Anemic crisis due to *Mycoplasma pneumoniae* complication in sickle cell patients

To the Editor

I read the interesting article by Zafer et al[1] on the anemic crisis due to *Mycoplasma pneumoniae* (*M. pneumoniae*) complication in sickle cell patients. Sickle cell disease (SCD), one of the common hemoglobinopathies all over the world, is associated with frequent and often severe infections as a result of immune function impairment and functional asplenia. Though *M. pneumoniae* is commonly correlated with acute chest syndrome in patients with sickle cell anemia and represents one of the leading causes of hospitalization, morbidity, and mortality, hemolytic crises do occur following infection with that pathogen in sicklers. I have 4 comments regarding the aforementioned study.

First, some concerns exist considering the sensitivity and specificity of the serological test adopted to diagnose *M. pneumoniae* infection in the studied patients, namely, the enzyme immunoassay (EIA) for *M. pneumoniae* (ImmunoWell, Gen Bio, San Diego, USA.). The IgM EIA serology test is a valuable tool for the early diagnosis of *M. pneumoniae* infections as long as the EIA used is specific. In one study,[2] it was found that there are few commercial serologic assays for the detection of *M. pneumoniae* infections with appropriate performances in terms of sensitivity and specificity. The assays tested were Platelia EIA (Bio-Rad, Veenendaal, Netherlands), Sero MP EIA (Savyon, Ashdod, Israel), Serion Classic EIA (Virion/Serion, Serion, Würzburg, Germany), Biotest EIA (Biotest, Colorado, USA), Ridascreen EIA (r-Biopharm, Darmstadt, Germany), AniLabsystems EIA (Ani Labsystems, Vantaa, Finland), Novum EIA (Novum Diagnostica, Dietzenbach Dietz, Germany), Diagnosys EIA (MP products, Amersfoort, Netherlands), Genzyme/Virotech EIA, ImmunoWell EIA (Gen Bio, San Diego, USA.), Immuno Card EIA (Meridian, Cincinnati, USA), Serodia Myco II micro particle agglutination (Fuji Rebio, Tokyo, Japan), and Complement Fixation Test (CFT, Krizikova. Czech). The results showed low specificities for both the Novum and the Immuno Card IgM assays. The IgM assays with the best performances in terms of sensitivity and specificity were AniLabsystems (77% and 92%), Sero MP (71% and 88%), and complement fixation test (65% and 97%). Polymerase chain reaction (PCR) remains superior to serology for diagnosis of *M. pneumoniae* infection during the early phases of infection.[3]

Second, in the clinical setting, chest radiograph is one of the reference standards in assessing and establishing the diagnosis in febrile patients, even in those lacking respiratory manifestations. Due to its great variability in clinical presentations and symptomatology, *M. pneumoniae* infection is often not considered as the first diagnosis. This might lead to a delay in the diagnosis and institution of an appropriate treatment. Though the studied patients lacked marked respiratory complaints at the initial presentation as shown in Table 1 of the study, chest radiograph deemed pertinent to be performed as it could alert the clinicians to potential existence of the *M. pneumoniae* infection through visualization of various radiological manifestations. These include bronchopneumonic consolidations, pleural effusion, perihilar adenopathy with interstitial infiltrates, and atelectatic areas.[4] I wonder whether the studied patients underwent any chest radiographs and what were their radiological findings if they had.

Third, the reticulocytopenia observed in one studied sickler might be due to the observation that in SCD, both mature sickle cells and sickle reticulocytes adhere more readily to macrophages. It appears that the HbS cells are destroyed by hyperactive macrophages where adhesion molecules expressed on red blood cell erythroid precursor cells and reticulocytes can interact with macrophages and cause hemolysis. Therefore, the reticulocytopenia observed is likely to be due to peripheral consumption (namely, destruction by macrophages either by direct contact lysis or by erythrophagocytosis) rather than suppression of erythropoiesis. Cessation of hemolysis during intravenous immunoglobulins (IVIGs) and steroid treatment might be due to IVIGs blocking of the adhesion of sickle cells and reticulocytes to macrophages together with steroid suppression of macrophage activity.[5]

Fourth, clinicians are needed to consider infection with *M. pneumoniae* as a potential etiology for any unexplained and worsening anemia in sickle cell patients. Efforts must be directed towards its documentation and institution of proper treatment to preserve the lives of these patients.

Prof. Mahmood D. Al-Mendalawi
Department of Paediatrics
Al-Kindy College of Medicine
Baghdad University
Baghdad, Iraq

Reply from the Author

No reply was received from the Author.

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Related topics

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