Brief comment on the case report by Fattori *et al.* 2005

The authors report a case of myocardial infarction in a young man affected with SCD and treated with hydroxyurea. Since apparently no evidence of coronary lesions was found the authors assume that a (moderate) increase in PCV could have been the cause of infarction, in association with hydroxyurea treatment.

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It is indeed a fact that an increased blood viscosity (and PCV) will increase the risk for infarction in the post capillary venules in sickle cell disease. It is also a fact that polymerization of the red cells is enhanced when hemoglobin have reached the deoxy state. It is also generally assumed that HU will decrease the adherence of the red cells to the wall tissue and increase the HbF content of the red cells in predisposed patients, both being beneficial effects against HbS polymerization and infarctions. Conditions favorable to HbS polymerization should not be present in the arterial circulation of the hearth and should be even less present in patients treated with HU.

The authors report an Hb of 10.7 gr. and a PCV of 31.3% at the time of the myocardial infarction and 10.2% HbF compared with the 3.2% before HU treatment. These parameters are not unusual in SCD and prove only a good response to the drug. The authors also report an MCV of 123 fl, which is very unusual in SCD. An MCV of 123 fl is measured at birth with near 100% fetal cells and 80% HbF or in conditions of significant vitamin B12 deficiency. Deficiencies of folate and vitamins B6 and B12 cause mild to moderate homocyst(e)inemia, which is considered to be strongly associated with the increased risk of vascular events.[1,2] Did the authors investigate the cause of the high MCV?

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