RBC Count and Its Differentiation Potential among α-Thalassemia (SEA type), β-Thalassemia and HbE Heterozygotes

Thanusak Tatu[1,3*], Daraporn Prakunwisit[1], Sumonthida Sayajak[1], Sawitree Chiampanichayakul[1,3] and Watchara Kasinrerk[2,3]

[1]Research Center for Hematology and Health Technology, Division of Clinical Microscopy, Department of Medical Technology, Faculty of Associated Medical Sciences, Chiang Mai University, Chiang Mai 50200, Thailand

[2]Division of Clinical Immunology, Department of Medical Technology, Faculty of Associated Medical Sciences, Chiang Mai University, Chiang Mai 50200, Thailand

[3]Biomedical Research Unit, Faculty of Associated Medical Sciences, Chiang Mai University, Chiang Mai 50200, Thailand

**Corresponding author. E-mail: asittt@chiangmai.ac.th*

ABSTRACT

In order to determine the differentiation potential of red blood cell parameters between severe and mild form of thalassemia heterozygotes, we have carried out automated blood cell analysis, one-tube osmotic fragility test (OFT), Hb H inclusion body test, hemoglobin identification by high-performance liquid chromatography (HPLC) and α-thalassemia 1 (Southeast Asian [SEA] type) genotyping in 58 thalassemia heterozygotes. Red blood cell (RBC) parameters in different thalassemia heterozygotes were compared. No difference in red blood cell (RBC) count was observed between α-thalassemia 1 (SEA type) and β-thalassemia heterozygotes. RBC count was significantly higher in α-thalassemia 1 heterozygotes (SEA type) and β-thalassemia heterozygotes than that in HbE heterozygotes. We concluded that the RBC count could not differentiate α-thalassemia 1 heterozygote (SEA type) from β-thalassemia heterozygote. However, if considered with MCV, MCH, MCHC and RDW, it provided great values in screening severe α-thalassemia 1 (SEA type) and β-thalassemia heterozygotes out of HbE heterozygote.

Key words : Red blood cell parameters, Red blood cell indices, Thalassemia screen, Thalassemia heterozygote, HbE heterozygote

INTRODUCTION

Thalassemia is a syndrome characterised by reduction or absence of globin chain synthesis, comprising two common types: α- and β-thalassemia. α-thalassemia is generally caused by deletion of α-globin gene(s) resulting in 2 genotypes: – and -α for severe α-thalassemia 1 and mild α-thalassemia 2 forms, respectively. β-thalassemia, on the other hand, is mostly resulted from point mutations within and flanking structural β-globin gene which also gives rise to 2 sub-types which