Metabolic acidosis negatively influences dietary intake, increases protein catabolism, and deteriorates nutritional status. In the present study, we evaluated in peritoneal dialysis (PD) patients whether parameters of blood acid–base equilibrium influence total and subpopulation lymphocyte counts (TLC, SLCs), which are markers of the immunologic and nutritional status of dialyzed patients. Studies were carried out in 55 patients, mean age 50.9 ± 12.4 years, treated with PD for a mean of 22.2 ± 11.4 months. Parameters of blood acid–base equilibrium were measured simultaneously with evaluation of TLC and SLCs. (Antigens CD3, CD4, CD5, CD8, CD19, CD16+56 were determined using flow cytometry.)

The study patients showed compensated metabolic acidosis (pH: 7.40 ± 0.04; HCO$_3$–: 22.9 ± 2.4 mmol/L). Statistical analysis revealed significant ($p < 0.05$) positive correlations of bicarbonate blood concentration and base excess with TLC and with CD3, CD5, and CD8 cell counts, but not with CD19 and CD16+56 cell counts. The CD4 cell count correlated only with blood bicarbonate level.

Patients on PD who show better correction of metabolic acidosis also show higher TLC and CD3, CD4, CD5, and CD8 cell counts. The numbers of B lymphocytes (CD19) and natural killer cells (CD16+56) are not directly related to bicarbonate blood concentration, at least in the examined range.

Key words
Lymphocytes, bicarbonate, base excess

Introduction
Metabolic acidosis negatively influences dietary intake, increases protein catabolism, and deteriorates nutritional status (1,2). In the present study, we evaluated in peritoneal dialysis (PD) patients whether parameters of blood acid–base equilibrium influence total lymphocyte count (TLC) and subpopulation lymphocyte counts (SLCs), which are markers of immunologic and nutritional status in dialyzed patients.

Patients and methods
The study was carried out in 55 stable uremic patients, mean age 50.9 ± 12.4 years, treated with PD for a mean of 22.2 ± 11.4 months.

Blood acid–base equilibrium was determined simultaneously with an evaluation of TLC and SLCs. Parameters of acid–base balance were obtained from arterialized capillary blood using a blood gas analyzer (Compact 1: Roche Diagnostics, Graz, Austria). The SLCs were determined using flow cytometry, with detection of antigens CD3, CD4, CD5, CD8, CD19, and CD16+56.

Total weekly Kt/V, total weekly creatinine clearance, and protein nitrogen appearance were used to evaluate PD adequacy. Creatinine clearance was normalized to body surface area (BSA), and protein nitrogen appearance, to ideal body mass (IBM). Dietary intake of various nutrients was analyzed from diet histories using the computer program Food version 3.0 [Institute of Nutrition and Food Products, Warsaw, Poland (license 86)].

Results are expressed as mean and one standard deviation. Correlation analyses were performed using the Spearman coefficient. A $p$ value below 0.05 was considered statistically significant.
Results
The study patients showed compensated metabolic acidosis. Blood pH was in the normal range (7.40 ± 0.04), but bicarbonate concentration was reduced (22.9 ± 2.4 mmol/L).

Metabolic acidosis, defined by a bicarbonate concentration below 22 mmol/L, was found in 40% of patients. Metabolic alkalosis, with a bicarbonate concentration above 26 mmol/L, was seen in 9% of patients.

In the entire group of study patients, TLC and CD3, CD5, and natural killer cell counts were in the normal range. The CD4, CD8, and CD19 cell counts were all lower than normal. Significant positive correlations were found between the TLC and CD3, CD5, and CD8 cell counts and bicarbonate level and base excess. The CD4 cell count correlated only with blood bicarbonate level. B Lymphocytes and natural killer cells showed no significant correlations with examined parameters of acid–base balance (Table I).

No significant differences were observed in PD adequacy between patients with bicarbonate concentrations below 22 mmol/L and above 22 mmol/L, evaluated using total weekly Kt/V (2.17 ± 0.58 vs. 1.90 ± 0.53), total weekly creatinine clearance (67.0 ± 21.5 L/1.73 m² BSA vs. 58.2 ± 17.9 L/1.73 m² BSA), and protein nitrogen appearance (0.86 ± 0.36 g/kg IBM/d vs. 0.76 ± 0.20 g/kg IBM/d).

Non acidotic PD patients showed higher dietary intake of various nutrients. Significant differences were seen for calcium and beta-carotene intake, as well as for energy delivered from protein expressed as a percentage of total ingested energy (Figure 1).

| Table I Correlations between lymphocyte counts and blood bicarbonate concentration and base excess (BE) in peritoneal dialysis patients |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| Cell type       | Cell quantity (g/L) | HCO₃⁻ | r Value | p Value | BE | r Value | p Value |
| TLC             | 1.68±0.62         | 0.291 | 0.031 | 0.309 | 0.022 | 0.302 | 0.025 |
| CD3             | 1.19±0.47         | 0.328 | 0.015 | 0.302 | 0.025 | 0.214 | 0.116 |
| CD4             | 0.53±0.25         | 0.291 | 0.031 | 0.249 | 0.067 | 0.214 | 0.116 |
| CD5             | 1.17±0.43         | 0.354 | 0.008 | 0.343 | 0.010 | 0.214 | 0.116 |
| CD8             | 0.36±0.16         | 0.334 | 0.013 | 0.298 | 0.027 | 0.214 | 0.116 |
| CD19            | 0.11±0.08         | 0.264 | 0.052 | 0.214 | 0.116 | 0.214 | 0.116 |
| CD16+56         | 0.31±0.21         | -0.010 | 0.939 | 0.062 | 0.655 | 0.214 | 0.116 |

Discussion
Permanent metabolic acidosis, usually compensated, is known phenomenon in dialyzed patients (3), but its prevalence varies from one center to another (1,4). Metabolic acidosis (defined as a bicarbonate concentration below 23 mmol/L) was shown in 43% of Chinese PD patients (1). As in our study, total Kt/V was not different between the acidotic and non acidotic patients. In Indian PD patients, a serum bicarbonate level below 22 mmol/L was found only in 6.2% of cases; in hemodialysis patients, the level was 75% of examined cases (4). As compared with our earlier results obtained in PD patients (2), metabolic acidosis was this time less pronounced (40% of patients vs. 50% of patients), but metabolic alkalosis was more frequent (9% of patients vs. 7% of patients). The differences were not statistically significant, however.

The influence of metabolic acidosis on circulating lymphocyte count may be related to correlations between acidotic state and protein turnover. The more pronounced the acidosis, the greater the catabolism and the negative nitrogen balance, resulting in reduced lymphocyte counts because the cells are sensitive to protein deficiency. Correction of acidosis reduces the catabolic stress and protein degradation that leads to protein deficiency. The better the protein nutrition, the higher the lymphocyte count (5–9).

In the present study, acidotic patients had a worse dietary intake. Among the lower intake nutrients was beta-carotene. Carotenoids are known to increase the helper lymphocyte count (10). In patients with chronic bacterial infection or with AIDS, and in children with acute pneumonia, high supplementation of vitamin A improves CD4 and CD8 cell counts and synthesis of immunoglobulins (11–14). The influence of beta-carotene on the immune system is probably connected with metabolism of vitamin A (15).

Conclusions
In PD patients who show better correction of metabolic acidosis, higher TLC and CD3, CD4, CD5, and CD8 cell counts are seen. The numbers of B lymphocytes (CD19) and natural killer cells (CD16+56) are not directly related to bicarbonate blood concentration, at least in the examined range. Thus, metabolic acidosis influences nutrition and the immune system in PD patients.
Lymphocytes and Bicarbonate in PD Patients

![Bar chart showing dietary intake comparison between acidotic and non-acidotic peritoneal dialysis patients](chart.png)

**FIGURE 1** Significant differences are seen in dietary intake between acidotic and non acidotic peritoneal dialysis patients.

**References**

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