, PLEX: Plasma exchange

The median follow-up starting from PLEX day 1 to the last visit was 19.2 months (Q1-Q3=1.7-40.4 months). Two additional deaths occurred after the first year of PLEX treatment. One patient (DAH) died due to COVID-19 at month 21, and the other (CAPS) died due to SLE related severe damage (heart and renal failure) at month 48.

Nine patients (53%) developed severe/opportunistic infections within the first 5 weeks of PLEX. Three of those patients had more than one type of infection. CMV was the most common type of infection and detected in 4 patients. Details on infections are given in the Table 1. Death was associated with infection only in 1 patient who also had concurrent active disease.

Organ Survival

Among the 5 patients with DAH, all recovered after PLEX therapy. Only 1 patient died due to a relapse of DAH at month 4. Among the 4 patients with CNS involvement, 3 recovered and one died due to active disease and infection while on PLEX therapy. Among the 5 patients with CAPS, 2 experienced multiple thrombotic complications such as multifocal cerebral infarcts, Budd-Chiari syndrome, and digital ischemia and died during PLEX therapy. The third patient who presented with extensive lower extremity deep vein thrombosis, Libman-Sacks endocarditis, and severe cutaneous necrosis, died at month 7 due to aspiration-related cardiopulmonary arrest. In the remaining two patients, one

had portal thrombosis and digital necrosis, and the other had Budd-Chiari syndrome and multiple cerebral infarcts. These two patients were still in remission at month 12. Two patients with TTP were also in remission at month 12. One with RPGN died while receiving PLEX therapy.

Discussion

Initially, PLEX therapy was introduced as a treatment for SLE with the assumption that removing pathogenic autoantibodies and immune complexes would help control disease activity. The first RCT conducted in SLE, where patients received six courses of PLEX within a span of two weeks, showed no clinical improvement.[17] However, this was a small study consisting of 10 mild SLE patients in each study arms. Furthermore, the patients included in this study probably did not represent the SLE patients who are most likely to benefit from PLEX therapy. The other RCT with a larger sample size (n=86), comparing PLEX plus prednisone and cyclophosphamide versus prednisone and cyclophosphamide alone in patients with LN also showed no benefit and had to be terminated early.[10] Subsequent controlled studies, which included small number of patients, repeatedly demonstrated no efficacy of PLEX in patients with LN.[8,18-20] The discouraging results of these studies have led to a significant decline in the use of PLEX therapy in lupus setting. However, data from the registries indicate that there are still some SLE patients receiving and benefiting from PLEX therapy.[21-23] Similarly, in our daily practice we prefer using PLEX therapy to treat some severe forms of SLE patients. Our findings, along with those from the registries support that PLEX therapy remains valuable for a carefully selected group of patients with specific indications such as DAH and CNS involvement. However, conducting prospective trials to precisely evaluate the role of PLEX therapy in these patient subgroups poses challenges due to the rarity of such cases.

The present study is a retrospective evaluation of 17 patients diagnosed with SLE who received therapeutic PLEX at our center. Within this cohort of patients, PLEX was primarily administered to five patients as a therapeutic intervention for CAPS, which is classified as Category I in the ASFA guideline. PLEX indications in the remaining 11 patients were DAH (n=5), CNS involvement (n=4) and TTP (n=2) all of which are Category II. Only in one patient PLEX indication was RPGN. Four out of 17 (24%) patients died during PLEX therapy; the cause was CAPS in 2 patients, CNS involvement in 1, and RPGN in 1 patient. Despite the high rate of severe/opportunistic infections (53%) observed within the first five weeks of PLEX therapy, only one patient died. It was difficult to definitively conclude whether the cause of death was due to the infection or the active disease in this patient.

Since the early years of PLEX therapy, concerns have been raised regarding an increased risk of infections due to the associated decrease in immunoglobulin levels.[24] The initial RCT conducted in SLE specifically assessing the infection rate in patients undergoing PLEX did not observe an elevated risk of infection in the PLEX group (68%) compared to SOC group (74%).[10] However, a recent meta-analysis conducted in ANCA-associated vasculitis revealed an increased risk of infection with PLEX therapy.[25] Additionally, non-rheumatic diseases such as TTP and autoimmune encephalitis exhibited a low infection rate.[26,27] These findings suggest that aggressive immunosuppressive therapy, high dose GC therapy and severe organ dysfunction may contribute to an elevated risk of infection in rheumatic diseases.

Study Limitations

Our study has several limitations. Firstly, it had a retrospective design. Secondly, the evaluation was conducted on a small number of SLE patients who received PLEX therapy, as it was based on the experience of a single center. Thirdly, it is difficult to attribute the observed benefits or complications solely to PLEX therapy given that almost all patients concurrently received high-dose GCs and immunosuppressive drugs. Fourthly, except for two patients (one with TTP and the other with RPGN), most of the patients were refractory to immunosuppressives which might be the explanation for the high mortality rate observed in this cohort.

Conclusion

In conclusion, despite the previous discouraging outcomes from RCTs, PLEX therapy continues to be employed at our center to manage severe SLE patients, similar to real-world data from registries. PLEX can be considered as an adjunctive treatment in addition to immunosuppressives, especially in a subgroup of SLE patients with DAH and CNS involvement. Although severe/opportunistic infections occurred in around half of our patients, infection-related mortality was observed in only one patient.

Ethics

Ethics Committee Approval: The study was planned according to the Declaration of Helsinki, and an independent ethics committee of Cerrahpasa Medical Faculty gave permission to conduct this study (date: 02.06.2022, approval number: 396452).

Informed Consent: Retrospective study.

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Concept: Y.Ö., T.A., S.N.E., T.E., A.E.E., S.U., G.H., E.S., M.M., İ.F., M.C.A., V.H., Design: Y.Ö., T.A., S.N.E., T.E., A.E.E., S.U., G.H., E.S., M.M., İ.F., M.C.A., V.H., Data Collection or Processing: Y.Ö., T.A., S.N.E., T.E., A.E.E., S.U., G.H., E.S., M.M., İ.F., M.C.A., V.H., Analysis or Interpretation: Y.Ö., T.A., S.N.E., T.E., A.E.E., S.U., G.H., E.S., M.M., İ.F., M.C.A., V.H., Literature Search: Y.Ö., T.A., S.N.E., T.E., A.E.E., S.U., G.H., E.S., M.M., İ.F., M.C.A., V.H., Writing: Y.Ö., T.A., S.N.E., T.E., A.E.E., S.U., G.H., E.S., M.M., İ.F., M.C.A., V.H.

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References

1. Katarzyna PB, Wiktor S, Ewa D, Piotr L. Current treatment of systemic lupus erythematosus: a clinician's perspective. Rheumatol Int 2023.
2. Accapezzato D, Caccavale R, Paroli MP, et al. Advances in the Pathogenesis and Treatment of Systemic Lupus Erythematosus. Int J Mol Sci 2023;24.
3. Kronbichler A, Brezina B, Quintana LF, Jayne DR. Efficacy of plasma exchange and immunoadsorption in systemic lupus erythematosus and antiphospholipid syndrome: A systematic review. Autoimmun Rev 2016;15:38-49.
4. Altobelli C, Anastasio P, Cerrone A, et al. Therapeutic Plasmapheresis: a revision of literature. Kidney Blood Press Res 2022;48:66-78.
5. Weinstein R. Basic principles of therapeutic plasma exchange. Transfus Apher Sci 2023;62:103675.
6. Connelly-Smith L, Alquist CR, Aqui NA, 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 Ninth Special Issue. J Clin Apher 2023;38:77-278.
7. Nakamura T, Ushiyama C, Hara M, et al. Comparative effects of plasmapheresis and intravenous cyclophosphamide on urinary podocyte excretion in patients with proliferative Lupus nephritis. Clin Nephrol 2002;57:108-13.
8. Wallace DJ, Goldfinger D, Pepkowitz SH, et al. Randomized controlled trial of pulse/synchronization cyclophosphamide/apheresis for proliferative lupus nephritis. J Clin Apher 1998;13:163-6.
9. Doria A, Piccoli A, Vesco P, et al. Therapy of lupus nephritis. A two-year prospective study. Ann Med Interne (Paris) 1994;145:307-11.
10. Lewis EJ, Hunsicker LG, Lan SP, Rohde RD, Lachin JM. A controlled trial of plasmapheresis therapy in severe lupus nephritis. The Lupus Nephritis Collaborative Study Group. N Engl J Med 1992;326:1373-9.
11. Clark WF, Cattran DC, Balfe JW, Williams W, Lindsay RM, Linton AL. Chronic plasma exchange in systemic lupus erythematosus nephritis. Proc Eur Dial Transplant Assoc 1983;20:629-35.
12. Garra W, Carmi O, Kivity S, Levy Y. Catastrophic antiphospholipid syndrome in lupus-associated immune thrombocytopenia treated with eltrombopag A case series and literature review. Medicine (Baltimore) 2023;102:e32949.
13. Man BL, Mok CC, Fu YP. Neuro-ophthalmologic manifestations of systemic lupus erythematosus: a systematic review. Int J Rheum Dis 2014;17:494-501.
14. 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.
15. Özgüler Y, Ak T, Esatoğlu SN. ANCA ile ilişkili vaskülitte plazma değişim tedavisi: Tek merkez retrospektif kohort çalışması. J Turk Soc Rheumatol 2022;14:131-6.
16. Pohl MA, Lan SP, Berl T. Plasmapheresis does not increase the risk for infection in immunosuppressed patients with severe lupus nephritis. The Lupus Nephritis Collaborative Study Group. Ann Intern Med 1991;114:924-9.
17. Wei N, Klippel JH, Huston DP, et al. Randomised trial of plasma exchange in mild systemic lupus erythematosus. Lancet 1983;1:17-22.
18. Pagnoux C. Plasma exchange for systemic lupus erythematosus. Transfus Apher Sci 2007;36:187-93.
19. Euler HH, Schwab UM, Schroeder JO, Hasford J. The Lupus Plasmapheresis Study Group: rationale and updated interim report. Artif Organs 1996;20:356-9.
20. Clark WF, Dau PC, Euler HH, et al. Plasmapheresis and subsequent pulse cyclophosphamide versus pulse cyclophosphamide alone in severe lupus: design of the LPSG trial. Lupus Plasmapheresis Study Group (LPSG). J Clin Apher 1991;6:40-7.
21. Korach JM, Petitpas D, Poiron L, et al. 14 years of therapeutic plasma exchange in France. Transfus Apher Sci 2001;25:73-7.
22. Norda R, Stegmayr BG, Swedish Apheresis Study G. Apheresis registry in Sweden: scope, techniques and indications for treatment. A report from the Swedish apheresis study group. Transfus Apher Sci 2001;24:49-55.
23. Clark WF, Rock GA, Buskard N, et al. Therapeutic plasma exchange: an update from the Canadian Apheresis Group. Ann Intern Med 1999;131:453-62.
24. Wing EJ, Bruns FJ, Fraley DS, Segel DP, Adler S. Infectious complications with plasmapheresis in rapidly progressive glomerulonephritis. JAMA 1980;244:2423-6.
25. Walsh M, Collister D, Zeng L, et al. The effects of plasma exchange in patients with ANCA-associated vasculitis: an updated systematic review and meta-analysis. BMJ 2022;376:e064604.
26. Zhang Y, Huang HJ, Chen WB, Liu G, Liu F, Su YY. Clinical efficacy of plasma exchange in patients with autoimmune encephalitis. Ann Clin Transl Neurol 2021;8:763-73.
27. Rock GA, Shumak KH, Buskard NA, et al. Comparison of plasma exchange with plasma infusion in the treatment of thrombotic thrombocytopenic purpura. Canadian Apheresis Study Group. N Engl J Med 1991;325:393-7.