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Malarial parasite infection and increase of erythrocyte sedimentation rate

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Abstract

A difference in the sedimentation of red blood cells from healthy and non-healthy ones can be seen. To support a previously published work on the idea that red blood cell inclusion could increase of erythrocyte sedimentation rate (ESR). The author hereby studied the ESR in the malarial infection model. It could be seen that ESR is increased in malarial infection and varies on the intensity of infection.

Keywords
malaria, erythrocyte sedimentation

Introduction

Erythrocyte sedimentation rate (ESR) is a non specific hematological parameter. It can be increased in many pathological conditions. A difference

In the sedimentation of red blood cells from healthy and non-healthy ones can be seen. Concerning the mechanism of erythrocyte sedimentation, there are two basic phases, aggregation phase and sedimentation phase (1). The first 10 minutes is “aggregation of red blood cells” which occurs in the early phase of the process, resulting in a mass of collected red blood cell (1). Then the next second phase, 50 minutes is for actual sedimentation of red blood cells (1). This occurs after an appropriate amount of collected red blood cell (1). In the recent published paper, Wiwanitkit reported that the increase of additional mass due to inclusion body could reduce the time for sedimentation (2). The situation of inclusion body in thalassemic cases was used as a model. In this work, the author makes a further implication of this hypothesis in the situation of malaria, an important tropical infectious disease. Experimental model based on mathematical modeling technique was used in this work.

The hypothesis/Idea

The basic hypothesis is based on the previous concept by Wiwanitkit.

Basically, any inclusion body is an object with specific weight that can affect the normal

sedimentation of red blood cell, therefore, faster reaching of proper state

settle down than normal in case of inclusion body that can result in high erythrocyte sedimentation can be hypothesized (2). Because malarial parasite is a blood infection and intracellular malarial particle can be considered as an inclusion body. Therefore, the high ESR in case of malaria can also be proposed.

Evaluation of the Hypothesis/Idea

To verify the idea, the author makes use of observed reported real laboratory test. The author searched the literature that reported the observation on the ESR on the malarial infection condition. The author further simulated this condition and calculated for theoretical ESR and compared it with the real observation in the report. The basic primary assumption for simulation is a) red blood cell has its weight about 50 picogram (3) a malarial parasite has its weight about 10 picogram (4 - 5), b) the common erythrocyte sedimentation rate for general population is about 15 mm/hr (1) and c) there is no additional confounding factor for plasma component and red blood cell amount between normal and malarial infected cases.

Experimental data

Similar to previous published work (2), hypothetical experimentation using mathematical modeling was used in this work.

There are two experiments. The first experiment is to compare between normal and malarial infection. First, it is assumed that the summative mass of collected cells at appropriate status for settling down is equal to A. The time to reach this stage for normal subjects can be (A/50 x N) where N means number of cells. The time to reach this stage for malaria infected patients can be [A/(50 + 10) x N] where N means number of cells. Therefore, the ratio of time comparing between normal and malarial infected subjects can be 1.2 times. This implies that the ESR of malarial infected patients can be only 0.83 of normal subjects.

The second experiment is to assess the difference in malarial infected cases with different intensity. Based on the fact that there can be different percentage of parasitemia in different malarial infected cases, the simulation for ESR at different percentage of parasitemia can be performed. First, it is also assumed that the summative mass of collected cells at appropriate status for settling down is equal to B. If 100 % of red blood cells is infected by malaria, the time to reach this stage for normal subjects can be (A/50 x N) where N means number of cells. However, if 50 % of red cells is infected by malaria, the time to reach this stage for normal subjects can be (A/0.5 x 50 x N) where N means number of cells and if 25 % of red cells is infected by malaria, the time to reach this stage This pattern can be simulated so on. for normal subjects can be (A/0.25 x 50 x N). This can give that the ratio of time comparing between 100 % and 50 % infection can be 2 times. This can also give that the ratio of time comparing between 100 % and 25 % infection can be 4 times. This pattern can be simulated so on.

Validation of this expected value to the reported data in different scenarios showed a good concordance in trend (Table 1).

Table 1. Reported data of erythrocyte sedimentation rate in normal and malarial infection.

Scenarios	Average ESR (mm/hr)
Normal subjects (1)	15
Malarial infected patients (6)	8 - 12

Discussion

Malarial infection is an important tropical mosquito borne infectious disease. This infection is classified as a blood infection. Intraerythrocytic parasite infection is the hallmark of this disease. Effect of malarial infection on ESR is hereby tested.

Following the previous original hypothesis (2), it can be imagined that the ESR in malarial infection should be high and variation due to the intensity of infection can be expected. According to this work, the trend of increased ESR by increased percentage of malarial infection can be seen. In literature, it can be seen that ESR of malarial infected subjects is faster than normal subjects (6 – 8) and it can also be seen that ESR of cases with more parasitemia is faster than that of lower (8 – 10).

However, it should be noted for some limitations on this work. The main limitation is that the author basically presumes that there is no additional confounding factor for plasma component and red blood cell amount between normal and malarial infected cases. Indeed, there can be and should be those factors that modify the ESR rate. The change in the C-reactive protein in malarial infection is the good example (6 – 7). This might explain no direct fit of the ESR results to the expected simulated value and can explain the more increase in rate in infected subjects. However, despite this fact, the observation from this study still confirms the hypothesis. The trend of increased ESR can still be seen. By the way, it might bring some usefulness that sedimentation rate less than 12 (0.83 of normal reference value) can be a suspicious point for febrile illness for malaria.

To further verify this observation, the author tried to assess the usefulness of the derived cut off sedimentation rate in the real experimental setting. Applied to the reported value in the experimental study of Erickson et al (6), this cut off can be useful. It can be seen that all studied cases (N = 258) in this series (6) had sedimentation rate according to this observation.

Conclusion

It can demonstrate that malarial infection acts as inclusion body and can increase ESR. This can support the hypothesis that additional mass due to any intraerythrocytic inclusion can increase ESR.

References

1.Wiwanitkit V, Siritantikorn A. Methods to determine erythrocyte sedimentation rate in the present day. Chula Med J 2002;46(4):87 –102.
2. Wiwanitkti V. Red blood cell inclusion will increase of erythrocyte sedimentation rate. Irn J Med Hypotheses Ideas, 2008, 2:11.
3. Rusu V, Lctuu D, Rileanu I. Red cell shape--a biophysical analysis. Rev Med Chir Soc Med Nat Iasi. 2007;111(1):194-199.
4. Arese P, Schwarzer E. Malarial pigment (haemozoin): a very active 'inert' substance. Ann Trop Med Parasitol. 1997 Jul;91(5):501-16.
5. Rudzinska MA. The fine structure of malaria parasites. Int Rev Cytol. 1969;25:161-99.
6. Eriksson B, Hellgren U, Rombo L. Changes in erythrocyte sedimentation rate, C-reactive protein and hematological parameters in patients with acute malaria. Scand J Infect Dis. 1989;21(4):434-41.
7. Khan AS, Malik SA. Haematological changes in falciparum malaria and tumor necrosis factor. J Pak Med Assoc. 1996 Sep;46(9):198-201.
8. Karunaweera ND, Carter R, Grau GE, Mendis KN. Demonstration of anti-disease immunity to Plasmodium vivax malaria in Sri Lanka using a quantitative method to assess clinical disease. Am J Trop Med Hyg. 1998 Feb;58(2):204-10.
9. Lortholary O, Danis M, Casassus P, Felix H, Gay F, Datry A, Gentilini M.
Subacute Plasmodium falciparum malaria in 43 patients returning from areas with chloroquine in Africa. Ann Med Interne (Paris). 1994;145(3):155-8.
10. Loudová J, Giboda M, Gutvirth J. The clinical picture of imported malaria and its relation to P. falciparum parasitemia. Bratisl Lek Listy. 1993 Apr;94(4):218-23