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# Measuring the Impact of Schistosomiasis Infection on Different Blood Parameters

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**Abstract.**
**Background**.
Schistosoma is a genus of trematodes, ***Schistosoma*** spp., commonly known as blood-flukes and bilharzia, cause the most significant infection of humans by flatworms and are considered by the World Health Organization (WHO) as second in importance only to malaria.
**Objective of the study** .This study was carried out in Sudan in the period from December 2009 to February 2010 to assess effects *Schistosomamansoni* on hemoglobin (Hb) level, red blood cell count (RBCs count), total leucocyte count (TLC), paced cell volume (PCV), mean cell volume (MCV), mean cell hemoglobin (MCH), mean cell hemoglobin concentration (MCHC), and total platelet count .
**Study design** .One hundred *Schistosomamansoni* infected patients included in this study ( 77 males , and 23 females).More fifty samples were collected from normal persons and used as control. Density of parasitemia was calculated using Kato Katz technique. 5 ml venous anticoagulated blood were drawn from each participant . blood was analysed for aboved parameters using Mythic 18 cell counter.
**Results**. the results showed that the means of different blood parameters were as followed : Hb:13 g/dl, PCV:40%, MCV:78 fl, RBC:5.2X $10^{12}$/L, MCH: 25pg, MCHC: 31g/dl, TLC: 7.4x$10^{9}$/L, and Total Platelet count:245x$10^{9}$/L in male group, and Hb: 12g/dl, PCV: 37%, MCV: 74 fl, TRBC: 5x$10^{12}$/L, MCH: 23 fl, MCHC: 31g/dl, TLC: 6.7x$10^{9}$, and total platelet count: 240x$10^{9}$/L for female group.
**Conclusion**. The study concluded that there a reduction in most parameters when compared to control group. Also this study reported a significant reverse association between density of parasitemia in all parameters except TLC and TRBC.
**Key words**. *Schistosoma mansoni, blood parameters, Halfa Aljadeeda town, Sudan*

## Introduction

Schistosoma is a genus of trematodes, *Schistosoma* spp., commonly known as blood-flukes and bilharzia, cause the most significant infection of humans by flatworms (schistosomiasis) and are considered by the World Health Organization as second in importance only to malaria, with hundreds of millions infected worldwide. Adult worms parasitize mesenteric blood vessels. Eggs are passed through urine or feces to fresh water, where larval stages can infect a new host by penetrating the skin. (1) There are many type of *Schistosoma* infectingpeople such as*S.japonicum,S. mekongi, S. haematobium*and*Schistosoma mansoni*, which found in Africa, Brazil, Venezuela, Suriname, the lesser Antilles, Puerto Rico, and the Dominican Republic. It is also known as *Manson's blood fluke* or *swamp fever*. Freshwater snails of the *Biomphalaria* genus are an important host for this trematode.(2)

**Table .1. Human Schistosomes**

| Scientific Name | First Intermediate Host | Endemic Area |
|---|---|---|
| ***Schistosoma mansoni*** | *Biomphalaria* spp. | Africa, South America, Caribbean, Middle East |
| ***Schistosoma haematobium*** | *Bulinus* spp. | Africa, Middle East |
| ***Schistosoma japonicun*** | *Oncomelania* spp. | China, East Asia, Philippines |
| ***Schistosoma intercalatum*** | *Bulinus* spp | Africa |

Adult schistosomes share all the fundamental features of the digenea. They have a basic bilateral symmetry, oral and ventral suckers, a body covering of a syncytialtegument, a blind-ending digestive system consisting of mouth, oesophagus and bifurcated caeca; the area between the tegument and alimentary canal filled with a loose network of mesodermcells, and an excretory or osmoregulatory system based on flame cells. Adult worms tend to be 10-20 mm long and use globins from their hosts' hemoglobin for their own circulatory system.(3)

 

*Schistosoma mansoni* is a significant parasite of humans, one of the major agents of schistosomiasis. Of the trematodes, schistosomes are atypical in that the adult stages have two sexes (dioecious) and are located in blood vessels of the definitive host. Most other trematodes are hermaphroditic and are found in the intestinal tract or in organs, such as the liver. The lifecycle of schistosomes includes two hosts: a definitive host (i.e., man) where the parasite undergoes sexual reproduction, and a single intermediate snail host where there are a number of asexual reproductive stages. *S. mansoni* is named after Sir Patrick Manson, who first identified it.(2)

Life Cycleof *Schistosoma mansoni* starts after the eggs of the human-dwelling parasite are emitted in the faeces and into the water, the ripe miracidium hatches out of the egg. The hatching happens in response to temperature, light and dilution of faeces with water. The miracidium searches for a suitable freshwater snail (*Biomphalaria glabrata*, *B. straminea* or *B. tenagophila*) to act as an intermediate host and penetrates it. Following this, the parasite develops via a so-called mother-sporocyst and daughter-sporocyst generation to the cercaria. The purpose of the growth in the snail is the numerical multiplication of the parasite. From a single miracidium result a few thousand cercaria, every one of which is capable of infecting man. The cercaria emerge from the snail during daylight and they propel themselves in water with the aid of their bifurcated tail, actively seeking out their final host. When they recognise human skin, they penetrate it within a very short time. This occurs in three stages, an initial attachment to the skin, followed by the cercaria creeping over the skin searching for a suitable penetration site, often a hair follicle, and finally penetration of the skin into the epidermis using proteolytic secretions from the cercarial post-acetabular, then pre-acetabular glands. On penetration, the head of the cercaria transforms into an endoparasitic larva, the schistosomule. Each schistosomule spends a few days in the skin and then enters the circulation starting at the dermal lymphatics and venules. Here they feed on blood, regurgitating the haem as haemozoin. The schistosomule migrates to the lungs (5–7 days post-penetration) and then moves via circulation through the left side of the heart to the hepatoportal circulation (>15 days) where, if it meets a partner of the opposite sex, it develops into a sexually mature adult and the pair migrate to the mesenteric veins(Schistosoma mansoni (Genome Project, 2007) .Schistosome eggs, which may become lodged within the hosts tissues, are the major cause of pathology in schistosomiasis. Some of the deposited eggs reach the outside environment by passing through the wall of the intestine; the rest are swept into the circulation and are filtered out in the periportal tracts of the liver resulting in periportal fibrosis. Onset of egg laying in humans is sometimes associated with an onset of fever (Katayama fever). This "acute schistosomiasis" is not, however, as important as the chronic forms of the disease. For *S. mansoni* and *S. japonicum* these are "intestinal" and "hepatic schistosomiasis", associated with formation of granulomas around trapped eggs lodged in the intestinal wall or in the liver, respectively. The hepatic form of the disease is the most important, granulomas here giving rise to fibrosis of the liver and hepatosplenomegaly in severe cases. Symptoms and signs depend on the number and location of eggs trapped in the tissues. Initially, the inflammatory reaction is readily reversible. In the latter stages of the disease, the pathology is associated with collagen deposition and fibrosis resulting in organ damage that may be only partially reversible.

Many individuals do not experience symptoms. If symptoms do appear, it usually takes four to six weeks from the time of infection. The first symptom of the disease may be a general ill feeling. Within twelve hours of infection, an individual may complain of a tingling sensation or light rash, commonly referred to as "swimmer's itch", due to irritation at the point of entrance. The rash that may develop can mimic scabies and other types of rashes. Other symptoms can occur two to ten weeks later and can include fever, aching, cough, diarrhea, or gland enlargement. These symptoms can also be related to avian schistosomiasis which does not cause any further symptoms in humans .

**Materials & Methods**

A total number of hundred patients of *Schistosoma mansoni* were included in this study. The consent of the selected individuals to the study was taken after being informed with all detailed objectives of the study and its health emphasis in the future. All Schistosoma infected individuals were treated under supervision of a physician. In Patients with other diseases, pregnancy, and presence of any other parasite in the stool with Schistosoma eggs(co-infection) were not included in this study. Stool samples were collected in screw capped containers labeled with code number.

Five ml venous blood collected using sterile disposable plastic syringe after cleaning the venipuncture area with 70% ethanol. 2.5 ml were dispensed in EDTA containers for CBC. The remaining 2.5 ml were placed in plain containers from which serum was separated (by centrifugation at 2500 rpm in bench centrifuge) and transferred in eppendorf tubes. A drop of normal saline was placed in a clean glass slide, a small piece of stool added to the saline, mixed well, covered with cover glass, then examined under microscope using times 10 and times 40 objective lenses.

 

Positive result was indicated by presence of *Schistosoma mansoni* eggs in the preparation, while absence of the eggs indicated negative result. The negative results were confirmed by duplicating the test. Cellophane faecal thick smear (kato-katz) examination technique has been proved to be an efficient means of diagnosis of schistosomiasis and intestinal helminthes. In this study this test used to measure the density of parasitaemia. The complete blood count and blood film examination are usually indicate where ever there is any abnormalities of globins chain synthesis .Here we have used the automated method by usingMYTHIC 18 automated hematological analyzer. The counting of the cellular elements in a blood sample was done with the impedancemetry technique. This technique was based on the modification of the impedance of a calibrated aperture soaking in an electrolyte and going through a constant course delivered by two electrodes located on both sides of the aperture.

A vacuum applied on a side of the aperture allows the cells passage. They oppose their physical volume to the course passage. A voltage impulse is registered at the electrodes terminal. The height of this impulse is proportional to the cell volume.

**Results.**

**Table .2. Full blood count for male patients and control. Most parameters were low in patients of both sexes.**

| parameters | Mean | | |
|---|---|---|---|
| | Patient male | Control male | P. value |
| Hemoglobin concentration (g/dl) | 13.052 | 14.480 | 0.017 |
| Packed cell volume (%) | 40.816 | 39.750 | 0.603 |
| Mean cell volume (fl) | 78.242 | 85.530 | 0.001 |
| RBCs count($10^6$/µl)) | 5.2284 | 4.9460 | 0.183 |
| Mean cell hemoglobin (pg) | 25.023 | 29.070 | 0.000 |
| Mean cell hemoglobin concentration (g/dl) | 31.994 | 33.400 | 0.000 |
| Total leukocyte count (($10^3$/µl)) | 7.4065 | 6.6100 | 0.366 |
| Total platelet count ($10^3$/µl) | 245.3636 | 265.4000 | 0.523 |

**Table 3. The full blood count, serum iron and serum ferritin for female patients and control**

| parameters | Mean | | |
|---|---|---|---|
| | Patient female | Control female | P. value |
| Hemoglobin concentration (g/dl) | 12.052 | 12.109 | 0.926 |
| Packed cell volume (%) | 37.796 | 38.773 | 0.550 |
| Mean cell volume(fl) | 74.817 | 83.191 | 0.003 |
| RBCs count($10^6$/µl)) | 5.0335 | 4.6727 | 0.022 |
| Mean cell hemoglobin (pg) | 23.835 | 26.009 | 0.050 |
| Mean cell hemoglobin concentration (g/dl) | 31.778 | 31.155 | 0.121 |
| Total leukocyte count (($10^3$/µl)) | 6.7522 | 6.5091 | 0.800 |
| Total platelet count ($10^3$/µl) | 240.0870 | 341.2727 | 0.000 |

 

**Table .4 . Full blood count, and density of parasitaemia. All parameters except red blood cells count were affected by density of parasitemia**

| parameters | Mean | | |
|---|---|---|---|
| | Mild | Sever | P. value |
| Hemoglobin concentration (g/dl) | 13.406 | 11.692 | 0.000 |
| Packed cell volume (%) | 41.668 | 36.904 | 0.001 |
| Mean cell volume (fl) | 79.683 | 73.164 | 0.000 |
| RBCs count($10^6$/µl)) | 5.2396 | 5.0448 | 0.221 |
| Mean cell hemoglobin (pg) | 25.572 | 23.144 | 0.000 |
| Mean cell hemoglobin concentration (g/dl) | 32.130 | 31.564 | 0.004 |
| Total leukocyte count (($10^3$/µl)) | 7.5319 | 6.8840 | 0.345 |
| Total platelet count ($10^3$/µl) | 217.1489 | 270.4400 | 0.005 |
| Serum iron (µg/dl) | 65.8298 | 40.2800 | 0.046 |
| Serum ferritin (µg/l) | 162.1915 | 123.4800 | 0.010 |

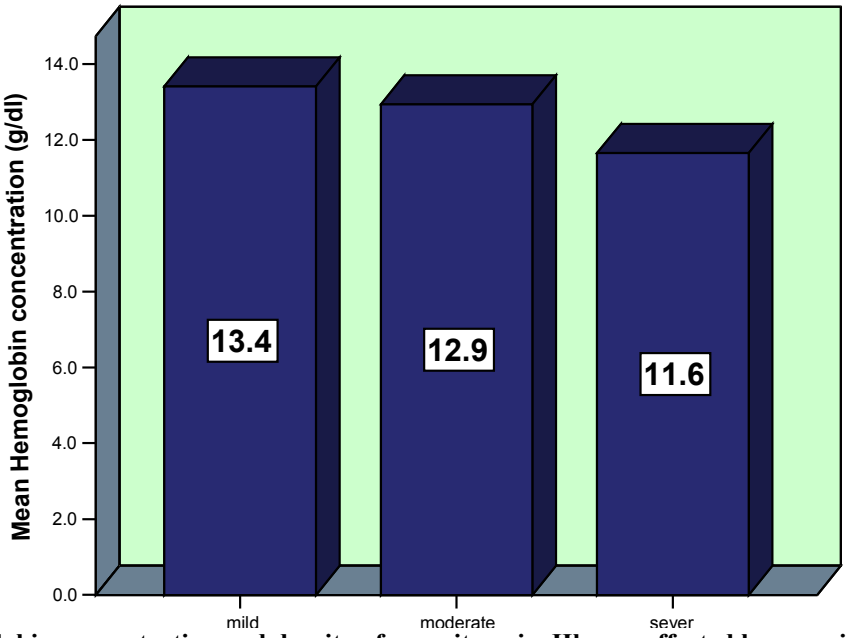


**Figure .1 Hemoglobin concentration and density of parasitaemia. Hb was affected by parasitemia**

 

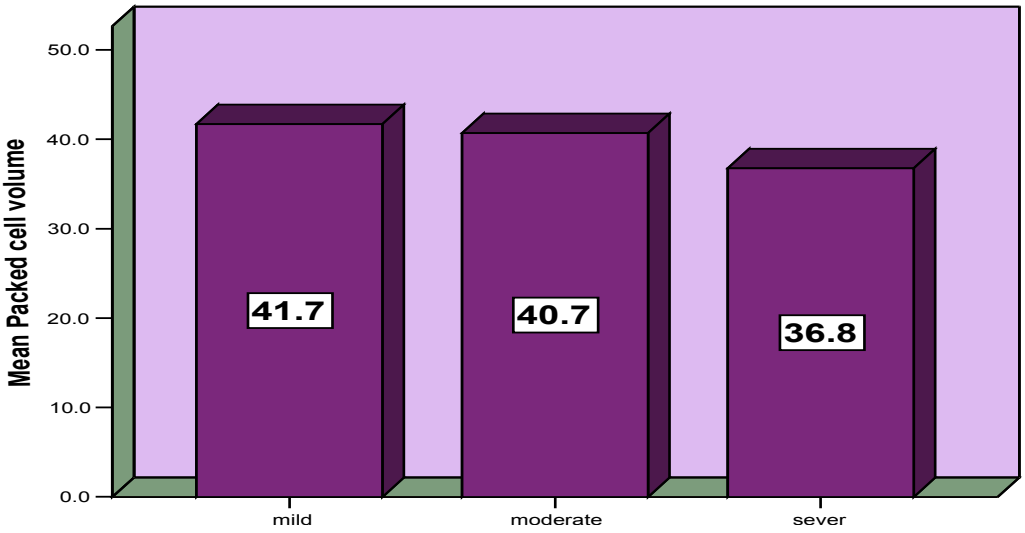


**Figure 3.3 packed cell volume (%) and density of parasitaemia. PCV was reduced due to the affect of parasitemia**

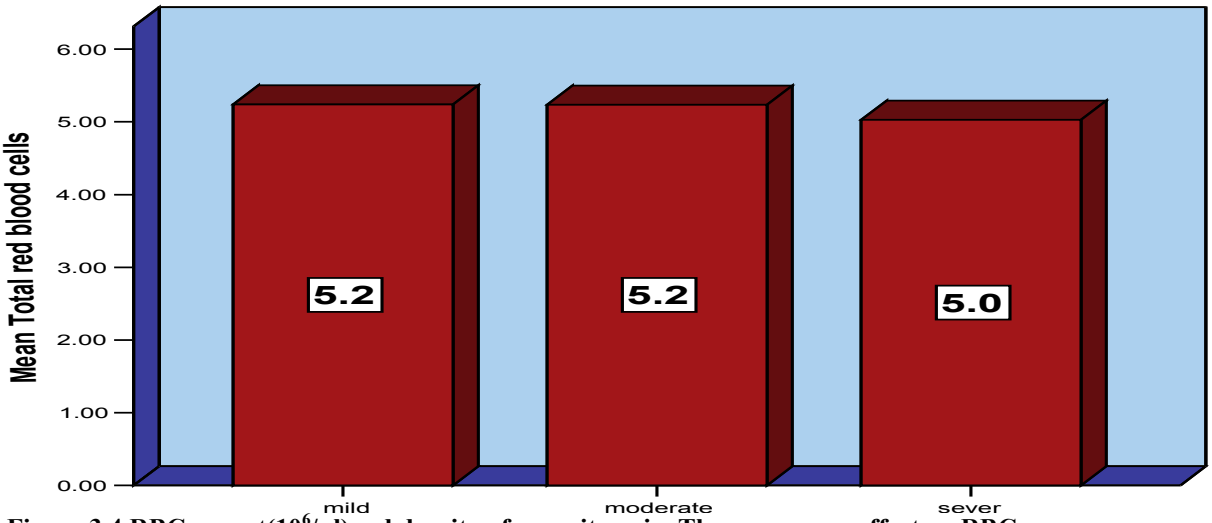


**Figure 3.4 RBCs count($10^6$/µl)and density of parasitaemia. There was no effect on RBCs cou**

 

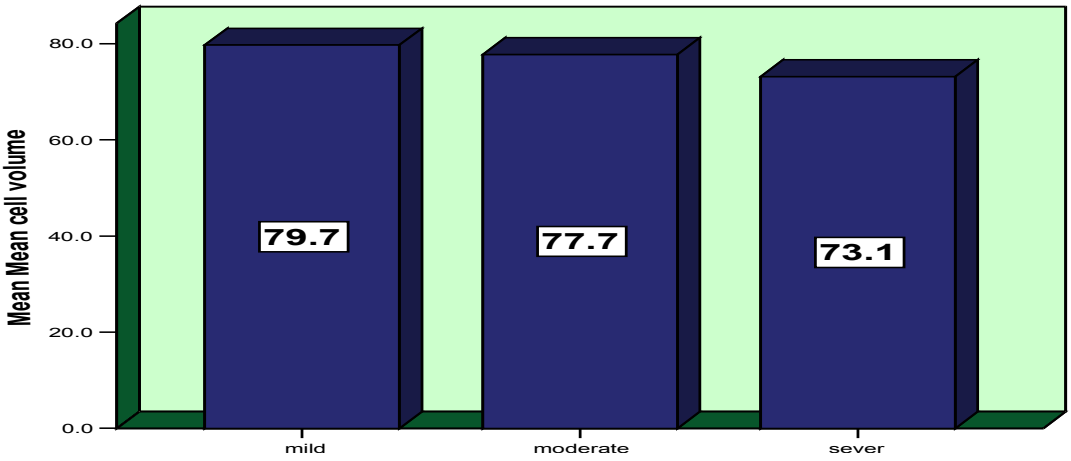


**Figure 3.5 mean cell volume (fl) and density of parasitaemia. The MCV was reversibly affected by density of parasitemia**

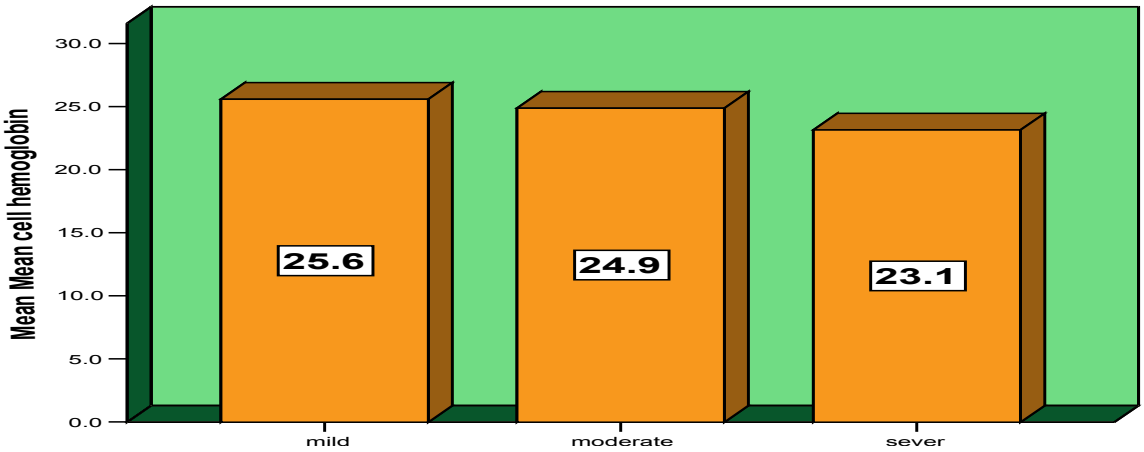


**Figure 3.6 mean cell hemoglobin (pg) and density of parasitaemia. The MCH was reduced when parasitemia is sever**

 

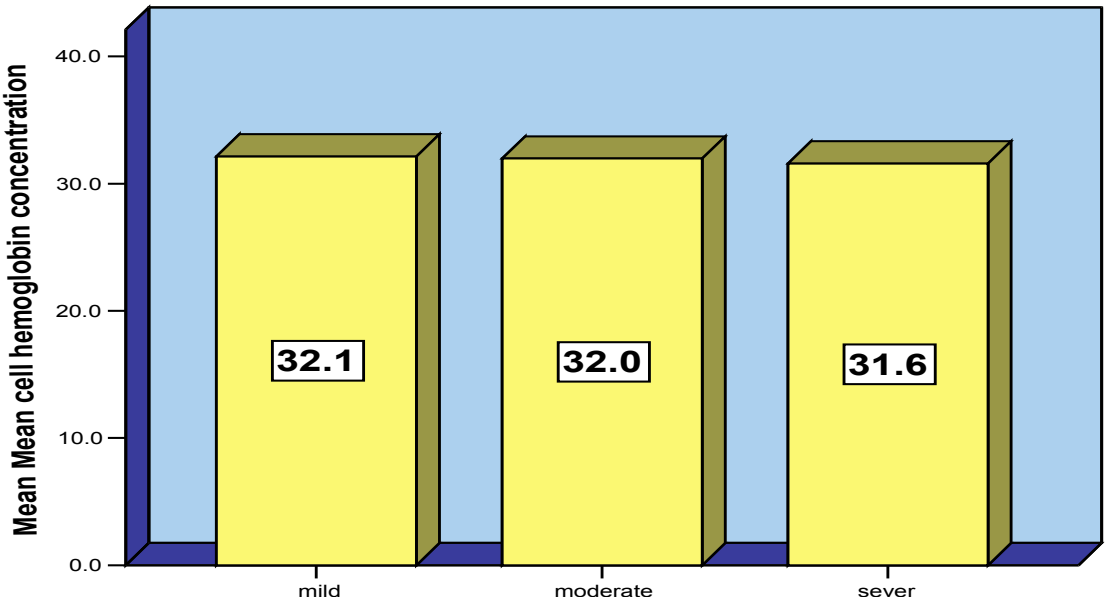


**Figure 3.7 mean cell hemoglobin concentration (g/dl) and density of parasitaemia. MCHC was affected by the density of parasitemia**

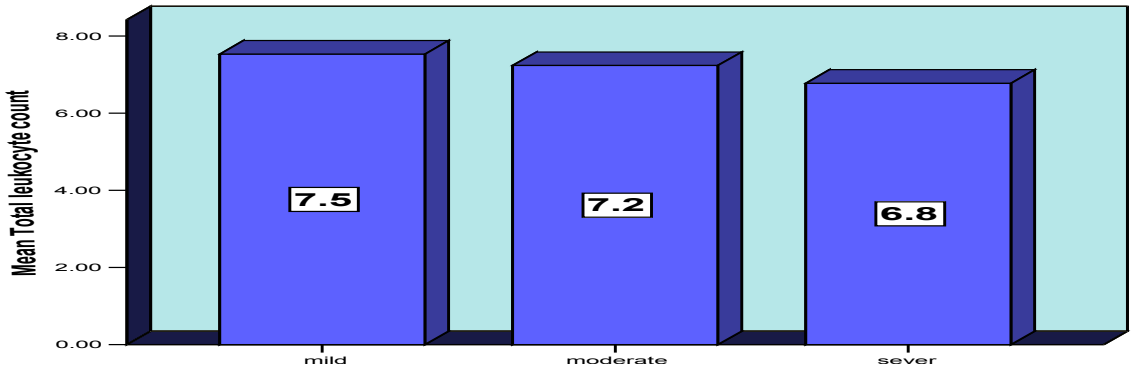


**Figure 3.8 total leukocytes count ($10^3$/µl) and density of parasitaemia. Density of parasitemia showed no effect on TLC.**

 

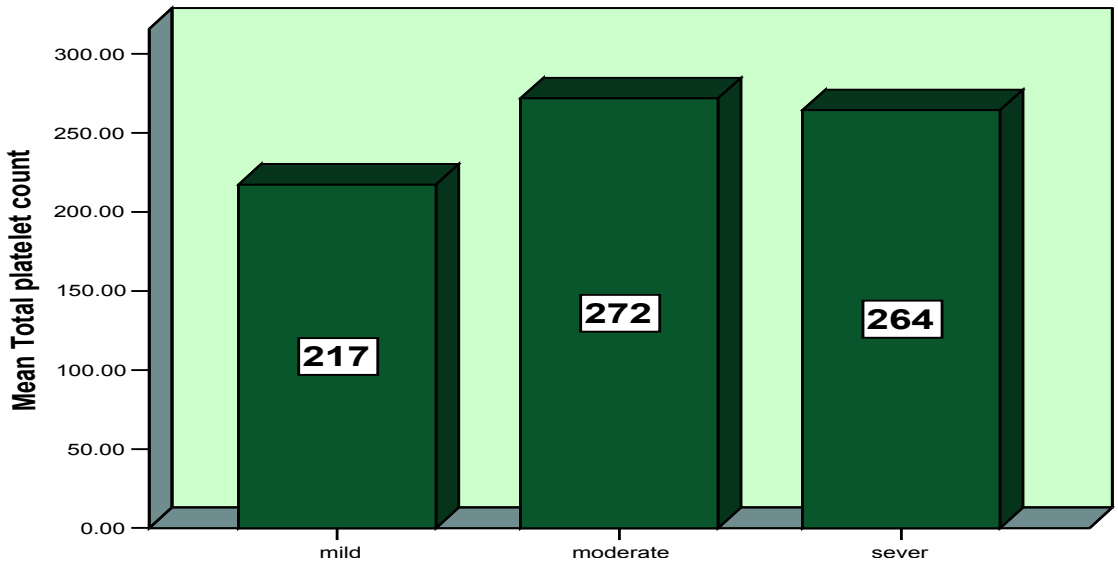


**Figure 3.9 total platelets count and density of parasitemia. Platelets count was not affected by density of parasitemia.**

**Discussion**

This recent study showed different variations between the study group and control group in both sexes. 33 patients showed microcytic hypochromic cells, when examined microscopically which goes with the reduction in MCV, MCH and MCHC. The MHC was reduced in male patients comparing to that of control group (P. value 0.017). This result was similar to result obtained by Xiao-Hua Wu *et al*. 2002 (P. value 0.01). The female group showed insignificant difference (P.value 0.92). The MHC was significantly affected with the severity of parasitemia .

In this study no difference in the hematocrit of both sexes when compared to control group,while it was reversibly affected with the degree of parasitemia when the mean was compared in mild and sever infections(P. value 0.001).

MCV was decreased in both male and female group when the mean was compared to the control group with P. value 0.001 and 0.003 respectively. The MCV was significantly affected with the severity of parasitemia .

In the current study we found that there was no difference in the RBCs countof male patient group and male control group (P. value 0.18), while it was significantly reduced in females (P. value 0.022). RBCs was not affected with the severity of parasitemia.

This study showed there was a significant reduction in the MCH in the male group (P. value 0.00), while it was insignificant in the female group (P. value 0.05). Severely parasitized patient showed a significant reduction in their MCH when compared with mildly infected patients (P. value 0.00).

Recent study reveals that there was a significant variation in the MCHC in male patients (P. value 0.00). This study showed no difference in the mean of this parameter in the female group (P. value 0.121). MCHC was affected by the density of the parasitemia.

This study indicated that the white blood cells count did not influenced by neither *Schistosoma mansoni* infection, in both sexes , nor the density of parasitemia .

The mean platelet count of study group was normal similar to that of control group in both sexes, although there was a reduction in sever parasitic infection comparing to the mild one (P. value 0.005). This may be due to splenomegaly.

The current study found that there was no difference in the serum iron of male patients and control group of the same gender(P. value0.61) and significantly decreased in the female group (P. value 0.00). This study found that there was a reverse association between density of *Schistosoma mansoni* infection and this parameter.

Serum ferritin was shown to be increased during infections, giving false negative results for iron deficiency anemia (Lipschitz *et al*. 1974). For this reason, it has been suggested to use a high cut-off value for serum ferritin to determine iron deficiency in population where infection and/or inflammatory diseases are highly prevalent (Punnonen K *et al*. 1997). In this study serum ferritin showed no difference in both males and females when

 

compared to the corresponding gender of the control group (P. value 0.122 and 0.14 respectively). The study found that there was a significant effect of density of parasitemia on the serum ferritin (P. value 0.01).The same results were shown by Tjalling Leenstra *et al.* 2006.

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