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Review Article

Plasmapheresis- Review Article

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ABSTRACT

Plasmapheresis, which is defined as the removal of plasma, can be either "adjusted plasma" or "exchange of plasma". The former is defined as selective withdrawal of certain (un)-pathological plasma components in different ways such as perfusion and then returning the remained donor plasma to him, the latter is non-selective removal of all components of plasma to provide blood products for injection into patients or to be used as the input of blood transfusion refinery or to remove the pathogen contained plasma before compensating for the volume losses with an equal volume of plasma or more commonly, replacing plasma with a substitute fluid (colloid or crystalloid) such as albumin. Plasmapheresis was divided generally into two groups:

1- Plasma products by donor plasmapheresis

2- Therapeutic plasmapheresis

Therapeutic plasma exchange or TPE are often attributed to plasma that exit from the body of patient then compensated by any kind of replacement fluid volumes to support neurmolemic situation of patients. Plasmapheresis is currently used as a therapeutic modality in a wide array of conditions. Generally, plasmapheresis is used when a substance in the plasma, such as immunoglobulin, is acutely toxic and can be efficiently removed. Myriad conditions fall under this category, including neurologic, hematologic, metabolic, dermatologic, rheumatologic, and renal diseases, as well as intoxications, that can be treated with plasmapheresis.

Keywords: **Plasmapheresis, Exchange the Plasma**

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Introduction

Aphaeresis is derived from a Greek word and it means removing or separating (1).

Hemaphaeresis means bleeding from a donor and removing certain components and returning the remaining components to him (2-3).

There are two kinds of Hemaphaeresis (4):

1- Cytapheresis is withdrawal of the whole blood cells that have different titles by the type of cell. If it is separated from leukocytes, it is known as "Leukopheresis", if separated from red blood cells, it is "Erythropheresis" and if separated from platelets it is as "Plateletpheresis" or "Plasmapheresis".

2- Plasmapheresis, which is defined as the removal of plasma, can be either "adjusted plasma" or "exchange of plasma". The former is defined as selective withdrawal of certain (un)-pathological plasma components in different ways such as perfusion and then returning the remained donor plasma to him, the latter is non-selective removal of all components of plasma to provide blood products for injection into patients or to be used as the input of blood transfusion refinery or to remove the pathogen contained plasma before compensating for the volume losses with an equal volume of plasma or more commonly, replacing plasma with a substitute fluid (colloid or crystalloid) such as albumin (5-7).

History

Hendon (1902s) was the first to conduct research on the therapy method of Hemaphairesis by withdrawing blood from animals to be washed and reinjected. Fleig (1909) after Hendon used this method for the treatment of human uremia (2). Abel *et al.* (1914) have explained the removal of plasma technique through the treatment of poisoned dogs whose kidneys had been removed. To that effect, in a bid to get more anti-serum from immunized horses for serum therapy, they correctly did plasmaphresis while preventing blood losses (3, 4).

At the end of 1940s and early 1950s, simultaneously the appearance of plastic advanced blood bag and advanced refrigerated centrifuge, hemaphresis was used more broadly as a treatment method. In 1952, Carifols -Lucas used the manual plasmapheresis procedure for the preparation of blood products. Thus, whole blood has been transfused from a donor, and then the plasma was separated from red blood cells by using centrifuge, and red blood cells after a week bleeding was returned to the donor (1, 8-10). He also reported the usefulness of pheresis in the patients` therapy with hypertension disease. In 1950s, Dr. Edwin Cohn had established the using a continuous flow centrifuge with more than 60 years since it was invented by De Laval for use in the dairy industry to prepare blood products and improve the effectiveness of a therapeutic plasmapheresis during the Second World War. These devices can be separated an on-line blood component in a closed system and in this system, the obtained products were sterile (1, 11-13).

In 1962, Judson provided a device for separation of leukocytes from the whole blood by supporting IBM and NCI and managed to produce 2,990 units of IBM successfully. This device consists of disposable plastic tubes that were sterile previously, and worked with a steady stream of closed system and it was the first device that enables functionality to provide the processing of large volumes of blood. This device is capable of exchanging plasma, collecting platelets, granulocytes, lymphocytes and erythrocytes (1, 14-17).

CS3000 were accessible commercially in the early 1980s, produced by Fenwall. It is a continuous -flow device consisting of two rotating chamber with a special form. In 1988, Cobe Company began selling a new device as SPECTRA. This type of devices is automatically

equipped with computer systems that can platelet aphaeresis with minimal contamination of white blood cell. Moreover, SPECTRA have been moving for medical procedures affairs that are easily transferred to the patient's room (18).

Subsequent development of the basic principles established by the people, eventually leading to the construction of several kinds of semi-automatic separation devices for clinical applications in blood that the wider use of plasmaphresis was followed as a method of treatment. Continuation of this trend depends on the development of consumer devices and software whatever needs to be more selective separation of blood components, and other ingredients can be returned to the donor or patient (18, 19).

Plasma pheresis was divided generally into two groups (1, 20):

Plasma products by donor plasmapheresis

Therapeutic plasma pheresis

Plasma Products by Donor Plasmapheresis

Description

The donor plasmapheresis interpreted the donated plasma by volunteers to be used in transfusion or conversion to some specific products in plasma processing center (21).

Complications of Donor Plasma

The donor plasma is far less than the whole donor blood cell with the rate of adverse reactions in donors' blood components (22-23).

AFFP: apheresis fresh frozen plasma; 0.5% to 2.5%;

Rare: < 0.5%; very rare: < 0.10%; Frequent: 5% to 20%

Adverse reactions are more in first time donors and it has less vasovagal responses (24).

In contrary the whole blood donors, there are two reasons to reduce the blood volume (hypovolemia) that it has less reduction of blood volume than other donors in donors` plasmapheresis. First, an ejecting of the whole blood volume is often less than whole blood donors during donor plasmapheresis and or the loss volume will be compensated if it needed alternative and replacement fluids. Second, when plasmapheresis has been done, simultaneously donors have a long relaxation time that it was compared with the whole blood donors and allows to refill the intervascular to the interstitial capillaries with blood (24, 25-27).

There are plasma hematoma and pain of needle in the injection space of the body that is the most common complication in the donor plasma but the incidence of hematoma was not more than whole blood donors.

There are rarely allergic reactions when it was prepared to rub the iodine fluid to skin. It was an important adverse effect associated with anticoagulant citrate when used as anticoagulant in vitro system and it was part of the remaining blood components back to the donor's bloodstream. If donors have a low weight, maybe they have many symptoms of citrate (28-30).

Sometimes there are rare mild reactions to citrate (anesthesia around the mouth, tremor or tingling sensation, tingling, or feeling cold or stuffy nose) and If the patients are faced with these reactions it is not alarming but demonstrated that doctors should control this process to reduce and prevent serious symptoms of citrate toxicity. Sometimes reactions have acute forms due to citrate toxicity (muscle cramps, whole body vibration, nausea, vomiting and tetanus) that are rare in plasmapheresis but potentially have been a serious problem and disorder (31-33).

Therapeutic PlasmaPheresis or Therapeutic Plasma Exchange (TPE)

Description

Therapeutic plasma exchange or TPE are often attributed to plasma that exits from the body of patient then compensated by any kind of replacement fluid volumes to support neurmolemic situation of patients (34). Table 1 shows Indications for Therapeutic Apheresis.

Table 1- Indications for therapeutic apheresis generally accepted for reimbursement by third-party payers (8,9)

Plasma exchange
Ig*G
Myasthenia gravis
Eaton-Lambert syndrome
Goodpasture syndrome
Myeloma with renal failure
Guillain-Barre syndrome
Hemophilia with factor VIII inhibitor
Chronic inflammatory demyelinating Polyradiculopathy
Immune complex
Glomerulonephritis, rapidly progressive
Rheumatoid vasculitis
Metabolic diseases
Refsum`s diseases
Hyperlipoproteinemia, familial
Cholestasis- intractable pruritus
Diseases of unknown cause
Thrombotic thrombocytopenic
Thyroid storm
Scleroderma, refractory
Polymysoitis, refractory
Cytapheresis
Acute leukemia-debulking
Hairy cell leukemia- maintenance
Thrombocytosis, symptomatic
Chronic myelogenous leukemia, acute symptoms
Red cell exchange
Sickle cell diseases

* Immunoglobulin

What features should Pathogenic materials withdrawn by TPE must have?

There are clinical reactions to the withdrawal plasma by TPE, if the pathogen has the following properties (35):

1 – A withdrawal material must be sufficiently large and as big as macromolecules (molecular weight more than 15,000), so that it is not easily purified by a cheaper techniques such as hemofilteration or unrecoverable high flux hem-dialysis.

2- Relatively removed material has a long half-life so that in vitro removal of such substances into the body is passed much faster than the clearing way of the body called as metabolism. For example, the IgG with an approximately 21 day's half-life even if it has a treatment with immune-suppressors and stops producing new antibodies after 21 days, its decrease was at 50%.

3- A synthesis of the desired materials has a low speed. Intravascular concentration is often increased and its deletion depends on the speed of their synthesis.

4- Pathogen is an intravascular factor. The ultimate effect of TPE depends on concentration of pathogen, and also the concentration of that substance in any of the vessels and the rate of exchange between these two parts is that if each percentage of both inside and outside of the vessel is greater than the other part and the rate of exchange between the two parts is low, the effectiveness of TPE would be high.

5- The removed materials must be really toxic and resistant to conventional therapy so that its quick elimination from the extracellular fluid by TPE be possible due to the fact that plasma exchange will be time-consuming and costly (35).

At every stage of the TPE process, how much plasma should be removed?

Plasma's volume that should be removed at each stage of TPE and its performance distances, the plasma factor depends on the pathogen (36-38). Once the removed plasma (one volume plasma) has been occurred, an equal volume of plasma clearance was not 100% and about 38% of the material remains in the plasma due to the simultaneous dilution and advanced plasma in the patient during the performance of TPE process by replacement fluids. After exchanging 1.5 volumes of plasma, it remains 22% and after

replacement two volumes are 15% in considered substances (39).

If the high volume of plasma replaced, a smaller fraction of remained substances can be removed until the rate of removing decreased and it is reduced the removed volumes about 1.5-2 equivalent of the plasma volume. It should be noted that the exchange of more than one plasma volume, caused to length the processing time of exchange and decreasing the patient endurance and also increasing the cost (replacing a plasma volume about 1.5-2 hours long and by 2 to 3 equivalent of the volume exchange adds time of the process). According to decreasing the pathogen removal efficiency in volumes greater than 1.5 times of the plasma volume that estimated for each individual and adverse results of increasing the time of performing TPE process caused to increase the volume of exchange, most therapeutic plasma exchange procedures aimed to remove about 1-1.5 plasma volumes in each turns (40-48).

How often should the TPE procedure be performed?

The differences in plasma levels exchange are depends on factors such as the rate of patient illnesses, the rate of regeneration (re-synthesis) and redistribution (the transfer of desired material to the intravascular) of extra vascular substances (49). For example, the exit of IgG needs to a daily TPE process (due to the high rate of synthesis and entry into the intravascular space outside the vessel) and or in the patient who are awaiting a liver transplant, sometimes TPE is necessary to sustain life every 12 hours and generally, if a amount of new IgG production is low (created during immunosuppressive therapy), that the amount of extra-vascular exchange is about 1-2% in hours against intravascular exchange required 5 times TPE during 7 to 10 days to return 90% of initial body lift immunoglobulin (50-51).

How much plasma exchange is in a TPE process?

General recommendations of AABB for the TPE process indicated that in cases where there is its IgG, the exchange of plasma volume estimated about 1-1.5 equivalent in each person and once every 2-3 days with total exchanging in 3-5 days (52). It should be, of course, noted that the frequency of plasma exchange may change according to the patient's clinical responses and laboratory testing results. Table 2 shows the final target of plasmapheresis therapy (53).

Table 2- The final target of plasmapheresis therapy (26)

Substance to Remove	Treatment Volume (ml/kg)	Treatment Interval (in hours)	Treatment Endpoint
Autoantibodies	40-60	24-48	Four to six treatments
Immune complexes	40-60	24-48	Treat for response
Paraproteins	40-60	24	Treat for response
Toxins	40-60	24-72	Treat for response
Thrombotic thrombocytopenic purpura/ hemolytic uremic syndrome	40	24	Treat to establish remission
Immunologic rebound	40-60	24-48	Two to three treatment followed by immunosuppressive medication

Which kind of Fluid replacement used in TPE?

Crystalloid fluids, among which the saline fluids are used most widely, would suffice when 500-1000 cc of plasma is removed through a manual plasmapheresis (54). Especially TPE is used to solve hyperviscosity as well. Crystalloid fluids alone is not sufficient when it discharging more than one liter of plasma volume because it caused hypoproteinemia and coagulopathy by prescribing in high volumes in patient.

Therefore, they need to prescribe crystalloid fluid volume for discharging equivalent to 2-3 volumes of plasma that it is for the rapid departure of crystalloid fluids from vessels, and led to hypotension and edema by spreading to the extra-vascular vessels (55-57).

However, it has the necessary effectiveness for un-transmission of diseases and its low cost. Now saline has been used with albumin as some of (30-40%) the fluid replacement in plasma exchange. Additionally it is useful in the preparation of anticoagulant fluid and instruments (58).

Five percent albumin is used mostly in replacement fluid. For most patients, this liquid plays an important rule as hyper-oncotic one and forced to flow this pure fluid to intravascular and make a mild anemia. To prevent from mild anemia must be made 5% albumin to 4-4.5 % albumin by saline. Many patients could tolerate to control this disease by exchanging 25-50% of the volume of saline (59).

An important advantage of albumin is impossible transmission of virus agents'. Albumin prescribed easily to patients that it is not required to test the blood group and without needing to melt or prepare it. The patients have rarely been reported an uncommon albumin reactions but pyogenic materials have fever and prekallikrein activated reactions (60-62).

Currently, based on standard method it is replaced with to use 5% albumin fluid (amount of 60-70% the plasma volume) and remained replaced with crystalloid such as normality saline (63).

FFP is one of another replacement colloid fluid that sometimes is used as replacement fluid for patients with TTP disease which needs replacement therapy of special protein plasma in accessing the metalloproteases von Willebrand. In the TTP treatment, it is formed the replacement volumes as an amount of 60-70% FFP and remained for saline (64).

Synthetic colloid fluids such as HES can also be used to replace all or part of the liquid. It is derived from vegetable starch containing scratch of macromolecules that saline is added to the colloidal fluid. An advantage of this fluid is low cost and non- transmission to others. The disadvantages are allergic reactions to HES that can occur in a small number of patients (64-66).

Therefore HES used as a replacement fluid in patient with reaction to albumin or FFP and sometimes religious peoples are not tending to access blood derivation by following plasma exchanges (67).

Enrichment of replacement fluids (saline, albumin, HES) can reduce the incidence of hypocalcaemia due to citrate toxicity with calcium. The volume of calcium should be checked in large volumes (1-2 volumes) in plasmapheresis therapy for several sessions especially after each plasmapheresis session when plasma is used as a replacement therapy that have a large amounts of citrate (68). The best time to control the amount of calcium is a few hours after plasma exchange or in the next morning after and before the replacement of plasmapheresis session (69). Table 3 shows advantages and disadvantages of any replacement fluids in TPE.

Table 3- Advantages & disadvantages of any replacement fluids in TPE*

Replacement fluid	Advantages	Disadvantages
Cr**ystalloids**	Low cost	2-3 volumic required
	Hypoallergenic	Hypo-oncotic
	No viral risk	No coagulation factors
		No immunoglobulins
Albumin	Iso-oncotic	High cost
	No contaminating	No coagulation factors
	"inflammatory mediators"	No immunoglobulins
	No viral risk	
Hydroxyethyl starch	Moderate cost	No coagulation factors
	Iso-oncotic	Long-term residual
	No contaminating	Levels of HES
	"inflammatory mediators"	Contraindicated with renal failure
		Possible coagulopathy
Plasma	Maintins normal levels of:	Viral transmission risk
	Immunoglobulins	Citrate load
	Complement	ABO incompatibility risk
	Antithrombin	Allergic reaction
	Other proteins	Sensitization

* Therapeutic Plasma Exchange

What type of anticoagulant and how much is needed?

Citrate, heparin, or both combinations can be used during plasmapheresis to prevent the blood circulation outside the body. Patient should be carefully evaluated in terms of the ability against citrate or heparin, to determine the best anticoagulant materials. Other considerations include how to assess intravascular volume of patient, the type and volume of replacement fluid, intravenous access and blood flow rate in the catheter (70-72).

Citrate has been accessed in 4 shapes as follows (73):

ACD-A, ACD-B, sodium citrate and concentration of tri-sodium citrate

ACD-A has been prescribed in the amount of 9:1 to 14:1 that including 3% sodium citrate and also used in the determined rate of anticoagulation (WB/ACD) in blood. In contrast to ACD-A, ACD-B is including 2% sodium citrate that usually used at the rate of 6:1 to 9:1 from WB/ACD to reduce the citrate toxicity risk.

Unlike heparin, citrate has not any quick metabolisation. It has the exact half-life of approximately 90 minutes and thus leads to have systemic anticoagulant effects in patients (74).

When these particular characteristics are replaced with non-plasma fluids during plasmapheresis (e.g. albumin 5% or HES) it is difficult to remove the coagulation factors in plasma and their replacement. Heparin can be used alone or in combination with ACD-A and or ACD-B. When they use the combination of heparin and citrate to create effective anticoagulant effects, heparin is needed less and less ACD volumes. Therefore, this combination reduced the incidence of systemic toxicity citrate anticoagulation due to minimizing the heparin consumes alone and also reduces the whole volumes of the injected fluid during TPE process (75).

In vitro, what kind of tests need during the TPE process?

Laboratory studies are based on the ultimate goal of therapy and pursuiting of the reduction factor. Plasmapheresis can be evaluated and studied an early CBC consideration, electrophoresis serum protein, electrolytes and coagulant factors (76). More detailed laboratory analysis is required when desired a large number of low- frequency plasma exchange. We should give several hour chances to body to follow the necessary laboratory TPE tests until to shift extra- and intra-vascular fluids to reach equilibrium and bleeding has to be taken especially in biochemistry (77).

TPE Indications

The appropriate use of therapeutic plasma exchange (TPE) based on two American societies for aphaeresis (ASFA) and American Association of Blood Bank (AABB) organizations rules are as follows (78-83):

Before reading the table, it is necessary to be familiar with the following concepts:

Group I: The standard acceptable therapy of TPE in first-line for these diseases.

Group II: There is sufficient evidence to suggest the efficacy of plasma exchange therapy as second-line or adjunctive therapy.

Group III: There are inconclusive evidence in efficacy of plasma exchange therapy with uncertain risks in final therapy way.

Group IV: There is lack of efficacy in controlled TPE trials.

Table 4, 5, 6 shows Plasmepheresis Therapy Indications.

Table 4- Plasmepheresis therapy indications

Disease	Procedure	Indication Category
Neurologic dirorders		
Chronic inflammatory demyelinating Polyradiculoneuropathy (CIDP)	Plasma exchange	I
Acute inflammatory demyelinating Polyradiculoneuropathy (AIDP or Gullian-Barre syndrome)	Plasma exchange	I
Myasthenia gravis	Plasma exchange	I
Lambert-Eaton myasthenia gravis	Plasma exchange	II
Multiple sclerosis and related disorders	Plasma exchange	III
Acute fulminant central nervous system demyelination		III
Relapsing or progressive	Plasma exchange	III
Paraneoplastic Neurologic syndrome	Immunoadsorption	III
Paraproteinemic polyneuropathies	Plasma exchange	I
Demyelinating polyneuropathy	Immunoadsorption	III
Ig*G/Ig*A	Plasma exchange	II
	Immunoadsorption	III
Polyneuropathy with Ig*M	Plasma exchange	II
(±Waldenstrome`s macroglobulinemia)		
Cryoglobulinemia with polyneuropathy	Plasma exchange	III
	Plasma exchange	III
Multiple myeloma with polyneuropathy	Plasma exchange	IV
POEMS syndrome		
Systematic (AL) amyloidosis	Plasma exchange	III
Inflammatory myopathies	Leukapheresis	IV
Polymyositis or dermatomyositis	Plasma exchange	III
	Leukapheresis	IV
Inclusion- body myositis	Plasma exchange	III
Rasmussen encephalitis	Plasma exchange	III
Stiff-person syndrome	Plasma exchange	III

Syndrome`s chorea/pediatric autoimmune pneuropsychiatric associated with streptococcal infections (PANDAS)	Plasma exchange	II
Hematologic disease	Plasma exchange (recipient)	II
ABO-incompatible hematopoietic cell transplant	Erythrocytapheresis	
RErytherocytosis/polycythemia vera	Cytapheresis	II
Leukocytosis and thrombocytosis	Plasma exchange	I
Thrombotic thrombocytopenic purpura	Plasma exchange	I
	Red cell exchange	I
Post-transfusion purpura	Plasma exchange	II
Sickle cell diseases	Plasma exchange	II
Myeloma/paraproteins/hyperviscosity	Plasma exchange	II
Myeloma/acute renal failure	Plasma exchange	III
Coagulation factor inhibitors	Photopheresis	I
Aplastic anemia/pure red cell aplasia	Leukapheresis	III
Cutaneous T-cell lymphoma	Plasma exchange	III
	Plasma exchange	III
Hemolytic disease of the newborn	Immunoadsorption	III
Platelet alloimmunization and refractoriness	Red cell exchange	III
Malaria/babesiosis		
Renal and metabolic disease	Plasma exchange	I
Antiglomerular basement membrance antibody disease (Goodpasture`s syndrome)	Plasma exchange	II
	Plasma exchange	III
Rapidly progressive glomerulonephritis		
Hemolytic-uremic syndrome	Plasma exchange	IV
Renal transplantation	Plasma exchange	III
Rejection	Plasma exchange	III
Presensitization	Plasma exchange	III
Recurrent focal glomerulosclerosis		
Heart transplant rejection	Photopheresis	III
	Plasma exchange	III
Acute hepatic failure	Selective adsorption	I
Familial hyper cholesterolemia	Plasma exchange	II
Overdose poisoning	Plasma exchange	III
Phytanic acid storage disease (Refsum`s)	Plasma exchange	I
Autoimmune and rheumatic disease	Plasma exchange	II
	Immunoadsorption	II
Cryoglobulinemia	Plasma exchange	III
Idiopathic thrombocytopenia purpura	Plasma exchange	III
Raynaud`s phenomenon	Plasma exchange	III
Vasculitis	Immunoadsorption	II
Autoimmune hemolytic anemia	Lymphoplasmapheresis	II
Rheumatoid arthritis	Plasma exchange	IV
Scleroderma/progressive systematic sclerosis	Plasma exchange	III
Systematic lupus erythematosus	Plasma exchange	III

Category I = Standard acceptable therapy

Category II = sufficient evidence to suggest efficacy usually as adjunctive therapy

Category III = inconclusive evidence of efficacy or uncertain risk/benefit ratio

Category IV = lack of efficacy in controlled trails.

*Immunoglobulin

Table 5- Indications of plasmapheresis therapy based on ASFA and AABB classification in neurologic diseases

Disease	Procedure	IndicationCategory
Guillian-Barre syndrome	Plasma exchange	I
Chronic inflammatory demyelinating	Plasma exchange	I
Polyradiculoneuropathy	Plasma exchange	I
Polyneuropathy with IgG/IgA monoclonal protein	Plasma exchange	II
Polyneuropathy with IgM monoclonal protein	Plasma exchange	I
Myasthenia gravis	Plasma exchange	III
Stiff-person syndrome	Plasma exchange	II
Lambert-Eaton myasthenia syndrome	Plasma exchange	III
Paraneoplastic Neurologic syndrome	Plasma exchange	III
Polymyositis or dermatomyositis	Leukapheresis	IV
Multiple sclerosis	Plasma exchange	III
Idiopathic inflammatory demyelinating disease	Plasma exchange	II
Refsum`s disease	Plasma exchange	III
Rasmussen`s encephalitis	Plasma exchange	III
Sydenham`s chorea	Plasma exchange	II
PANDAS	Plasma exchange	II

PANDAS = Pediatric Autoimmune Pneuropsychiatric Disorders Associated with Streptococcal Infections

Table 6- Indications of plasmapheresis therapy based on ASFA and AABB classification in blood disease and disproteinemia

Disease	AABB category	ASFA category
ABO-incompatible marrow transplant	II	II
Aplastic anemia	III	III
Autoimmune hemolytic anemia	III	III
Coagulation factor inhibitors	II	II
Cryoglobulinemia	II	II
HELP syndrome (postpartum)	NR*	NR**
Hemolytic-uremic syndrome	III	III
Hyperviscosity/multiple myeloma	II	II
Immune thrombocytopenia purpura	II+	II+
Platelet alloimmunization	III	III
Post-transfusion purpura	I	I
Pure red cell aplasia	III	III
Red cell alloimmunization	III	III
Thrombocytopenic purpura	I	I

*Disorder not ranked by either AABB or ASFA.

**This disorder is ranked only in context of staphylococcal protein A

Immuniadsorption

AABB = American Association of Blood Banks; ASFA = American Society for Apheresis; Category I = Standard acceptable therapy; Category II = sufficient evidence to suggest efficacy usually as adjunctive therapy; Category III = inconclusive evidence of efficacy or uncertain risk/benefit ratio; Category IV = lack of efficacy in controlled trails; HELLP = Hemolysis, elevated liver enzymes, and low platelet.

Conclusion

Plasmapheresis, which is defined as the removal of plasma, can be either "adjusted plasma" or "exchange of plasma". Plasmapheresis is currently used as a therapeutic modality in a wide array of conditions. Generally, plasmapheresis is used when a substance in the plasma, such as immunoglobulin, is acutely toxic and can be efficiently removed. Myriad conditions fall under this category, including neurologic, hematologic, metabolic, dermatologic, rheumatologic, and renal diseases, as well as intoxications, that can be treated with plasmapheresis.

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