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Increased Myeloid-Derived Suppressor Cells in Newly Diagnosed Multiple Sclerosis: Immunophenotyping and Functional Validation

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ABSTRACT

Introduction: Myeloid-derived suppressor cells (MDSCs) are key regulators of immune responses during chronic inflammation. Their role in early multiple sclerosis (MS) remains incompletely defined. We aimed to evaluate circulating monocytic (M-MDSC) and polymorphonuclear (PMN-MDSC) levels in newly diagnosed, treatment-naïve MS patients and to assess their functional suppressive capacity.

Methods: Eighteen newly diagnosed MS patients and ten age- and sex-matched healthy controls were included. Peripheral blood MDSC subsets were quantified by flow cytometry. Clinical characteristics, MRI findings, and cerebrospinal fluid (CSF) parameters were recorded. Proof-of-concept functional validation was performed using T-cell proliferation assays assessed using CFSE fluorescence dilution by flow cytometry.

Results: Both M-MDSC and PMN-MDSC percentages were significantly higher in MS patients compared with controls ($p<0.001$ for both). Myeloid-derived suppressor cell levels did not differ according to sex and showed no correlation with time from symptom onset, MRI activity, or CSF parameters. Functionally, sorted MDSC subsets suppressed activated T-cell proliferation.

Conclusion: Newly diagnosed, untreated MS patients exhibit an expansion of circulating MDSCs with preserved suppressive capacity. These findings suggest that early MS may involve a systemic immunoregulatory response that is not fully captured by conventional clinical or radiological measures.

Keywords: Flow cytometry, multiple sclerosis, myeloid derived suppressor cells, T-cell suppression

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INTRODUCTION

Multiple sclerosis (MS) is a chronic immune-mediated disorder of the central nervous system characterized by inflammatory demyelination and neuroaxonal injury. While adaptive immune mechanisms, particularly autoreactive T and B lymphocytes, play a central role in MS pathogenesis, innate immune regulation is increasingly recognized as a critical determinant of immune balance (1).

Myeloid-derived suppressor cells (MDSCs) are immature myeloid cells with potent immunosuppressive activity and are broadly classified into monocytic (M-MDSCs) and polymorphonuclear (PMN-MDSCs) subsets (2). These cells expand under inflammatory conditions driven by cytokines such as granulocyte-macrophage colony-stimulating factor and interleukin-6 and suppress T-cell responses through arginase-1 activity, nitric oxide and reactive oxygen species production, and inhibitory checkpoint signaling (2–4).

Experimental autoimmune encephalomyelitis and human MS studies suggest that MDSCs act as context-dependent immunoregulatory cells, with potentially protective roles during early inflammatory stages (5–9). However, human data focusing on treatment-naïve patients at disease onset are limited, and functional validation of MDSCs in early MS remains scarce (8,10).

Highlights

- **Circulating M-MDSC and PMN-MDSC levels are increased in newly diagnosed MS.**
- **Expanded MDSCs suppress T-cell proliferation.**
- **MDSC levels are independent of disability, MRI activity, and CSF markers.**
- **Findings support early systemic immunoregulation in MS.**

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In this study, we aimed to quantify circulating M-MDSC and PMN-MDSC levels in newly diagnosed, untreated MS patients, assess their relationship with clinical and paraclinical parameters, and confirm their functional suppressive capacity.

Beyond multiple sclerosis, accumulating evidence from other autoimmune and inflammatory conditions indicates that expansion of myeloid-derived suppressor cell–like populations may reflect a compensatory immunoregulatory response rather than a direct marker of disease severity (11,12). In autoimmune blistering disorders, Oktem et al. demonstrated a marked increase in circulating polymorphonuclear MDSCs (PMN-MDSCs) that retained potent T-cell suppressive capacity, while showing limited association with baseline clinical severity and a closer relationship with treatment response (12). These observations suggest that MDSC expansion may accompany active immune dysregulation without necessarily translating into measurable clinical or tissue burden.

At the same time, recent studies have emphasized that phenotypic identification of MDSCs based solely on surface markers may be insufficient to define their functional properties (13). In oncologic settings, Horzum et al. showed that CD66b^{+} myeloid cells phenotypically overlapping with PMN-MDSCs could exert pro-inflammatory and T-cell costimulatory effects rather than immunosuppression, underscoring the importance of functional validation (14). Together, these findings highlight the need to assess both the quantitative and functional characteristics of MDSC subsets in immune-mediated diseases such as MS (2,13,14).

METHODS

Study Population

Eighteen newly diagnosed MS patients fulfilling the 2017 McDonald criteria and ten age- and sex-matched healthy controls without autoimmune disease or malignancy were included. All MS patients were treatment-naïve at the time of sampling.

Clinical and Paraclinical Assessment

Demographic data, clinical presentation, Expanded Disability Status Scale (EDSS) scores, MRI activity (presence of gadolinium-enhancing lesions), and cerebrospinal fluid (CSF) parameters were recorded.

Blood Collection and Processing

Blood collection and processing involved collecting blood samples in EDTA tubes (Vacutest) and processing them within 2 hours. For density gradient separation of peripheral blood mononuclear cells (PBMC), the samples were diluted (1:1 with 1xphosphate-buffered saline (PBS)), layered over 1.077 g/mL Ficoll-Hypaque (Sigma-Aldrich), and centrifuged at 400x g for 30 minutes without brake or acceleration. The total PBMC was collected, washed, counted, and used in further experiments.

Flow Cytometry

Immunophenotyping

Immunophenotyping was performed using monoclonal antibodies: anti-human CD45 (HI30), CD66b (G10F5), HLA-DR (L243), and CD14 (M5E2) (BioLegend, USA). The percentage of positive cells was determined using isotype-matched antibodies. Fully stained multi-color samples were acquired, and the compensation matrix was applied in real-time or during post-acquisition analysis. This subtracted the calculated spillover from each channel, revealing the "true" fluorescence signal for each marker. The samples were run on the Canto II (BD Biosciences, USA), and data were analyzed using FlowJo software (v. 10).

Cell sorting and purification

CD14^{+} population amongst PBMC were isolated with anti-CD14 magnetic-activated cell sorting (MACS) immunobeads, respectively, according to the manufacturer's instructions (Miltenyi Biotec). Then, the isolation of CD14^{+} monocyte subpopulations, and CD14+HLA-DR^{+} monocyte population and CD14^{+}HLA-DR^{-} M-MDSC populations was continued with fluorescence-activated cell sorting (FACS). Briefly, for the purification of CD14^{+} monocytes and M-MDSC, freshly collected PBMC were resuspended in MACS buffer (1×PBS/2 mM EDTA/0.5% BSA) and incubated with anti-CD14 immunobeads, applied onto MACS-LS

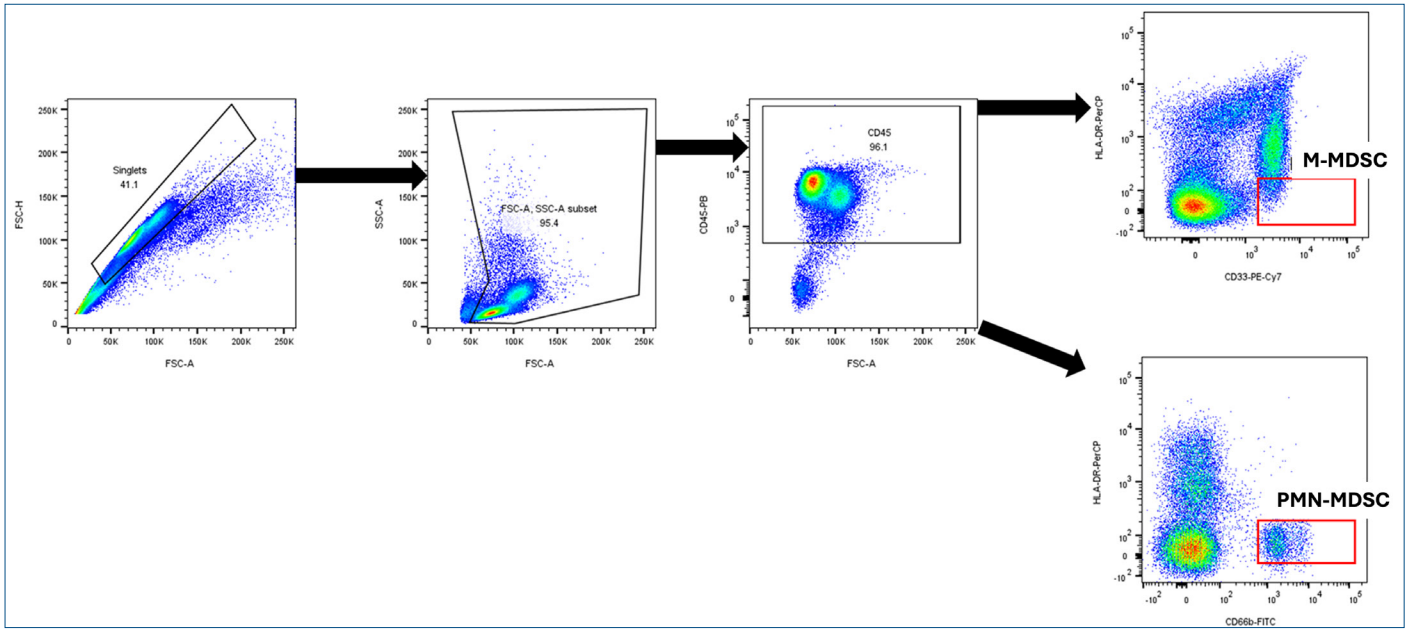

Figure 1. Flow cytometry gating strategy for identification of circulating MDSC subsets. Representative gating strategy used for identification of circulating MDSC populations. After exclusion of doublets and debris, leukocytes were gated according to forward- and side-scatter characteristics and CD45 expression. Monocytic MDSCs were identified as CD33+HLA-DR- cells, whereas polymorphonuclear MDSCs were defined as CD66b+HLA-DR- cells (MDSC: myeloid-derived suppressor cell; M-MDSC: monocytic myeloid-derived suppressor cell; PMN-MDSC: polymorphonuclear myeloid-derived suppressor cell; FSC: forward scatter; SSC: side scatter).

columns (Miltenyi Biotec), and the immobilized cells were positively selected. After that, (BioLegend); CD14+HLA-DR+ and CD14+HLA-DR- populations were purified by FACS. The purity of sorted cells was assessed by flow cytometric immunophenotyping.

Analysis of monocytes' and M-MDSC'influence on T-cell responses

CD3+ T-cells were isolated from healthy donors' and MS patients peripheral blood mononuclear cells (PBMC) using CD3 MACS (Miltenyi Biotec). Subsequently, these cells were labeled with carboxyfluorescein succinimidyl ester (CFSE, 5 µM) (BioLegend, USA) according to the manufacturer's protocol. Purified HLA-DR+ monocytes and HLA-DR$^-$ M-MDSCs from MS patients (25000 cells/200 µL in U-bottom 96-well plates) at various ratios for 72 hours in the presence of anti-CD3/CD28 beads, which mimics the initial signal for T-cell activation. CD8+ and CD4+ T cells were gated, and their proliferation was assessed using CFSE fluorescence dilution by flow cytometry after 72 hours of co-culture.

Statistical Analysis

Continuous variables were assessed for normality and are presented as mean ± standard deviation or median (interquartile range), as appropriate. Between-group comparisons were performed using independent-samples t tests for normally distributed variables and Mann-Whitney U tests for non-normally distributed variables. Correlation analyses were conducted using Pearson or Spearman correlation coefficients according to data distribution. Statistical analyses were performed using IBM Statistical Package for Social Sciences (SPSS) program version 23.0 (IBM Corp., Armonk, NY, USA) and GraphPad Prism version 10. All statistical tests were two-sided, and a p value <0.05 was considered statistically significant.

Ethical consideration

This study was approved by Hacettepe University Health Sciences local ethics committee (GO2020/11-37) and conducted in agreement with the guiding principles of the Declaration of Helsinki and the good clinical practice. Informed consent was obtained from the participants and all methods were performed in accordance with the relevant guidelines and regulations.

RESULTS

A total of 18 treatment-naïve patients with newly diagnosed multiple sclerosis and 10 healthy controls were included in the study. Demographic and clinical characteristics of the study population are summarized in Table 1. The mean age did not differ significantly between MS patients (33.3±10.4 years) and healthy controls (37.2±4.8 years; p=0.343). The sex distribution was also comparable between groups (female: male ratio 12:6 in MS vs 6:4 in controls; p=0.185). The median EDSS score in the MS cohort was 2.5 (range 0–3.0).

In contrast, circulating MDSC subsets were markedly increased in MS patients compared with healthy individuals. Both monocytic MDSCs (M-MDSCs) and polymorphonuclear MDSCs (PMN-MDSCs) were significantly elevated in MS patients, both as percentages of peripheral blood mononuclear cells and as absolute counts per microliter (Figure 1).

In contrast, circulating MDSC subsets were markedly increased in MS patients. The proportion of monocytic MDSCs (M-MDSCs) was significantly higher in MS patients compared with healthy controls (13.64±7.8% vs 0.75±0.45%, p <0.001). Similarly, polymorphonuclear MDSCs (PMN-MDSCs) were significantly elevated in the MS group (12.48±9.32% vs 0.73±0.46%, p <0.001). Consistent with these findings, absolute counts of circulating MDSC subsets were also significantly increased in MS patients. Absolute M-MDSC counts were 335.09±297.84 cells/µL in MS patients compared with 15.68±7.90 cells/µL in controls (p <0.001), while PMN-MDSC counts were 238.16±245.34 cells/µL versus 15.02±9.03 cells/µL, respectively (p=0.001) (Table 1, Figure 2). These results indicate a substantial expansion of both MDSC subsets in newly diagnosed, untreated MS patients.

Within the MS cohort, no significant differences were observed in M-MDSC or PMN-MDSC percentages between female and male patients. Similarly, Expanded Disability Status Scale (EDSS) scores and cerebrospinal fluid parameters, including IgG index and CSF protein levels, did not differ by sex.

Correlation analyses revealed no significant association between the interval from first clinical symptom to blood sampling and the proportion of M-MDSCs (r=-0.028, p=0.912) or PMN-MDSCs (r=0.089, p=0.726). Furthermore, comparison of patients with and without radiologically active lesions showed no statistically significant differences in either M-MDSC or PMN-MDSC percentages (p=0.338; p=0.871 respectively).

Functional validation confirmed the potent immunosuppressive identity of M-MDSCs identified in newly diagnosed MS patients. In proof-of-concept suppression assays, M-MDSCs significantly inhibited T-cell proliferation relative to control conditions. Stimulated T-cells in control cultures exhibited a high proliferation rate in CD4$^+$ T-cell (%92.3±1.4), CD8$^+$ - cell (%91.2±1.1) at 1:1 ratio of monocyte and T-cells; however, co-culture with sorted M-MDSCs resulted in a marked reduction in T-cell expansion CD4$^+$ T-cell (%62.3±1.4), CD8$^+$ T-cell (%60.1±1.1), demonstrating a suppression rate exceeding 50% (Figure 3). Collectively, these findings indicate that the expansion of the M-MDSC compartment at disease onset represents a functionally active immunoregulatory

Table 1. Demographic, clinical, and MDSC characteristics of patients with multiple sclerosis and healthy controls

	MS (n=18)	Control (n=10)	p-value
Age ± SD	33.3±10.4	37.2±4.8	0.343
Female: Male (%)	12:6 (66:34)	6:4 (60:40)	0.185
EDSS (min- max)	2.5 (0.0–3.0)	-	
CSF IgG index ± SD	1.15±0.53	-	
CSF protein (mg/dL) ± SD	42.59±12.07	-	
M-MDSC% ± SD	13.64±7.8	0.75±0.45	<0.001
PMN-MDSC% ± SD	12.48±9.32	0.73±0.46	<0.001
M-MDSC count (/µL) ± SD	335.09±297.84	15.676±7.902	<0.001
PMN-MDSC count (/µL) ± SD	238.16±245.34	15.016±9.034	0.001

SD: standard deviation; min: minimum; max: maximum; EDSS: expanded disability status scale; CSF: cerebrospinal fluid; IgG: immunoglobulin G; M-MDSC: monocytic myeloid-derived suppressor cells; PMN-MDSC: polymorphonuclear myeloid-derived suppressor cells; a p value <0.05 was considered statistically significant.

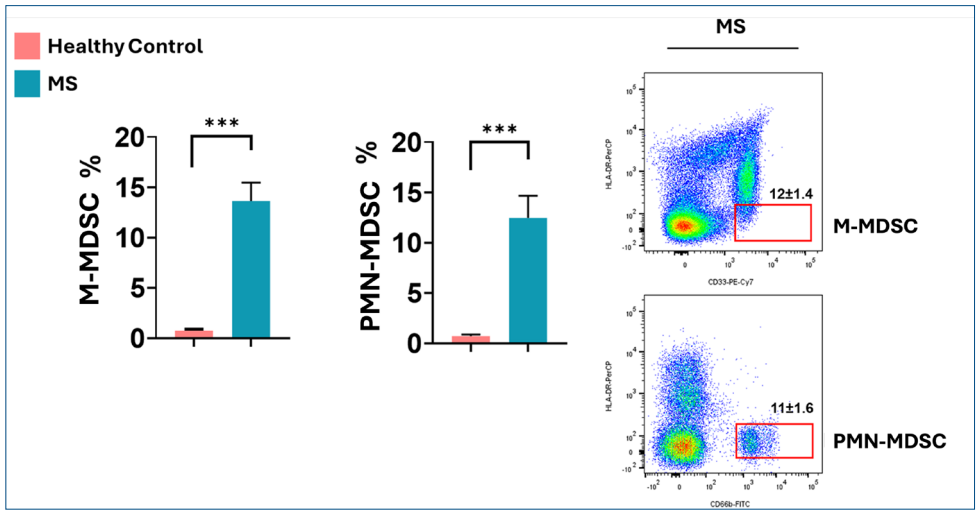

Figure 2. Increased circulating MDSC subsets in newly diagnosed multiple sclerosis patients. Peripheral blood monocytic myeloid-derived suppressor cells (M-MDSCs) and polymorphonuclear MDSCs (PMN-MDSCs) were quantified by flow cytometry in newly diagnosed, treatment-naïve MS patients and healthy controls. Both M-MDSC and PMN-MDSC percentages were significantly higher in MS patients compared with controls. Representative flow cytometry plots illustrating identification of MDSC subsets are shown on the right. Data are presented as mean ± standard deviation (***p <0.001; MS: multiple sclerosis; MDSC: myeloid-derived suppressor cell; M-MDSC: monocytic myeloid-derived suppressor cell; PMN-MDSC: polymorphonuclear myeloid-derived suppressor cell).

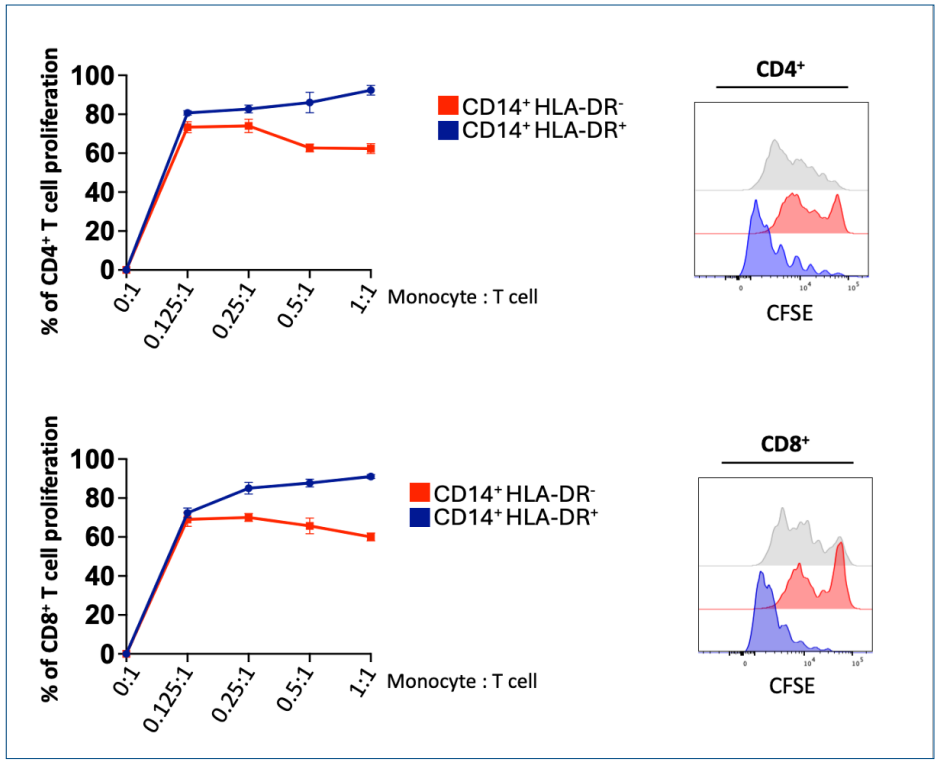

Figure 3. Functional suppressive activity of M-MDSCs on T-cell proliferation. Functional validation of monocytic MDSC suppressive activity was performed using CFSE-based T-cell proliferation assays. Sorted CD14+HLA-DR- M-MDSCs significantly inhibited proliferation of activated CD4+ and CD8+ T cells in a dose-dependent manner compared with CD14+HLA-DR+ monocytes. Representative CFSE dilution histograms and quantitative analysis of T-cell proliferation at different monocyte-to-T-cell ratios are shown (M-MDSC: monocytic myeloid-derived suppressor cell; CFSE: carboxyfluorescein succinimidyl ester).

component, potentially contributing to the immunological dysregulation characteristic of early MS pathology.

Overall, although both M-MDSC and PMN-MDSC levels were significantly elevated in MS patients compared with healthy controls, no meaningful associations were identified between MDSC frequencies and clinical disability, radiological activity, or cerebrospinal fluid parameters in this cohort.

DISCUSSION

In this study, we demonstrate a significant expansion of both monocytic and polymorphonuclear myeloid-derived suppressor cells in newly diagnosed, treatment-naïve multiple sclerosis patients, accompanied by preserved suppressive activity, particularly within the monocytic subset. These findings support the concept that MDSC expansion represents an early immunoregulatory response rather than a passive byproduct of accumulated tissue damage or clinical disability (15). Increasing evidence indicates that MDSCs exert context-dependent functions in multiple sclerosis, with their regulatory properties varying across disease stages rather than uniformly reflecting disease severity (6,16).

Experimental and human studies suggest that MDSCs may be preferentially mobilized during early inflammatory phases as part of a compensatory mechanism aimed at limiting excessive autoreactive T-cell responses (17–19). In this context, the increased circulating MDSCs observed in our cohort—despite the absence of advanced disability or extensive radiological burden—may reflect a systemic attempt to restore immune homeostasis at disease onset. Similar dynamics have been described in experimental autoimmune encephalomyelitis models, where MDSC populations expand during early inflammation and subsequently decline

or undergo functional reprogramming as chronic inflammation becomes established (6).

Our findings extend previous immunophenotyping studies investigating peripheral myeloid alterations in early multiple sclerosis. Several studies reported increased frequencies of monocytic MDSCs and inflammatory monocytes in treatment-naïve relapsing–remitting MS patients using multiparametric flow cytometry (16,20). However, those studies primarily focused on phenotypic characterization without functional assessment. In contrast, our data demonstrate that the expanded monocytic MDSC population retains suppressive activity, providing functional evidence that early MDSC expansion represents an active immunoregulatory response rather than merely a phenotypic alteration.

Human studies evaluating MDSCs in multiple sclerosis remain limited and have yielded heterogeneous results. Iacobaeus et al. showed that monocytic and granulocytic MDSC subsets increase during relapsing–remitting disease and retain suppressive capacity, whereas this function appears impaired in secondary progressive MS, suggesting stage-dependent functional plasticity of MDSCs across the disease course (16). Our findings extend these observations by demonstrating that MDSC expansion is already present at the earliest clinically detectable stages of untreated disease.

Notably, several studies indicate that MDSC expansion does not necessarily correlate with conventional measures of disease severity (5). Similar observations have been reported in other autoimmune conditions, where increased circulating PMN-MDSCs display potent suppressive activity despite limited association with baseline disease activity (12). These findings highlight that phenotypic identification of MDSCs alone may be insufficient to define their immunological role and underscore the importance of functional validation (14). Accordingly, functional assays in the present study focused on monocytic MDSCs, which are more consistently associated with suppressive mechanisms involving arginase-1, nitric oxide production, and immune checkpoint signaling (13,21,22).

Previous studies have also reported reduced circulating MDSCs in certain MS cohorts. For example, Cantoni et al. observed decreased monocytic MDSC levels in relapsing–remitting MS patients together with alterations in regulatory pathways such as STAT3 and ARG1 (23). These discrepancies may reflect differences in disease stage, treatment exposure, or immunological context. Early inflammatory phases may promote expansion of suppressive myeloid populations, whereas chronic or treated disease states may be associated with quantitative or functional alterations.

Consistent with this interpretation, we did not observe significant correlations between circulating MDSC levels and clinical disability, MRI activity, or cerebrospinal fluid inflammatory markers. These findings suggest that MDSC expansion in early multiple sclerosis reflects a systemic immunoregulatory response rather than direct modulation by disease burden.

Several limitations should be acknowledged. Functional assays were restricted to monocytic MDSCs because PMN-MDSCs show low recovery after density-gradient separation and are highly susceptible to activation during ex vivo manipulation. In addition, the cohort size was relatively small and consisted exclusively of newly diagnosed, treatment-naïve patients. Finally, the cross-sectional design precludes evaluation of longitudinal changes in MDSC populations during disease progression or treatment.

Future prospective studies incorporating longitudinal sampling will be required to clarify the temporal dynamics of MDSC subsets across disease stages and under disease-modifying therapies, as well as to better define the functional role of polymorphonuclear MDSCs in human autoimmune disease.

In conclusion, early multiple sclerosis appears to be accompanied by systemic expansion of functionally suppressive MDSCs, supporting the presence of an intrinsic immunoregulatory response that is not directly reflected by conventional clinical or radiological measures.

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Informed Consent: Informed consent was obtained from the participants.

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Conflict of Interest: The authors declared that there is no conflict of interest.

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A generative artificial intelligence tool (ChatGPT) was used for language editing and improvement of grammar and clarity in parts of the manuscript. No AI tool was used for data analysis, interpretation, or generation of scientific content. All scientific responsibility remains with the authors.

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