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Letter to Editor

Sterile 'Abscess' of the Spleen and the Sickle Cell Trait

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Dear Editor,

I read with interest the case report by Dr. P. Magro *et al.*[1] regarding a boy with sickle cell trait (AS), who was appropriately treated for *Plasmodium falciparum* malaria and who, upon ultrasound imaging, was thought to have multiple abscesses in the spleen, eventually interpreted as splenic infarction.

This case history brought to my mind a segment of medical literature so ancient, that understandably the Authors may not have been familiar with it. Probably the most comprehensive article was that published by T M Kolawole and S P Bohrer[2] in 1973. They reported from Ibadan, Nigeria a series of 20 patients with 'primary splenic abscess': in 15 of them hemoglobin electrophoresis had been carried out, and 12 of these (80%, as against a population frequency of about 24%) were heterozygous for Hb S (6 AS and 6 SC). In a few cases cultures yielded *Salmonella*, an organism to which patients with sickle cell disease are very susceptible (see Magnus et al.[3]); but in the majority of cases the 'abscess' was sterile – an oxymoron. From a review of their patients and of previous literature Kolawole & Bohrer[2] suggested that in these patients one was dealing with infarction rather than infection; and that a crucial factor in pathogenesis is the presence of hemoglobin S (and therefore of red cells that can sickle). Whether malaria is a common trigger of this serious pathology is not clear; but it is a fact that patients often presented with high fever.

Thus, the syndrome of left upper quadrant abdominal pain and abnormal spleen imaging simulating abscess(es), but probably due in most cases to arterial thrombosis followed by infarction and colliquation, is well known; as is its association with Hb S. Given that in Nigeria alone there are at least 20 million AS heterozygotes, we can safely say that the syndrome is very rare; and therefore it does not detract from the conventional teaching that the AS trait is almost entirely asymptomatic. At the time this syndrome was first recognized, it was regarded as an indication for surgery. However, today I would commend Paola Magro and colleagues for having opted for conservative management, since splenectomy is not free from complications, and it is imperative only if the spleen is ruptured. It will be interesting to do follow-up imaging on their patient: from experience with patients who have undergone therapeutic selective splenic artery embolization (for the very different indication of paroxysmal nocturnal hemoglobinuria: see Araten et al.),[4] I think one would expect gradual healing without sequelae.

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