

Original Research Article


# Hematological profile of people living with HIV infection in Government Dharmapuri Medical College, Dharmapuri

M. S. Shruthi<sup>1</sup>, T. Elavarasan<sup>2\*</sup>, R. D. Puvitha<sup>2</sup>

<sup>1</sup>Assistant Professor <sup>2</sup>Associate Professor

Department of Pathology, Government Dharmapuri Medical College, Dharmapuri, India

\*Corresponding author email: [elasu@yahoo.com](mailto:elasu@yahoo.com)

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## Abstract

**Introduction:** The HIV epidemic is a convincing illustration of the balance between the power of science and the humanism of modern medicine. The epidemic is so serious that between 1981 and 2000, 21.8 million people had died of HIV/AIDS. Clinically significant hematologic abnormalities are common in HIV infection. These abnormalities may occur as a result of HIV infection itself, as sequelae of HIV related opportunistic infections or malignancies or as a consequence of therapies used for HIV infection and associated conditions.

**Aim of the Study:** To analyze the hematological profile of people living with HIV/AIDS (PLHA's) and to identify the possible correlation between WHO clinical stage and hematological abnormalities.

**Materials and methods:** All patients with HIV infection attending Govt. Dharmapuri Medical College Hospital, Dharmapuri during the study period were evaluated for the conditions which could alter the Hematological parameters and if found so, they were excluded from the study. Those included in the study were investigated for Hb% total count, differential count, ESR and platelet count. CD4 count done by flow cytometric analysis was obtained.

**Results:** The mean total count was found to be  $587 \pm 2210$  cells /  $\text{mm}^2$  out of the 100 patients 41 of them had leucopenia. In our study the differential count distribution showed neutropenia in 30 patients, lymphocytopenia in 30 patients and monocytopenia in 20 patients. The median CD-4 count was 89 cells/microliter. Almost all the patients enrolled in our study had CD4 count  $<200$ /microliter with only a minor fraction having counts  $>200$ /microliter. All patients who had lymphocytopenia were having CD4 count  $<200$ /microliter. This establishes the relationship between lymphocytopenia and low CD4 counts. The mean platelet count was  $1.56 \pm 0.35$  lakh/ $\text{mm}^3$  thrombocytopenia was found

in 40 patients. The mean value ESR was  $24.63 \pm 10.92$  mm /hr. Elevated ESR was found in 71%. ESR has not much of diagnostic value and therefore can be elevated in any chronic inflammation (or) infection. Hence, this finding may not be that significant.

**Conclusion:** Majority of them were in stage III (55%) and had CD4 count  $<200$ /microliter (87%). Leucopenia was found in 41% of them. Neutropenia was detected in 29%. Thrombocytopenia was found in 40%. Anemia and elevated ESR detected in 79% and 71% respectively.

### Key words

HIV infection, CD4 count, Leucopenia, Thrombocytopenia, Elevated ESR.

### Introduction

Disorders of the hematopoietic system including lymphadenopathy, anemia, leucopenia, and/or thrombocytopenia are common throughout the course of HIV infection and may be the direct result of HIV, manifestations of secondary infections and neoplasms, or side effects of therapy [1]. Direct histologic examination and culture of lymph node or bone marrow tissue are often diagnostic. A significant percentage of bone marrow aspirates from patients with HIV infection have been reported to contain lymphoid aggregates, the precise significance of which is unknown. Initiation of HAART will lead to reversal of most hematologic complications that are the direct result of HIV infection [2]. Hematologic abnormalities secondary to HIV infection include anemia, neutropenia, thrombocytopenia, venous thrombo embolism, hemophagocytic syndrome, AIDS - related lymphoma including primary effusion lymphoma, castlemann's disease and rarely Hodgkin's disease and myeloma. Anemia is common in HIV-infected individual's occurring in approximately 10 to 20% at initial presentation and diagnosed in approximately 70 to 80% of patients over the course of disease [3, 5]. The incidence increases with the clinical stage of disease. It is dependent of CD4 count and viral load i.e. frequency and severity of anemia appear to correlate with HIV related factors. Another important cause of hypo proliferative anemia in patients with HIV infection is medications particularly ART drug zidovudine (AZT). AZT, the first licensed anti-retroviral agent is uniformly associated with macrocytosis (Mean cell volume  $>100$ fl), which can be used as an

objective indication that the patient has been compliant with this medication [4]. Transfusion dependent anemia (Hob  $<8.5$ g/dl) has been reported in approximately 30% of patients with full blown AIDS who were receiving AZT at doses of 600mg/day. Incidence of severe anemia is only 1% when the same dose of AZT is used in patients with asymptomatic HIV disease. Infection of the marrow by Parvo virus B19 is another cause of hypoproliferative anemia in HIV-infected patients, resulting in specific infection of the pronormoblast [5]. Although marrow failure affecting all three cell lines has been described with parvovirus B19 infection, a pure red cell aplasia is the usual consequence. Approximately 85% of adults have serologic evidence of prior parvo virus infection, but among HIV infected patients it is only 64%. The diagnosis of parvovirus B19 can be made on marrow examination, revealing giant pronormoblasts with clumped basophilic chromatin and clear cytoplasmic vacuoles. Diagnosis can be confirmed by in situ hybridization using sequence-specific DNA probes [6]. Therapy for parovirus induced red cell aplasia consists of infusion of intravenous  $\gamma$ -globulin (IVIG) from plasma donors [7].

### Materials and methods

All patients with HIV infection attending Govt. Dharmapuri Medical College Hospital, Dharmapuri from December 2016 - May 2017 are included in the study.

### Inclusion criteria

- All patients with HIV infection.

- HIV infection proven by ELISA and western blot assay.

#### Exclusion criteria

- Chronic infection like tuberculosis.
- Alcoholics
- Worm infestations.
- Chronic kidney disease.
- Drug intake (phenytoin)
- Patient on anti-retroviral therapy.

All patients with HIV infection attending Govt. Dharmapuri Medical College Hospital, Dharmapuri during the study period were evaluated for the conditions which could alter the Hematological parameters and if found so, they were excluded from the study. Those included in the study were investigated for Hb% total count, differential count, ESR and platelet count. CD4 count done by flow cytometric analysis was obtained. They were staged as per the WHO clinical staging given by the National AIDS control organization (NACO).

#### Statistical Analysis

Data entry and analysis done values are presented as Mean+ Standard deviation and median +Q (interquartile range) as appropriate. Percentages were used to describe the proportions of discrete variables. A p value of <0.05 was considered statistically significant.

#### Results

100 patients with HIV infection were included in the study after excluding for all possible parameters that could affect the blood cell counts. Before the initiation of anti-retroviral therapy they were investigated for Hb, total count, differential count, CD4 count, platelet count and erythrocyte sedimentation rate (ESR).

The mean total count was found to be  $587 \pm 2210$  cells /  $\text{mm}^2$  out of the 100 patients 41 of them had leucopenia (**Table – 1**).

In our study the differential count distribution showed, neutropenia in 30 patients,

lymphocytopenia in 30 patients and monocytopenia in 20 patients (**Table – 2**).

**Table – 1:** Total count distribution.

TOTAL COUNT (CELLS/MM <sup>3</sup> )	N	%
2000-3000	9	9%
3001-4000	22	22%
4001-5000	11	11%
5001-6000	13	13%
6001-7000	8	8%
7001-8000	14	14%
8001-8000	16	16%
9001-10000	7	7%

**Table – 2:** Differential count.

White Blood Cells	Mean
Neutrophils	54+12.6%
Lymphocytes	29.8+8.3%
Monocytes	6.76+4.1%
Eosinophil's	2.36+3.79%
Basophils	0%

**Table – 3:** CD-4 count distribution.

CD4 Count (Cells/microlitre)	n	%
<200	87	87.0
200-499	13	13.0
> 500	0	0

**Table - 4:** Platelet count distribution.

Platelet Count (Lakh/mm <sup>3</sup> )	n	%
<0.5	0	0
0.5 - 0.99	21	21.0
1.00 - 1.49	19	19.0
1.50 - 1.99	37	37.0
2.00 - 2.49	15	15.0
2.50 - 2.99	7	7.0
> 3	1	1.0
The mean platelet count was $1.56 + 0.35$ lakh / $\text{mm}^3$		

The median CD-4 count was 89 cells/microliter. Almost all the patients enrolled in our study had CD4 count <200/microliter with only a minor fraction having counts >200/microliter (**Table – 3**).

**Table – 5:** Erythrocyte sedimentation rate.

Elevated ESR (mm/hr.)	n	%
Male	41	21.0
Female	30	20.0

**Table - 6:** Hemoglobin distribution.

Hemoglobin (g/dl)	Male	Female	%
8.0 - 8.99	1	1	2.0
9.0 - 9.99	10	3	13.0
10.0 - 10.99	12	11	23.0
11.0 - 11.99	16	9	25.0
12.0 - 12.99	16	13	29.0
13.0 - 13.99	8	0	08.0

Thrombocytopenia was found in 40 patients (Table – 4).

The mean value ESR was  $24.63 \pm 10.92$  mm /hr. Elevated ESR was found in 71 cases. ESR has not much of diagnostic value and therefore can be elevated in any chronic inflammation (or) infection. Hence, this finding may not be that significant (Table – 5).

The mean Hob value was  $11.40 \pm 1.36$  g/dl (Table – 6).

## Discussion

The observation made in the PLHAs with respect to Hemoglobin total count differential count, platelet count, CD4 count, erythrocyte sedimentation rate and WHO stages were analysed and the following inferences were drawn [8]. The mean age of the study population was  $36.85 \pm 6.29$  yr. About 56% of them were in 31-40 year age group i.e., age at the diagnosis of HIV infection. As per the data released by Tamil Nadu State AIDS control society (TANSAC) about 50% of HIV infected patients belonged to 30 - 49 years at the time of diagnosis .In the western countries about 36% of them were in the 35-44 years age group at the time of diagnosis, which happens to be the Major age group affected. Our data is in concordance with these data [9]. The mean total count in our study was  $5872 \pm 2210$  cells / mm<sup>3</sup>. The total count varied

between 2000 to 10,200 cells/mm<sup>3</sup>. 41% of them had leucopenia. The percentage of PLHA's with leucopenia at the time of diagnosis was found to 16% and 25% by Amballi, et al. and Amanda, et al. respectively. Erhabor et al, in his study on the effect of anti-retroviral therapy and hematological profile of people PLHA's found leucopenia in 62% [10]. Higher incidence of leucopenia in our study may be due to the diagnosis of HIV infection at advanced stage. This again reiterates the fact that diagnosis of HIV infection at advanced stage would increase the incidence of complication. The awareness campaigns and counseling programs have to be intensified, so that the diagnosis of HIV infection can be done at earlier asymptomatic stage. In our study more patients in advanced stage of HIV infection were having neutropenia. This poses them at the increased risk of developing opportunistic infections [11]. The mean lymphocyte count was  $29.82 \pm 8.3\%$  and the median CD4 count was 89 cells/micro liters. CD4 count varied between 6 cells to 459 cells/ml Lymphocytopenia was found in 30% of the PLHA's and CD4 count <200 cells in 87% of the patients. Platelet count varied between 0.5 to 2.8 lakh/mm<sup>3</sup> and the mean count was  $1.56 \pm 0.35$  lakh/mm<sup>3</sup> [12]. Thrombocytopenia was found in 40% of the PLHA's in our study. In his study conducted among HIV positive pregnant women khandekar et al, found thrombocytopenia in 9% of them. Pechere et al detected thrombocytopenia in 40% of HIV infected patients during the course of the disease and as the first symptoms or sign of HIV infection in approximately 10%. Murphy, et al. concluded that thrombocytopenia was found in 30% (6 of 20) of patients with advanced HIV disease and 8% (5 of 59) in those with asymptomatic HIV infection. In our study it was found in 38% (26 of 67) and 42% (14 of 33) of advanced HIV disease and asymptomatic HIV infection respectively [13]. Savona, et al. concluded that in HIV infection, early stages may have decreased platelet count due to decreased survival and in late advanced disease due to marrow failure. The mean Hemoglobin in our study was  $11.4 \pm 1.36$  g/dl and it varied between 9g/dl to 14g/dl [14]. In our study 79

patients had anemia out of which 55 (87.3%) were males and 24 (64.9%) were females. The occurrence of anemia was found to be more common among males. The prominent elevation of ESR in all these patients is not surprising. Although ESR is neither sensitive nor specific when used as a general screening test, it is usually elevated in the presence of infectious disease and chronic illness [15].

### Conclusion

100 PLHA's were analyzed for the Hematological abnormalities in HIV/Aids. Majority of them were in stage III (55%) and had CD4 count <200/microliter (87%) [16]. Leucopenia was found in 41% of them. Neutropenia was detected in 29% [17]. Thrombocytopenia was found in 40%. Anemia and elevated ESR detected in 79% and 71% respectively [18]. Lymphocytopenia was detected in 30% of PLHA's who also had low CD 4 count as per the WHO documents [19]. The analysis of correlation between WHO staging and hematologic abnormalities revealed statistically significant relation only with anemia [20].

### References

1. Kent A Sepkowitz. AIDS - The first 20 years. The New England journal of Med., 2001; 344(23): 1764-1722.
2. Coyle TE. Management of HIV infected patient, Part II. Med Clin North Am., 1997; 81: 449-470.
3. Mitsuyasu R. AIDS Clin Review 1993/4, Pg.189, Marcel Dekker, New York, 1993.
4. Zon LI, Arkin C, Groopman JE. Hematologic Manifestations of the HIV. Semin Hematol., 1988; 25: 208.
5. Sullivan PS, Hanson DL, Chu SY, et al. Epidemiology of anemia in HIV infected persons. Results from the multistate Adult and Adolescent spectrum of HIV disease surveillance project. Blood, 1998; 91: 301.
6. Levine AM, Berhane K, Masri - Lavine L. Prevalence and correlates of anemia in a large cohort of HIV infected women; women's interagency HIV study. Jour of acquired immune defic syndr., 2001; 26: 28.
7. Mocroft A, Kirk O, Brarton SE, et al. Anemia is an independent predictor of clinical prognosis in HIV infected person from across Europe, AIDS, 1999; 13: 943.
8. Abrams DI, Steinhart C, Franscino R. Anemia in HIV infected patients. Int Jour of STD/AIDS, 2000; 11: 659-665.
9. Moore JD. Anemia and survival in HIV infection. Jour of Acquire immune defic Synd., 1998; 19: 29-33.
10. Kerr JR. Parvovirus B19 infection. Eur J Clin Microbiol Infect Dis., 1996; 15: 10-29.
11. Collier AC, Kalish LA, Busch MP, et al. Leucocyte reduced red blood cell transfusion in patients with anemia and HIV infection: The viral activation transfusion study; a randomized trial. JAMA, 2001; 285: 1592-1601.
12. Spivak JL, Barnes DC, Fuchs, et al. Serum Immunorec erythropoietin in HIV infected patients. JAMA, 1989; 261: 3104-3107.
13. Seneviratne Ls, Tulpule A, Mummanani M, et al. Clinical, immunological and pathologic correlates of bone marrow involve in 253 patients with AIDS related lymphoma. Blood, 1998; 92: 244A.
14. Walker RE, Parker RI, Kovacs JA, et al. Anemia and erythropoiesis in patients with AIDS and Kaposi sarcoma treated with zidovudine. Ann intern Med., 1988; 108: 372.
15. Richman DD, Fischl MA, Grieco MH, et al. The toxicity of AZT in the treatment of AIDS and AIDS related complex. A double blind, placebo controlled trial. N Engl. J. Med., 1987; 317: 192.
16. Anderson LJ. Human Paroviruses. J Infect Dis., 1990; 161: 603.

17. Frickhofen N, Abkowitz JL, Safford, et al. Persistent B19 parvo virus infection in patients infected with HIV- A treatable cause of anemia in AIDS. *Ann intern med.*, 1990; 926.
18. Ravick MU, Espina B, Mocharunk R, et al. Thrombotic thrombocytopenic purpura in HIV infection: A report of three cases and review of literature. *Am J Hematol.*, 1992; 40: 103.
19. Sasadeuz J, Buchanan M, Speed B. Reactive hemophagocytic syndrome in HIV infection. *Jour of infect dis.*, 1990; 20: 65.
20. Sproat CL, Pantanowitz L, LUCM, et al. HIV-associated hemophagocytosis with iron deficiency anemia and massive splenomegaly. *Clin infect dis.*, 2003; 37: 170.