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# Study of naïve and switched memory B cell level in Egyptian patients with common variable immunodeficiency

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## Abstract

Common variable immunodeficiency (CVID) is one of the primary immunodeficiency disorders. The phenotype of peripheral blood memory B cells is a useful tool in the classification of patients into clinically and functionally relevant groups. This study aimed to assess the level of naïve and switched memory B cells level and their correlation with the clinical phenotypes and complications in patients with CVID. This case control study included 30 adult patients with CVID and 30 normal controls, matched for age and sex. Complete blood count, cluster of differentiation 3 (CD3)+, CD4+, CD8+ T cells and CD19+27-IgD+ for naïve B cells and CD19+27+IgD- switched memory B cells levels were assessed. The mean age of the onset of symptoms was 16.9±15.1 years, the mean age of diagnosis was 27.30±14.39 years, with a diagnostic delay of 10.43±10.29 years, and the body mass index was significantly lower in CVID group. Infections including (upper respiratory tract infection, chronic diarrhea, pneumonia and bronchiectasis) were the most frequent phenotypes. CD4+, CD4+/CD8+ T cells, CD19+ and CD19+27+IgD- switch memory B cell, IgG, IgA, and IgM were significantly lower in CVID group than in the control group ($p$<0.001 and $p$<0.015, respectively). CD8+ T cells and CD19+27-IgD+ naïve B cells were significantly higher in the CVID group ($p$<0.001). CD19+27-IgD+ naïve B cells level was significantly lower in cases with bronchiectasis with low baseline serum IgG in lymphadenopathy group ($p$=0.049), and higher level of CD3+ T cells in cases with splenomegaly. There was no significant difference in laboratory results in CVID patients presented with autoimmune diseases, Granulomas nor enteropathy. In conclusion, high level of CD19+27-IgD+ naïve and low level of CD19+27+IgD- switch memory B cells are characteristic features of CVID. Moreover, the reduced CD19+27-IgD+ naïve B cells level can be a predictor of the development of bronchiectasis in CVID patients.

**Keywords:** Naïve B Cell, Switch Memory B Cell, Common Variable Immunodeficiency.

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## Introduction

Common variable immunodeficiency (CVID) is the most common manifested primary immunodeficiency disorder with an estimated prevalence of 1:50,000 to 1:25,000.[1,2] It displays impaired terminal B-cell differentiation and defective antibody responses.[3] A high

proportion of CVID patients have a low frequency of switch memory B cells.[4] In fact, CVID is mainly considered to be a humoral immunodeficiency disorder, although several immune cell types might be affected.[5] CVID is usually complicated by infections, autoimmune diseases, enteropathy, malignancies, and lymph proliferative diseases.[6] Pulmonary infections most commonly develop in CVID patients, which may further lead to bronchiectasis. Noninfectious pulmonary complications exhibit granulomatous-lymphocytic interstitial lung disease. Multiple systems can be involved because of immune dysregulation, such as cytopenia, splenomegaly and various autoimmune diseases.[6]

Switch memory B cells are a B cell sub-type that is formed following a primary infection. In the first primary response involving a particular antigen, the responding naïve cells proliferate to produce a colony of cells. Most of them differentiate into plasma cells which produce antibodies and clear away with the resolution of infection.[7] The rest persist as memory cells that can survive for years, or even a lifetime.[8] There are two types of memory B cells: IgM+ memory B cells (CD27+IgM+IgD−) and switched memory B cells (CD27+IgM−IgD−). In normal individuals the memory B cell compartment is comprised of 50% CD27+IgM+ memory B cells and the other half of CD27+IgM−IgD−switched memory cells.[9]

Impaired post germinal B cell maturation in the periphery (i.e., in blood and secondary lymphoid tissues) is a hallmark of CVID. However, CVID is a rather heterogeneous disease from the clinical, genetic, and immunologic point of view.[9] Overall, impaired differentiation of mature post-germinal center B-cells, consisting of severely reduced circulating class-switched memory B-cells and strongly decreased (i.e., undetectable) plasma cell production, are the most consistent defects in CVID.[10] In turn, depending on the specific defects encountered in the post-germinal center switch memory B cells, and the severity of such defects, distinct CVID patient subgroups, associated with distinct clinical profiles, have also been identified.[4] Apart from the alterations in post germinal center B-cells and plasma blasts/plasma cells, an increasing number of evidences indicate that the production and maturation of B-cells in bone marrow is also altered in at least one third of all CVID patients due to either a maturation blockade, and/or an altered bone marrow environment which is non-permissive for B-cell maturation.[9]

Circulating lymphocyte markers were the first extensively studied biomarkers in CVID, and quite successful in identifying patients at risk for developing non-infectious manifestations. Overall, patients with severely reduced peripheral switched memory B cells, and reduced T cells (especially naïve and regulatory T cells) were more likely to have these complications. In the B cell compartment, switched memory B cell phenotypes were the most widely utilized markers. Decreased switched memory B cells (cut-off: ≤ 0.55% of B cells) was an independent risk factor for granulomas, splenomegaly, and auto-immunity.[10] Similarly, a cut-off of <2% switched memory B cells was identified as a risk factor for granulomas and splenomegaly in another study [11]. Our study aimed to assess the level of switched memory B cells and naïve B cell and their correlation with the clinical phenotypes and complications in Egyptian patients with CVID.

## Patients and Methods

This case control study, conducted during Sep 2022 through May 2024, and included 30 adult patients (≥18 years old), diagnosed with CVID according to the International Consensus Document (ICON).[6]who attended the Clinical Immunology clinic and inpatient at Ain Shams University Hospitals and 30 normal controls, with matched age and sex.

A full clinical examination, complete blood count, and assessment of lymphocytes subsets: CD3, CD4, CD8, CD4/CD8 ratio, CD19, CD27 IgD using flow cytometry technique were performed in both cases and control groups.

Patients with other causes of secondary hypogammaglobulinemia including; drug induced, single gene defects hyper-IgM syndrome/X-linked agammaglobulinemia if suspected by high IgM level, chromosomal anomalies chromosome 18q-syndrome/

Trisomy, if clinically suspected (Mental retardation/Speech delay/Craniofacial malformation/Short stature/ Physical abnormalities as cleft palate) were excluded from the study. Infectious diseases; Human immunodeficiency virus (HIV)/Epstein-Barr virus (EBV) if clinically suggestive, Medical disorders affecting immunoglobulin levels such as Poor nutrition, Nephrotic syndrome, Protein-losing enteropathy if clinically suggestive were excluded from the study. And, patients with Splenectomy and pregnant females were also excluded.

*Cellular Testing*

Flow cytometry analysis for cellular immunity (CD3+, CD4+, CD8+ T cells and CD4+/CD8+ ratio, CD19+ and CD27+/-IgD+/- B cells) were done using a flow cytometer (Navios, 773232AH flow cytometer, Coulter, Electronics, Hialeah, FL, USA), according to the manufacturer's instructions.

Under complete aseptic conditions, a venous blood sample (6 ml) was obtained from each patient by a clean venipuncture tube. Samples were dispensed gently into 3 sterile EDTA tubes for complete blood picture and flow cytometry. Specimen for CD3, CD4, CD8, CD19 and CD27 IgD analysis were kept at a temperature of 18-25$^{o}$C and they were not shaken and analyzed within 24 hours. Using a 50 µl pipette a specimen with adjusted leukocytic count to 5-10 x 103 /µl was placed in tubes that were labeled with patient's name and the relevant monoclonal antibody (McAb). Then the determined volume appropriate fluorochrome conjugated McAbs was added (according to titration of the monoclonal agent). A sample of 1-2 ml of lysing solution was added and left in the dark and room temperature for 2-10 minutes till complete lysis of red blood cells (RBCs) and the suspension became clear. The results are expressed as a percentage of positive events in relation to all events acquired by gating.

*Statistical Methods*

The collected data were coded, tabulated, and statistically analyzed using the Statistical Package for Social Sciences (SPSS, IBM software version 28.0), IBM Corp., Chicago, USA, 2021. Quantitative data were tested for normality using Shapiro-Wilk, then if normally distributed, described as mean±SD (standard deviation) as well as minimum and maximum of the range. They were then compared using the independent t-test (two independent groups) and paired t-test (paired data). Qualitative data are described as number and percentage and compared using Chi square test. The level of significance was considered at $p \leq 0.05$.

## Results

Demographic variants of the study group showed that in CVID patients the mean age was 33.8±11.4 and 30.7±9.8 years, with male (n=23) predominance of 76.7% with low body mass index (BMI) 21.6±4.7 kg/m$^2$ ($p<0.001$). Clinical presentation was at the age of 16.9±15.1 years with diagnosis delay of 10.43±10.29 years.

There was heterogeneity of the clinical phenotypes among the study group such as, URTI, chronic diarrhea, pneumonia, enteropathy, splenomegaly, autoimmune thrombocytopenia, autoimmune arthritis, lymphadenopathy, and malignancy were 76.7%, 66.7%, 60%, 33.3%, 26.7%, 10%, 13.3%, 13.3%, 13.3%, and 3.3%, respectively. (Figure 1)

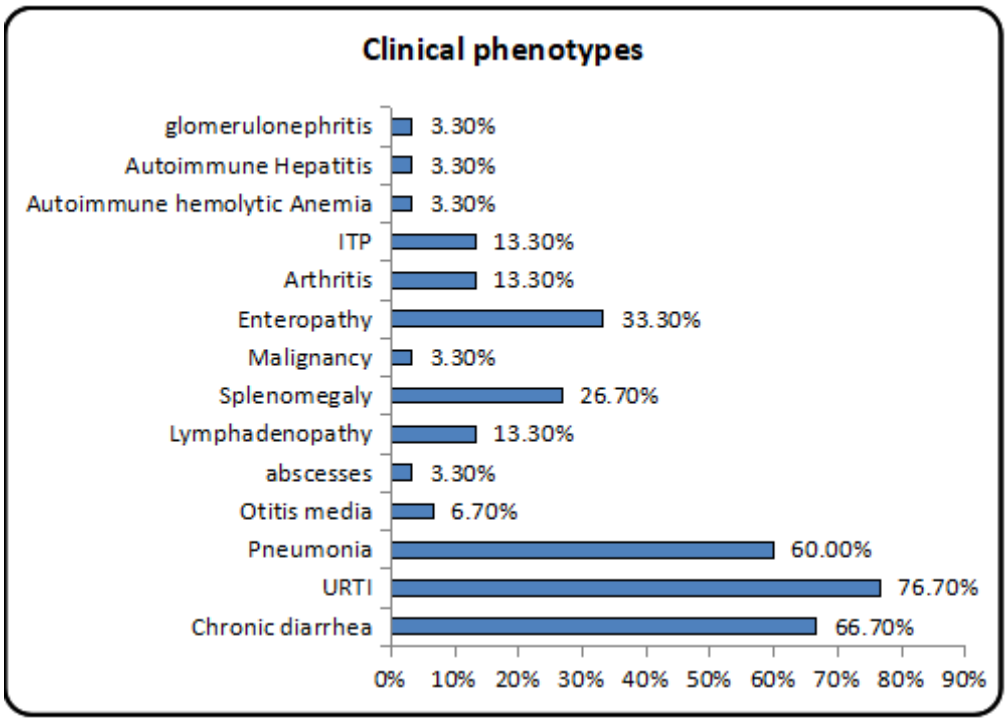


**Figure 1.** Clinical phenotypes among the common variable immunodeficiency (CVID) patients.

CD4+ T cells, CD4+/CD8+ ratio, CD19+27+IgD- switch memory B cell, IgG, IgA and IgM were significantly lower in the CVID group (p<0.001) and CD 19+ B cells also were lower in CVID group than control group (*p*=0.015). CD8+ T cells and CD19+27-IgD+ naïve B cell were significantly higher in the CVID group (*p*<0.001). No significant difference was observed between the studied groups regarding CD3 (*p*=0.169). (Table 1)

**Table 1.** Comparison of T cells and B cells subsets and immunoglobulin levels between common variable immunodeficiency (CVID) patients and controls.

| Variables | | CVID group (Total=30) | Control group (Total=30) | *p*-value |
|---|---|---|---|---|
| CD3 (%) | Mean±SD | 78.4±14.4 | 74.2±8.1 | NS |
| | Range | 36.4–98.0 | 60.5–88.8 | |
| CD4 (%) | Mean±SD | 31.7±12.6 | 41.2±4.8 | <0.001 |
| | Range | 10.0–61.0 | 33.7–50.4 | |
| CD8 (%) | Mean±SD | 49.7±13.2 | 29.9±5.2 | <0.001 |
| | Range | 20.0–83.3 | 22.1–44.5 | |
| CD4/ CD8 ratio | Mean±SD | 0.76±0.58 | 1.40±0.15 | <0.001 |
| | Range | 0.16–3.05 | 1.08–1.64 | |
| CD19 (%) | Mean±SD | 8.1±5.8 | 11.3±3.9 | 0.015 |
| | Range | 0.0–21.0 | 6.1–20.4 | |

**Table 1.** Continued.

| Variables | | CVID group (Total=30) | Control group (Total=30) | *p*-value |
|---|---|---|---|---|
| CD19 +27 +IgD- memory cell (%) | Mean±SD | 4.0±3.9 | 31.3±8.8 | <0.001 |
| | Range | 0.3–16.8 | 10.5–45.6 | |
| CD19+27-IgD+ naïve cell (%) | Mean±SD | 87.6±9.8 | 54.6±11.3 | <0.001 |
| | Range | 60.7–98.6 | 30.1–77.5 | |
| Baseline IgG (mg/dl) | Mean±SD | 179.4±150.6 | 997.5±173.8 | <0.001 |
| | Range | 10.0–555.0 | 720.0–1420.0 | |
| Baseline IgA (mg/dl) | Mean±SD | 12.6±13.5 | 138.0±44.5 | <0.001 |
| | Range | 0.0–60.0 | 88.0–233.0 | |
| Baseline IgM (mg/dl) | Mean±SD | 13.2±9.5 | 123.5±35.1 | <0.001 |
| | Range | 0.8–35.0 | 65.0–199.0 | |

*p* > 0.05 is not significant (NS).

There was no statistical difference between CD19+27+IgD- switched memory B cells level and complications nor age of onset of symptoms. (Table 2)

**Table 2.** Relation of CD19+27-IgD+ naïve B cell and complications.

| | R | | *p* value |
|---|---|---|---|
| Age of onset | 0.087 | | NS |
| Frequency of infection | 0.159 | | NS |
| Diagnostic delay | 0.324 | | NS |
| | Yes | No | |
| Chronic diarrhea | 4.1±3.4 | 4.0±4.9 | NS |
| Upper respiratory tract infection (URTI) | 4.34 ± 4.01 | 3.10 ± 3.59 | NS |
| Pneumonia | 4.91 ± 4.43 | 2.75 ± 2.57 | NS |
| Bronchiectasis | 4.7±4.7 | 3.4±2.8 | NS |
| Granuloma | 2.8±2.6 | 4.2±4.1 | NS |
| Enteropathy | 4.35 ± 4.29 | 3.90 ± 3.78 | NS |
| Autoimmune complications | 4.9±3.4 | 3.7±4.1 | NS |
| Splenomegaly | 4.4±4.2 | 3.9±3.9 | NS |

*p* > 0.05 is not significant (NS).

CD3 was significantly lower in cases with chronic diarrhea ($p$=0.022). Baseline IgA and IgM follow up were significantly higher in cases with chronic diarrhea ($p$= 0.019 and $p$=0.014, respectively). CD19+27-IgD+ naïve B cells level was significantly lower in cases with bronchiectasis ($p$=0.049), low baseline serum IgG in the lymphadenopathy group, and higher level of CD3+ T cells in cases with splenomegaly as shown in Figure 2. While there was no significant difference in laboratory results in CVID patients presented with autoimmune diseases, Granulomas nor enteropathy.

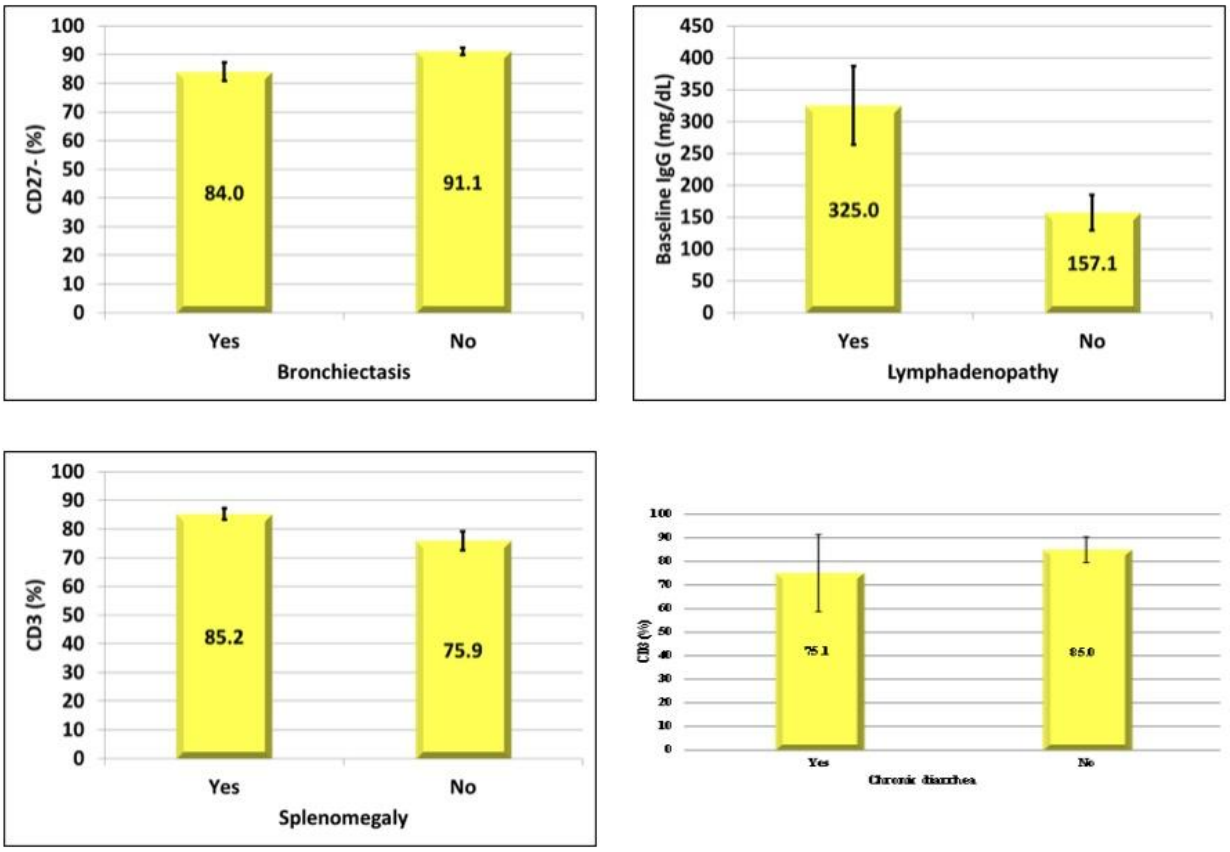


**Figure 2.** Correlation between clinical and immunological phenotypes among common variable immunodeficiency (CVID) patients.

## Discussion

Memory B cells are a B cell sub-type that are formed following a primary infection. In the last years, studies have proposed classifications of CVID patients based on numbers of memory B and class-switched memory B cells. Enumeration of memory cells in CVID was proposed in both assessment and as prognostic markers. In the last decades, few studies investigated the relation between class switched memory B cells and CVID phenotypes and complications.

Our aim, in this study, was to assess the correlation of immunoglobulins, T lymphocytes, and class switched memory B cells and naïve B cells between clinical and immunological phenotypes in Egyptian patients with CVID. In our study, there was a statistically significant difference regarding BMI among CVID patients being much lower than controls ($p$=0.001), this can be explained with the malnutrition secondary to recurrent infection, malabsorption

due to enteropathy and increased loss of nutrients due to diarrhea and sputum (bronchiectasis). These findings was consistent with those of Chen et al., 2023 who claimed that over 85% of their patients had low body weight and malabsorption and 85% had body mass indexes below 18 kg/m$^2$.[12]

In contrast to our study, studies by *Chapel and Patel, 2019* and *Blanco et al., 2019* found that reduced class switched memory B cell levels were associated with greater frequencies of non–respiratory tract infections and autoimmunity.[13,14] Also, the study by Sánchez-Ramón, 2008 found that reduced numbers of switched memory B cells (cutoff ≤0.55% of B cells) were an independent risk factor of granulomas, autoimmune diseases and splenomegaly ($p$<0.001).[15] In addition, the study by Ahn & Cunningham, 2009 found that several clinical conditions were associated with reduced switched memory B cells, including autoimmunity, splenomegaly, granulomatous disease, and lymphadenopathy.[16]

Regarding the relation between the level of naïve B cell and complications, naïve B cells were lower in CVID patients with bronchiectasis ($p$=0.049), there were no statistical differences between CD19+27-IgD+ naïve cell level and other CVID phenotypes. This was consistent with those of Kasahara et al., 2024 who found no statistical differences regarding naïve cell level in CVID patients with autoimmune diseases.[17] However, this was against findings of *Matson et al., 2021* who observed elevated levels of naïve B cells in CVID patients with pulmonary complications.[18]

Regarding other phenotypes, in our study, there were four patients who had lymphadenopathy (13.3%) and there was no relation between age of onset and duration of illness and development of lymphadenopathy. Also there was no relation between the development of lymphadenopathy and levels of CD3, CD4+, CD8+ T cells, CD19+CD27-IgD+ naïve and CD19+CD27+IgD- memory B cells except IgG level which was higher in CVID patients with lymphadenopathy ($P$=0.035). This was consistent with those of Costagliola & Consolini, 2021 who found lymphadenopathies were evidenced in 15–20% of the patients with common variable immunodeficiency,[19] and also consistent with those of Lima et al., 2022 and Janssen et al., 2021 who found that lymphadenopathy was observed in 30.2% and 30% of CVID patients respectively.[20,21] Also, several other studies found even higher prevalence (57%, 47.6%) of lymphadenopathy among CVID patients including Więsik-Szewczyk et al., 2021 and chen et al., 2023.[22,12]

In contrast to our findings, the study by Markocsy et al., 2024 found that 34% of the patients had lymphadenopathy and a lower percentage of switched memory B cells,[23] and Ahn and Cunningham, 2009 who also found switched CD27+ memory B cells were reduced in number and associated with lymphadenopathy.[16]

In our study, there were eight patients with splenomegaly (26.6%) this may be due to recurrent infection or secondary to autoimmune cytopenia, also, we found that CD3 was significantly higher in those patients with splenomegaly ($p$=0.024) but there was no significant relation between splenomegaly and memory B cell ($p$=0.786). In consistence to our study, findings of studies by Markocsy et al., 2024, Janssen et al., 2021, Lima et al., 2022 and Więsik-Szewczyk et al., 2021 found splenomegaly in (20%, 29%, 19.2%, 21%, receptively) of CVID patients.[20-23] The study by *Filion et al., 2019* also found that 20.3% of the patients had splenomegaly and those patients had low level of CD19+CD27+IgD− class switched memory B cell. [24] This was in contrast to that of *Chen et al., 2023* who found splenomegaly in 52.4% of CVID patients but their study had potential biases due to a limited number of patients.[12] Also, the study by Sánchez-Ramón, 2008 found that patients with switched memory B cells of ≤0.55% had splenomegaly 4.4 fold higher [11] but, there was no relation between IGs level and splenomegaly. Also, the study by Hana et al., 2006 found that reduced percentage of CD19+CD27+IgD− memory B cell but serum immunoglobulin levels was not associated with a significantly higher prevalence of splenomegaly ($p$ = 0.001). [25]

In conclusion, we concluded that low level of memory B cell and high level of naïve B cell are

a characteristic feature of CVID. Reduced naïve B cell number was associated with development of bronchiectasis, reduced CD3 count and elevated IgA was associated with development of diarrhea, high IgG also was associated with lymphadenopathy, and high CD3 may be a marker for splenomegaly.

## Author Contributions

AE; conceptualized the study. AE and MF; designed the study. MF; designed the data collection tool. AE and MIMH; carried out data collection. YGM; performed the data analysis and interpretation. MAE; wrote the original draft, and ENO; revised the article before submission.

## Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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## Ethical approval

The study protocol was reviewed and approved by the Research Ethics Committee of the Faculty of Medicine, Ain Shams University (FMASU MD 266, 2022).

## Informed consent

All participants provided their written informed consent before sample collection.

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