Journal of Clinical Apheresis

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Journal of Clinical Apheresis ASFA

RESEARCH ARTICLE OPEN ACCESS

Can Plasma Exchange Be Used to Lower the Circulating Burden of Microplastics in Human Patients?

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Received: 12 February 2026 | **Revised:** 28 April 2026 | **Accepted:** 11 May 2026

Keywords: apheresis | blood | microplastics | patients | plasmapheresis | therapeutics | toxins

ABSTRACT

Microplastic particles (MP) from industrial products and degradation of plastic waste are ubiquitous in our environment and convey serious potential risks to human health. There is currently no established method for removing MP from the human body. MP circulate in the blood and have been found in the eluate plasma from double filtration plasmapheresis procedures, suggesting that plasmapheresis could be used to remove MP from the body. We have measured circulating MP in the blood of 114 patients before and after 174 1-plasma-volume therapeutic plasma exchange (TPE) procedures using the Spectra Optia Apheresis System and have shown that circulating MP are reduced by TPE. The reduction is obscured at low starting levels of MP because of leaching of MP back into the circulation from the plastic apheresis tubing set. Nonetheless, this is the first demonstration that it is possible to reduce circulating MP in human patients.

1 | Introduction

Water-insoluble plastic waste particles, ranging in size from 1 μm to 5 mm (*micro*plastics) or <1 μm (nanoplastics) from the degradation of paints, cosmetics, medical devices, packing materials, and diverse industrial processes, are ubiquitous in oceans, freshwater, and ambient air [1–3]. Emerging evidence describes the entry of *micro*plastics (MP) into the human body through ingestion, dermal contact, and inhalation [1–4]. MP exposure may even result from exposure of blood to plastic tubing during intravenous injections during medical interventions [5]. The potential for MP to contribute to the development of cardiovascular, endocrine, cerebral, pulmonary, reproductive, and other organ system dysfunctions underscores the health risks posed by this class of environmental pollutants [3–9].

There is currently no established method for removing MP from the body, apart from the ambient excretion of some gastroenterically absorbed MP in urine and feces [8, 10]. However, once ingested through the skin, lungs or GI tract, MP may be transported throughout the body by various routes, including the bloodstream [1, 7]. They may then contribute to adverse effects in the digestive, respiratory, cardiovascular, immune and other systems where they contribute to oxidative stress, inflammation and metabolic disorders [11].

A recent European report described the detection of MP in the concentrated eluate of plasma collected during therapeutic double filtration plasmapheresis [12]. Although this observation was qualitative, it suggested, for the first time, that plasmapheresis techniques could be employed to remove MP from the human

body. In the present study, we sought to determine whether therapeutic plasma exchange (TPE) could be employed to significantly reduce the circulating MP burden in human subjects.

2 | Methods

Medically stable patients who were to undergo outpatient TPE were offered, and consented to, free blood testing for MP before and after a TPE session. The consent document for therapeutic plasma exchange includes a clause, that patients can accept with a check mark, that states that blood test results may be used in anonymized fashion for research studies. No specific research procedures were used in these plasma exchange treatments; therefore, IRB approval was not sought. Venous blood samples (100 μL) obtained from antecubital blood (via fingerstick in two patients), immediately before and after TPE, were applied to dedicated test cards and allowed to dry. The cards were sent by mail to Arrow Lab Solutions, Burton, MI, for the *PlasticTox* test [13–16]. This is a proprietary, commercially available assay for MP developed by SV Biotech BV, Leiden, the Netherlands, and made available in the United States through Arrow Lab Solutions. This assay involves drying a 100 μL blood sample on a dedicated sample card and sending it by mail to a central laboratory. There, the sample is subjected to a proprietary process that includes isolating the MP from proteins and cellular materials, staining the MP with Nile Red, and quantitating the MP using fluorescence microscopy. This test can detect as little as a single MP particle 1 μm in size. Although Arrow Lab Solutions claims that the assay and its method have been validated by a CLIA/COLA certified high-complexity reference laboratory (World Wide Clinical Labz, Detroit, MI), the validation data are maintained as proprietary by the company.

All TPE procedures were performed using a Spectra Optia Apheresis System (Terumo BCT, Lakewood, Colorado) using software version 11 or 12. All TPE were single-plasma-volume procedures with 5% human albumin (Albumin 5%, OctaPharma, Paramus, New Jersey) as the colloid replacement fluid and ACD-A (Anticoagulant Citrate Dextrose Solution USP (ACD) Solution A, Terumo BCT, Lakewood, Colorado) as the anticoagulant. Calcium gluconate (1000 mg/10 mL, Fresenius Kabi, Lake Zurich, Illinois) was directly added to the albumin bottles (5 mL or 1.12 mmol in each 500 mL bottle of albumin). Whole blood flow was at 80 to 100 mL/min, with citrate infusion maintained at 0.8 mL/min/L of whole blood volume. Circulating levels of MP in patients' blood before and after plasma exchange were compared using the Wilcoxon Signed Rank Test (SigmaPlot version 11, Systat Software Inc., Chicago, IL). For analysis, effect sizes at each of four starting levels of MP (0–9, 10–19, 20–29 and ≥ 30 MP/100 μL) were calculated using Cohen's d as $d = \mathrm{M1} - \mathrm{M2}/$ pooled SD where M1 is the pre-TPE mean, M2 is the post-TPE mean and pooled SD is $\sqrt{\left[(\mathrm{SD}_{\mathrm{pre}})^2 + (\mathrm{SD}_{\mathrm{post}})^2\right]/2}$ [17].

3 | Results

3.1 | Patients

Men and women who underwent TPE were of similar ages. Men accounted for approximately 58% of patients and 63% of TPE (see Table 1). Overall, 174 TPE were performed on 114 patients. Twenty-five patients were studied twice, two were studied three times, three were studied four times, and one was studied five times. All procedures took place in Functional Medicine outpatient clinics. Most procedures were performed for support of longevity. Other major indications included postural orthostatic tachycardia syndrome, myalgic encephalomyelitis, and long Covid. There were no significant adverse effects.

3.2 | Circulating MP

The effect of TPE on circulating MP level is shown in Table 2. Starting (pre-TPE) MP ranged from 0 to 123 per 100 μL of blood, and ending (post-TPE) MP ranged from 0 to 59 per 100 μL of blood. In 100 TPE with starting MP 0–9/100 μL, mean ± SEM MP was 4.400 ± 0.283/100 μL pre- and 14.400 ± 1.486/100 μL post-apheresis ($p < 0.001$, Wilcoxon signed rank test). This suggests that MP was actually added to the circulation during these TPE procedures.

In 33 TPE with starting MP 10–19/100 μL, mean ± SEM MP was 13.848 ± 0.502/100 μL pre- and 11.727 ± 2.067/100 μL post-apheresis ($p = 0.062$), suggesting that the numbers of MP added to and removed from the circulation during TPE were balanced.

In 20 TPE with starting MP 20–29/100 μL, mean ± SEM MP was 23.600 ± 0.663/100 μL pre- and 16.100 ± 3.410/100 μL post-apheresis ($p = 0.040$). This demonstrates net removal of MP from the circulation during TPE.

Finally, in 21 TPE with starting MP ≥ 30/100 μL, mean ± SEM MP was 52.190 ± 5.129/100 μL pre and 21.095 ± 3.424/100 μL post-apheresis ($p < 0.001$). This further indicates net removal of MP from the circulation during these TPE procedures.

3.3 | MP Contamination in the Tubing Set

(Please refer to Table 3). MP were measured by *PlasticTox* in saline sampled from the inlet tubing after priming (before connecting it to the patient) in order to assess whether MP particles were introduced into the system at the proximal end of the apheresis tubing set. These measurements amounted to (mean ± SEM) 16.7 ± 5.4 MP particles per 100 μL. Saline in the blood warmer tubing that is, at the distal end of the apheresis tubing set, contained 18.8 ± 6.9 MP particles per 100 μL before warming and 25.0 ± 12.3/100 μL after warming to 41°C for 52 min. A sample of saline obtained from a 1 L bag used for priming the system contained 11 MP particles per 100 μL. Thus while MP were removed from patients' circulating blood during TPE, MP were

TABLE 1 | Patients and procedures.

	Male	Female	Total
Subjects	66	48	114
Age range (mean ± SEM)[a]	18–95 (57.3 ± 2.2)	21–81 (52.9 ± 2.17)	18–95 (55.8 ± 1.57)
TPE	109	65	174

[a](male vs. female) $p = 0.151$ (Mann–Whitney rank sum test).

TABLE 2 | Effect of plasma exchange on circulating MP level.

Starting range (MP/100 μL)	0–9	10–19	20–29	≥30
n	100	33	20	21
Mean (median) ± SEM pre-TPE	4.4 (4.0) ± 0.28	13.8 (13.0) ± 0.5	23.6 (22.0) ± 0.66	52.2 (44.0) ± 5.13
Mean (median) ± SEM post-TPE	14.4 (8.5) ± 1.49	11.8 (8.0) ± 2.1	16.1 (9.0) ± 3.4	21.1 (18.0) ± 3.4
p (pre vs. post)[a]	< 0.001	0.062	0.040	< 0.001
Effect size[b]	−0.95	0.15	0.25	1.26

[a]Wilcoxon signed rank test.
[b]Cohen's *d*: [(MEAN$_{pre}$ − MEAN$_{post}$)/pooled SD] where pooled SD = $\sqrt{\left[(SD_{pre})^2 + (SD_{post})^2\right]/2}$.

TABLE 3 | MP in saline sampled from fluid bag and apheresis tubing set.

Saline sampling site	MP/100 μL[a]
1 L saline bag	11 MP/100 μL
Inlet tubing post prime ($n = 3$)	16.7 ± 5.4/100 μL[b]
Blood warmer tubing post prime ($n = 5$)	18.8 ± 6.9/100 μL[b]
Blood warmer tubing, 52 min at 41°C ($n = 5$)	25 ± 12.3/100 μL[b]

[a]Mean ± SEM.
[b]$p = 0.996$ (Kruskal-Wallis one way analysis of variance on ranks).

likely introduced, simultaneously, into the circulation by leaching from the plastic surfaces of the fluid bags and the tubing set.

4 | Discussion

We have demonstrated that a 1-plasma-volume therapeutic plasma exchange can decrease the circulating burden of MP in patients' blood. This is the first report of purposeful removal of MP from the human body using a therapeutic procedure. We employed a commercially available assay, *Plastictox*, for quantitation of MP in patient blood samples.

Human beings are exposed to environmental MP from a variety of sources and may unwittingly ingest them via inhalation, cutaneous absorption, or dietary consumption [1, 4, 9, 18]. These MP may pose significant health risks, including contributions to cardiovascular, metabolic, neurological, and endocrine disorders [3, 4]. MP have been identified in circulating human blood [15], and have recently been identified in the eluate plasma collected during double filtration plasmapheresis [12]. These findings suggested the possibility of employing plasmapheresis techniques to help to deplete environmental pollutants, such as MP, from the body [19]. Herein, we extend these observations and demonstrate that TPE can indeed lower the circulating burden of MP.

In measuring circulating MP before and after TPE, we perceived an apparent threshold effect whereby blood MP levels were reliably reduced by TPE from higher starting levels but not necessarily from lower starting levels. We divided our data into four starting ranges for analysis to see whether we could determine a threshold. At the lowest starting level of MP (0–9/100 μL) the mean post-TPE level of circulating MP was actually higher than the mean pre-TPE level. Based on the report by Mou et al. [5] who described the leaching of MP from medical fluid (e.g., saline) bags and tubing into the fluids that were inside them, we were concerned that we were seeing a similar phenomenon. At an intermediate starting level (10–19/100 μL), the removal and apparent addition of MP appeared balanced. At the next starting level of circulating MP (20–29/100 μL) a clear and statistically significant reduction of circulating MP could be demonstrated, but the effect was small. It was only at a starting MP level of ≥30/100 μL that the MP level was convincingly lowered by TPE, with a statistically significant result and a large effect. Mou et al. [5] reported that microplastics leached from infusion bags amounted to 1.0 ± 0.7 μg/L of polyethylene and polypropylene fiber fragments, and that the initial 12 mL of saline infused through plastic tubing contained 8.4 ± 3.6 μg/L of primarily polyvinyl chloride particles and fibers. Our observations suggest that leaching of MP from the clinical plastic tubing and fluid bags used in our procedures affected the results measured in our patients. Only at a starting level of ≥30/100 μL was the removal of circulating MP sufficient to overcome the amount of MP infused during the procedure. Manufacturers of clinical plastic tubing for intravenous therapies and apheresis will need to pursue new materials that do not cause their products to leach MP into patients' circulation [20].

The clinical significance of the observations reported herein largely depends on whether microplastic particles freely move between tissues and the vascular system. That would imply that plasma exchange may be used to deplete the accumulated burden of microplastics in the body. The demonstration of microplastic particles in human blood indicates that, after absorption, inhalation or ingestion, they make their way from tissue spaces to the vascular system [12, 15]. Earlier studies in mice have suggested that microplastics are transported among tissues through the vascular system [21]. While potentially encouraging, these observations do not establish the existence of a dynamic equilibrium between microplastics deposited in tissues and the vascular system. Thus, although plasma exchange can remove microplastic particles that have made their way to the bloodstream from lung, gut, and other organs, it is not possible, at this time, to assess whether plasma exchange provides an efficient therapeutic approach to the microplastic burden in the body. There is no accurate basis for estimating how many plasma exchange procedures would theoretically be required to significantly reduce the total body burden of microplastics. For patients who present with a high circulating microplastics burden (≥30/100 μL), plasma exchange may somewhat help to

protect against ongoing accumulation of microplastics in the body. Public health measures to lessen exposure to MP, coupled with TPE in selected patients, may help to mitigate the health challenges of MP to the public at large.

Our study has certain limitations. Because the detection threshold of the assay that we used to measure circulating microplastics is 1 μm, we are not able to determine whether plasma exchange removes nanoplastic particles from the bloodstream. In addition, the apparent confounding effect of the leaching of MP from clinical tubing into the system limits our ability to comment on the effectiveness of plasma exchange in removing MP from the blood at lower starting levels. Nonetheless, MP are susceptible to removal by TPE.

5 | Conclusion

Microplastics circulate in the human bloodstream where they are susceptible to removal by therapeutic plasma exchange. A 1-plasma-volume therapeutic plasma exchange can lower the circulating burden of MP in human patients.

Funding

The authors have nothing to report.

Ethics Statement

Blood draws were performed in the normal course of therapeutic procedures. No IRB approvals were required.

Consent

Patients consented to being tested for micro- and nanoplastics and for the anonymous use of their results in this publication.

Conflicts of Interest

Seven of the authors are associated with Circulate Health, a company that provides contract therapeutic plasma exchange services for private clinics. The other four authors are officers of the four clinics in which the data were gathered.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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