Scholastic Medical Sciences

Research Article

Volume 3 Issue 1 January 2026

Autologous Cytokine Therapy and IL-10 Recovery in Cancer Patients: A Case Series on Immunoregulatory Restoration After Conventional Treatments

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Received: November 27, 2025; **Published:** December 05, 2025

Abstract

Objective: Interleukin-10 is a key cytokine involved in the regulation of inflammatory responses and the maintenance of immune homeostasis. In patients undergoing intensive oncological and immunomodulatory treatments, its regulatory function may become markedly impaired, contributing to a persistently pro-inflammatory microenvironment. The objective of this study is to evaluate whether autologous cytokine therapy can modulate and restore IL-10 levels in a protective manner in patients exhibiting a pronounced post-treatment decline in this regulatory cytokine.

Materials and Methods: Five patients with different type of cancer who undergone conventional treatments (surgery, chemo/radiotherapy, or immunotherapy) were included. Serum IL-10 levels were measured at three predefined time points: before conventional treatments (T0), at the completion of these treatments (T1), and after a cycle of autologous cytokine therapy (T2). The autologous therapy consisted of cytokine fragments extracted from the patient's own blood and administered following a standardized protocol.

Results: All patients showed a significant reduction in IL-10 levels after conventional treatments (T1), with mean decreases exceeding 80% compared to baseline. Following autologous cytokine therapy (T2), IL-10 levels increased uniformly and consistently across the cohort, with final values approaching or surpassing baseline levels, thereby restoring a cytokine profile compatible with a functional regulatory response. The observed dynamics suggest that autologous cytokine therapy may support the re-establishment of immune regulation impaired by intensive oncological treatments, promoting a recovery of IL-10.

Conclusion: These preliminary findings suggest that autologous cytokine therapy may serve as a useful complementary strategy to restore immunoregulatory function in oncological patients undergoing intensive standard treatments.

Keywords: cytokine, cancer, inflammation, immunomodulation, cytokine modulation, biological therapy, autologous therapy, low-dose immunotherapy.

Abbreviations

Interleukin (IL)

Introduction

Interleukin-10 (IL-10) is a key cytokine in the regulation of immune responses, traditionally regarded as a major anti-inflammatory mediator capable of limiting tissue damage driven by excessive immune activation (1; 2). It is produced by multiple innate and adaptive immune cell populations—including monocytes/macrophages, dendritic cells, regulatory T cells and regulatory B cells—and modulates the expression of pro-inflammatory cytokines, co-stimulatory molecules and effector functions, thereby maintaining a balance between pathogen clearance and prevention of immunopathology (3; 4).

More recently, the role of IL-10 has been reframed in a "dual" perspective: beyond dampening harmful inflammatory responses, IL-10 can sustain protective immune functions, for example by supporting survival and metabolic fitness of CD8+ T cells under conditions

of chronic stress, including oncologic settings (5; 6). In several experimental models, appropriate levels of IL-10 have been associated with fine-tuning of the immune microenvironment, limiting cytokine storm while preserving—or even enhancing—effective anti-tumor and anti-infective responses (4; 7). In this sense, IL-10 emerges as an immunological "thermostat" balancing activation and tolerance, rather than a mere brake (8; 9).

At the clinical level, recombinant IL-10 and IL-10–based biologics have been evaluated in a range of inflammatory and neoplastic conditions, showing biological activity and acceptable safety, but with variable efficacy, partly due to the complexity of the cytokine network and the narrow therapeutic window (10; 1). In parallel, translational studies have demonstrated that changes in circulating or tissue IL-10 levels may correlate with different prognostic patterns depending on disease context: in some settings, high IL-10 is associated with immunosuppression and disease progression, whereas in others it is linked to protection from inflammatory damage and improved clinical outcome (5; 4; 7).

Within this framework, the concept of "protective IL-10" is particularly relevant. IL-10 production during the resolution phase of inflammation is crucial for orderly shutdown of the immune response and restoration of tissue homeostasis; its presence at adequate concentrations can therefore be interpreted as a marker of a functional regulatory response rather than simply of immunosuppression (4; 11). Consequently, therapeutic interventions capable of modulating IL-10—either reducing excessive, non-functional levels or, conversely, restoring protective concentrations after aggressive treatments such as major surgery, chemotherapy, radiotherapy or strong immunotherapies—may help rebuild a cytokine profile more favorable to clinical recovery.

Autologous cytokine-based therapies, which employ extracts derived from the patient's own plasma and processed cytokine fractions, have emerged in this context as strategies potentially able to act at a regulatory level, with an impact closer to physiological modulation than to high-dose exogenous administration (12; 1). The working hypothesis is that such interventions may modulate IL-10 circuits by restoring levels compatible with its role as an anti-inflammatory "shield" and as a supporter of tissue repair after the immunological impact of standard therapies. Based on this premise, the present study analyzes IL-10 dynamics in patients with different underlying diseases, previously exposed to conventional oncologic or immunomodulatory treatments and subsequently treated with autologous cytokine therapy, with the aim of assessing whether this approach can exert a functional stimulatory and protective effect on the cytokine profile.

Use of autologous cytokine fragments represents a non-conventional therapeutic approach: patient blood serves as the source, and cytokines are purified and fractionated into α and β subunits for personalized therapy (13). This strategy offers advantages in immunogenicity, precision, and adaptation compared to recombinant cytokines or monoclonal antibodies (12).

Based on this rationale, we conducted an observational study in a small cohort of patients who had undergone conventional oncologic and immunomodulatory treatments, assessing sequentially the trend of serum IL-10 levels before standard therapies, after their completion, and at the end of a subsequent cycle of autologous cytokine therapy. By evaluating serum IL-10 levels at three time-points—before standard treatments (T0), after their completion (T1), and at the end of the autologous cytokine therapy cycle (T2)—we aim to determine whether this intervention can functionally and protectively modulate the IL-10 profile, thereby contributing to the restoration of a disrupted immunoregulatory balance.

Materials and Methods

1.1 Study Design

The present work is a descriptive observational study aimed at evaluating the modulation of IL-10 levels in patients undergoing autologous cytokine therapy following conventional oncological and/or immunomodulatory treatments. Five subjects, suffering from different underlying pathologies, were included. All subjects had previously undergone major therapeutic interventions such as surgery, chemotherapy, radiotherapy, and/or non-autologous immunotherapy (Table 1).

s.no	Subject	Age	Gender	Disease	1st treatment	2nd treatment	3rd treatment
1	44	F	Ductal carcinoma	Surgery	Radiotherapy	Immune therapy	
2	48	F	Breast cancer	Surgery	Chemotherapy	-	
3	59	M	Prostate carcinoma	Radiotherapy	Inhibitor	-	
4	45	F	Ovarian carcinoma	Surgery	Chemotherapy	-	
5	52	M	Gastric cancer	Surgery	Chemotherapy	-	

Table 1: Clinical characteristics of the five subjects included in the study, indicating age, sex, underlying pathology, and the conventional oncological/immunomodulatory treatments performed prior to the initiation of autologous cytokine therapy.

A cytokine panel screening was performed for each subject using a serum assay with the ELISA technique (Table 2).

All subjects subsequently underwent autologous cytokine therapy according to a standardized internal protocol. Briefly, the patient's peripheral venous blood was drawn and processed to obtain a fraction rich in autologous cytokines. In this study, a nuclear (non-membrane) fraction was used with the specific intent of functionally stimulating the IL-10-mediated regulatory circuits.

The detailed technical procedure related to plasma processing, cytokine extraction, fractionation, and autologous therapy preparation is described in Section 2.2.

The autologous cytokine therapy was administered in repeated cycles, following a schedule established by the protocol, via the intramuscular route. No new cycles of radio-/chemotherapy or conventional immunotherapies were initiated during the period of treatment with autologous cytokines, in order to reduce the risk of confounding factors on the trend of IL-10 levels.

Subject	Screening	Range (Pg/Ml)	T0	T1	T2
1	IL -10	11-1330	800	20	900
2	IL -10	"	740	70	680
3	IL -10	"	520	200	800
4	IL -10	"	870	190	1100
5	IL -10	"	430	19	790

Table 2: Serum IL-10 levels measured at the three predefined time points (T0: before conventional treatments; T1: after conventional treatments and before cytokine therapy; T2: after autologous cytokine therapy). Values are reported for each subject together with the reference range.

1.2 Blood Sampling and Cytokine Extraction

Extraction of autologous cytokines followed a multi-phase procedure. After venous blood collection, plasma was separated by decantation. For each cytokine, the following steps were performed: suspension in solution, addition of magnetic microspheres specific to the cytokine, magnetic separation, removal of microspheres, disruption of molecular chains via centrifugation, separation of α helices and β sheets, dilution, stabilization, and storage of the preparation (14; 15).

1.2.1 Cytokine Extraction: Phase 1

The skin was disinfected with cotton soaked in antiseptic solution. Venipuncture was performed with a sterile 10 mL syringe and a 20G/0.9 mm needle, collecting 10 cc of blood. Smaller-gauge needles were avoided to prevent cytokine damage.

The sample was left to decant vertically for 5–48 hours at room temperature (15–25 °C), or in a temperaturecontrolled device for the same period. Decantation was chosen instead of centrifugation, since the latter would irreversibly damage cytokines, compromising the final product.

After decantation, the supernatant plasma (~5 cc) was collected with a micropipette under sterile conditions. Cytokines of interest are in the intermediate layer between plasma and serum. Collecting 1–2 mL of serum ensured retrieval of cytokines without interfering with the activity of magnetic beads. Without this step, extraction of the intermediate cytokine-rich fraction would be incomplete.

1.2.2 Cytokine Extraction: Phase 2

Plasma obtained by decantation was suspended in 0.9% NaCl solution containing magnetic microspheres coated with specific ligands for the target cytokine.

For separation, a secondary magnet from the kit was placed against the container wall. The magnet attracted the cytokines bound to the microspheres. The supernatant was removed with a micropipette, isolating the pellet containing cytokine-bound microspheres.

The bound cytokines were then eluted in 0.9% NaCl solution to release them from the microspheres. A magnet was again applied to attract the beads, leaving cytokines free in solution. The cytokine-containing supernatant was collected and centrifuged at 400g for 5 minutes to break cytokine molecular chains.

After centrifugation, the sample rested for 20–30 minutes, followed by differential centrifugation at 4500g for 10 minutes to separate α helices and β sheets. β sheets deposited at the bottom, while α helices remained suspended in a thin layer at the surface.

The final sample was diluted 1:1000 in 0.9% NaCl solution for therapeutic use. It was stabilized for 24 hours at –25 °C and stored at 0–5 °C until administration, with a maximum shelf life of 6 months.

This protocol was repeated for each cytokine of interest. Separation of α and β chains enabled selective use: α subunits for suppression/modulation, β subunits for stimulation.

The choice of chains for administration was based on quantitative analysis of each patient's cytokine profile, determined in the laboratory.

1.3 Measurement of IL-10 and Evaluation Timings

Serum levels of IL-10 were determined at three predefined time points for each patient:

- T0 (pre-conventional therapies): sampling performed before the initiation of conventional therapies (surgery, chemotherapy, radiotherapy, or official immunotherapy), in order to document the baseline cytokine profile, uninfluenced by major therapeutic interventions.
- T1 (post-conventional therapies and Pre-cytokine therapy): sampling performed at the end of the standard therapy cycle and immediately before the start of autologous cytokine therapy, to evaluate the net effect of conventional therapies on IL-10 levels.
- T2 (post-autologous cytokine therapy): sampling performed at the conclusion of the autologous cytokine therapy cycle, with the aim of assessing the functional response of IL-10 to the autologous administration.

The use of three time points allows for the distinction of the impact of conventional therapies (T0→T1) from the additional effect of autologous cytokine therapy (T1→T2), thereby supporting the temporal interpretation of IL-10 dynamics in relation to the therapeutic interventions.

All cytokine assays were performed using a high sensitivity chemiluminescence immunoassay. The data were compared longitudinally with baseline values (T0), using the same certified laboratory for all time points.

No severe adverse events or systemic reactions to the therapy were recorded. The data were analyzed in parallel to evaluate the correlation between cytokine trends and subjective clinical response.

Results

The sequential analysis of serum interleukin-10 levels in the five enrolled patients shows a markedly consistent pattern, despite heterogeneity in underlying diseases and therapeutic pathways. In all subjects, baseline values measured before the start of conventional therapies (T0) ranged between 430 and 870 pg/mL, indicating an initially preserved cytokine profile. Completion of standard oncologic and immunomodulatory treatments (T1), regardless of their nature (surgery, chemotherapy, radiotherapy or immunotherapy), resulted in a profound suppression of interleukin-10 production in every case, with a mean reduction greater than 85% compared to baseline values (Figure 1).

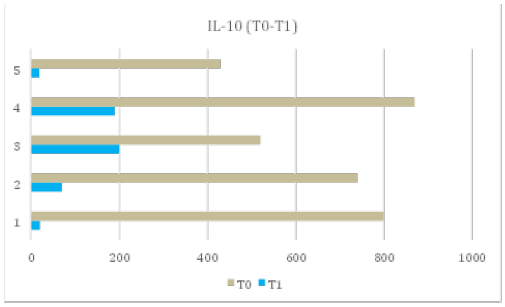

Figure 1: Changes in serum interleukin-10 levels (pg/mL) for each patient. The bars show a decrease from T0 to T1.

Subsequent administration of autologous cytokine therapy induced a robust increase in serum interleukin-10 levels in all patients, with final values (T2) ranging between 680 and 1100 pg/mL (Figure 2). The percentage increase from T1 was very high across the cohort, with recoveries exceeding 400% in most cases and increments up to 4400% in those subjects who were most immunodepressed at T1. In four out of five patients, T2 values surpassed the baseline levels observed at T0, suggesting a regulatory reactivation that goes beyond a simple restitutio ad integrum. In the remaining patient, the final value (680 pg/mL) was still extremely close to the initial level (740 pg/mL), indicating a complete functional recovery of the interleukin-10 circuit.

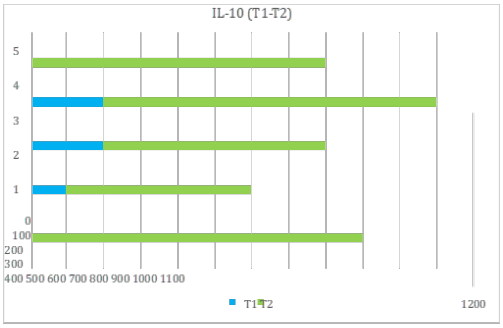

Figure 2: Changes in serum interleukin-10 levels (pg/mL) for each patient. The bars show the increase from T1 to T2, highlighting a marked and homogeneous functional recovery across the cohort.

This uniformity of response, observed despite differences in diagnosis and therapeutic history, supports the hypothesis that autologous cytokine therapy is able to restore the regulatory capacity of an immune microenvironment altered by conventional treatments. In particular, the recovery of interleukin-10 to high levels suggests a reconfiguration of the immunoregulatory compartment toward a "protective" profile, potentially associated with improved immune resilience and better control of chronic inflammation. The absence of adverse events or systemic reactions during treatment further supports the safety of this approach.

To provide a visual representation of this dynamic and to facilitate comparison between the three observation time points, the following graph summarizes the trend in serum interleukin-10 levels in the five patients.

Figure 3 provides an immediate and comparative illustration of serum interleukin-10 levels in the five patients at the three study time points (T0, T1, T2. The graph clearly highlights the biphasic effect of the therapeutic pathway: in all subjects, initial values (grey bars, T0) range between 430 and 870 pg/mL, indicating a relatively preserved basal cytokine profile. Following conventional oncologic and immunomodulatory therapies (blue bars, T1), a uniform and marked collapse of interleukin-10 production is observed, with values in some cases approaching near-zero levels (patients 1 and 5), underscoring the suppressive impact of standard treatments on the immunoregulatory compartment.

The effect of autologous cytokine therapy is represented by the green bars (T2), showing a clear and consistent increase in all patients, with final values between 680 and 1100 pg/mL. In four out of five subjects, recovered levels substantially exceed pre-treatment baseline concentrations, while in the remaining patient (subject 2) the final value is very close to baseline. This graphical pattern visually confirms that the autologous intervention not only restores interleukin-10 production compromised by conventional treatments, but tends to shift it to a higher and potentially more functionally protective level.

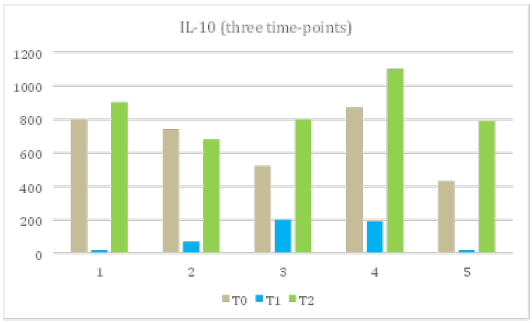

Figure 3: Individual trends of serum interleukin-10 levels at three times T0 (before conventional therapies), T1 (after conventional therapies) and T2 (after autologous cytokine therapy). The graph highlights the marked reduction of interleukin-10 in all patients after standard treatments and the subsequent uniform increase in response to autologous therapy, up to and beyond baseline levels.

The graph effectively summarizes the observed dynamic: a profound suppression induced by conventional treatments followed by a robust and homogeneous recovery after autologous cytokine therapy, providing further support to the hypothesis that this intervention can favorably modulate the immunoregulatory response.

Conclusions

The data from this small, clinically heterogeneous cohort nonetheless show a remarkably consistent pattern, indicating that autologous cytokine therapy can functionally modulate serum IL-10 levels in patients previously exposed to conventional oncologic and immunomodulatory treatments. In all five cases, a pronounced decline in IL-10 was observed after standard therapies, consistent with a substantial impairment of the immunoregulatory compartment, followed by a uniform and robust recovery after the autologous cytokine cycle, with values returning to – or exceeding – baseline levels.

This path suggests that appropriately processed cytokine extracts derived from autologous plasma are capable of re-engaging IL-10–mediated regulatory pathways, restoring a profile more compatible with a protective, homeostasis-oriented role rather than with mere immunosuppression. This observation is particularly noteworthy in the context of patients already exposed to major surgery, radio-/chemotherapy and/or conventional immunotherapy, all of which are known to exert a deep and often long-lasting impact on immune homeostasis.

Conceptually, these findings support the hypothesis that autologous cytokine therapy may act as a supportive intervention for the immune microenvironment after major therapeutic insult, promoting the re-establishment of a more physiological regulatory balance. The consistency of the response pattern across different diagnoses argues against a purely symptomatic effect and instead points toward a deeper modulation of cytokine networks.

While larger, controlled studies will be required to confirm and extend these observations, the present results provide preliminary evidence that autologous cytokine-based interventions may contribute to restoring an IL10–centered immunoregulatory equilibrium in patients undergoing intensive oncologic and immunomodulatory treatments.

Conflicts of Interest

The authors declare no conflicts of interest.

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