Research paper on sickle cell anemia

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According to an article written on November 14th, 2011, in New York Times by Donald McNeil on recent research findings on sickle cell anemia element responsible for its protective nature when it comes to malaria, a lot could be deciphered. Donald first provides an overview of the prevalence of Sickle cell anemia in West Africa and how it can be inherited either resulting to sickle cell trait or sickle cell anemia (McNeil, 2011). The article begins by providing a summary of the morphological changes that occurs at the red blood cell level when a malaria parasite enters a given cell (McNeil, 2011). Donald describes a recent study on sickle cell anemia carried out by scientists in Burkina Faso and Germany that found out that malaria parasites often reshape the red blood cells' actin into scaffolding hence allowing them to mount a certain protein shuttling structure named Maurer's cleft (McNeil, 2011). The research found out that it is via that cleft that sticky proteins are produced which result to adherence of the red blood cells on capillary walls thereby evading being taken to the spleen for destruction (McNeil, 2011). The research found out that those with sickle cell trait had protection against malaria since their red blood cells resisted actin reshaping. The research thereby concluded that those individuals that carried the sickle cell gene more so became more protected against malaria as a disease due to its unique features (McNeil, 2011).

According to an article by Samir Ballas on various neurological complications exhibited by sickle cell patients published in the journal of America medical Association, a lot regarding the cognitive function of such patients became reported (Samir, 2010). The research on cognitive functioning of the brain became conducted on sickle cell patients with a control of normal patients

without the disease (Samir, 2010). The study revealed that adults with sickle cell anemia became more prone to experience cognitive problems like difficulty in making decisions, organizing thoughts or learning (Samir, 2010). The research entailed 149 sickle cell patients aged between 19 and 55 years (Samir, 2010). A comparison was then made on a healthy study population of 47 participants of similar age plus education background of African-American origin (Samir, 2010). The study continued to explain that some brain injuries in sickle cell patients resulted due to stroke leading to further neurological complications. The research became beneficial in understanding sickle cell as a disease, plus helps improve on the management of the disease (Samir, 2010).

According to an article on Arq Neuropsiquiatr written by Belisário et al (2012), regarding a research on alpha thalassemia being protective against cerebrovascular disease (CVD) in patients with sickle cell disease (Belisário et al., 2012). The research was conducted on children and acknowledged in the article cerebrovascular disease as a complication of sickle cell (Belisário et al., 2012). The article points out those Filho et al., discovered the Bantu globin gene haplotype (βS) association to occurrence of CVD, whereas alphathalassemia did not have any association. In the study, they discovered that the alpha thalassemia genotypes greatly became associated with reduced risk of CVD (Belisário et al., 2012). The study also did not find out any given association between HBF concentration and CVD in patients with sickle cell (Belisário et al., 2012).

References

Belisário AR, et al. (2012). Alpha-thalassemia protects against cerebrovascular disease in children with sickle cell anemia. Arq Neuropsiquiatr, 70 (8), 645.

McNeil, D. G. (2012, Novemeber 4). Biochemistry: Scientists Decode the Protective Element Sickle Cell Anemia Offers Against Malaria. Retrieved October 17, 2012, from New York Times: http://www. nytimes. com/2011/11/15/health/biochemistry-scientists-decode-the-protective-element-sickle-cell-anemia-offers-against-malaria. html? _r= 1 Samir K. Ballas (2010). Neurocognitive Complications of Sickle Cell Anemia in Adults. JAMA, 303 (18), 1862-1863